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ORIGINAL ARTICLE

Parental punitive discipline, negative life events and gene–environment interplay in the development of externalizing behavior

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Background. To investigate the extent to which three putative 'environmental' risk factors, maternal punitive discipline (MPD), paternal punitive discipline (PPD) and negative life events (NLEs), share genetic influences with, and moderate the heritability of, externalizing behavior.

Methods. The sample consisted of 2647 participants, aged 12–19 years, from the G1219 and G1219Twins longitudinal studies. Externalizing behavior was measured using the Youth Self-Report, MPD, PPD and exposure to NLEs were assessed using the Negative Sanctions Scale and the Life Event Scale for Adolescents respectively.

Results. Genetic influences overlapped for externalizing behavior and each 'environmental' risk, indicating gene–environment correlation. When controlling for the gene–environment correlation, genetic variance decreased, and both shared and non-shared environmental influences increased, as a function of MPD. Genetic variance increased as a function of PPD, and for NLEs the only interaction effect was on the level of non-shared environment influence unique to externalizing behavior.

Conclusions. The magnitude of the influence of genetic risk on externalizing behavior is contextually dependent, even after controlling for gene–environment correlation.

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Key words: Externalizing behavior, gene–environment correlation, gene–environment interaction, negative life events, punitive discipline.

Introduction

Adoption studies (Cadoret *et al*. 1995) and molecular genetic studies (Caspi *et al*. 2002; Foley *et al*. 2004) have suggested that gene–environment interactions (G × E) are likely to be important in understanding risk for antisocial behavior. These studies suggest that genetic factors increase susceptibility to environmental risk. For instance, Cadoret *et al*. (1995) demonstrated that negative family environment only increased children's problem behavior in the context of genetic risk (having antisocial biological parents). Similarly, molecular studies have demonstrated that exposure to maltreatment was only a substantial risk for antisocial outcomes when it occurred in the context of a particular allele of the monoamine oxidase A gene (Caspi *et al*. 2002). These findings are consistent with the stress-diathesis model (Shanahan & Hofer, 2005), which proposes that environmental context may trigger an underlying genetic risk for a phenotype.

There is emerging evidence from twin studies for other types of gene–environment interplay in relation to antisocial outcomes, whereby environmental processes moderate genetic risk, and ultimately estimates of heritability (Button *et al*. 2005; Tuvblad *et al*. 2005). Thus in one study heritability of antisocial behavior was higher in those with low *versus* high levels of family dysfunction (Button *et al*. 2005). These findings are consistent with the bioecological model (Bronfenbrenner & Ceci, 1994), which states that the mechanisms by which genotypes actualize into phenotypes vary as a function of environmental context. When proximal processes are weak, that is when the environment is not conducive to expression of that genotype, heritability is low, as genetic potential is not realized. Results of this kind are also consistent with the 'social push perspective' (Raine, 2002), which suggests that a highly negative environment predisposes to negative outcomes to such an extent as to suppress genetic influence. If replicated, such findings

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hold important implications for our understanding of the risk processes for antisocial behavior, and also for intervention strategies.

In addition, there is the confounding issue of gene–environment correlations (rGE) – genetic influences on aspects of the environment. rGE is likely to be important in many aspects of psychopathology (Plomin *et al*. 1977; Scarr & McCartney, 1983), and perhaps especially so for antisocial behavior. First, antisocial parents provide suboptimal rearing environments for their children, as well as transmitting a genetic predisposition for antisocial behavior (passive rGE). Second, antisocial behavior in children can evoke negative reactions from others (evocative rGE). Third, the outcomes of antisocial behavior can function to 'select' individuals into adverse environments (active rGE).

In practice, both rGE and G × E probably operate in the development of antisocial behavior (Rutter & Silberg, 2002). In such instances, it is difficult to determine whether increased associations between genes and environment are the result of the environment modifying the effects of genes (G × E), or the genetic risk being more prevalent within certain environments (rGE). Consequently, rGE may mask detection of G × E and vice versa. In instances where we want to test for interactions, but suspect gene–environment correlation, we need to use statistical approaches that can disentangle these effects.

This paper explores these issues of co-occurring gene–environment interaction and correlation in relation to three well-established correlates for externalizing behavior. Maternal punitive discipline (MPD) and paternal punitive discipline (PPD) may reflect passive and/or evocative gene–environment correlations, whereas dependent negative life events (NLEs) could represent active gene–environment correlation.

Parental discipline

Aspects of parental discipline (Kerr *et al*. 2004), particularly harsh (Nix *et al*. 1999) and inconsistent (Rutter *et al*. 1998) discipline, are well-documented risk factors for externalizing behavior. There is evidence of a genetic contribution to maternal discipline (Wade & Kendler, 2000), with genes contributing between 10% and 40% depending on the rater. Thus, discipline may index a risk for behavioral problems as well as being a consequence of them. Harsh discipline may impact upon children's development, thereby influencing their behavior, but equally importantly children's behavior can evoke negative reactions from parents (Ge *et al*. 1994; O'Connor *et al*. 1998; Riggins-Caspers *et al*. 2003). Moreover, genetic liability appears to be moderated by parental discipline. For example, Riggins-Caspers *et al*. (2003) found that the association between biological risk for behavioral problems and harsh physical discipline by the adoptive parent was mediated by the child's own negative behavior (rGE), but only in those exposed to environmental risks for the negative behavior (G × E). To date, evidence of gene–environment interaction in the presence of gene–environment correlation has been limited to adoption designs.

Negative life events

Although an association between NLEs and externalizing behavior was proposed almost 30 years ago (Robins, 1978) and studies have demonstrated a correlation (Champion *et al*. 1995; Wiesner & Windle, 2004), there has been limited research into the association. There are, however, several plausible reasons to expect such links. First, negative events may carry risk for externalizing phenotypes. Alternatively, individuals who are predisposed to externalizing behavior may elicit, or seek out, such events, for example by placing themselves in high-risk situations. This interpretation is supported by evidence of a genetic contribution to negative life events (Thapar & McGuffin, 1996), particularly those dependent on individuals' own behavior (Billig *et al*. 1996).

The aims of this study were to identify whether there is a shared genetic liability between externalizing behavior and three 'environmental' risk factors, and to investigate whether heritability is moderated by levels of exposure to these 'environmental' risks. We expected the covariation between externalizing behavior and MPD, PPD and NLEs to be partially due to common genetic risk, and that the heritability of externalizing behavior would be contextually dependent.

Method

Participants

The analyses use the G1219 and G1219Twins longitudinal studies. G1219 consists of all adolescent offspring of adults from a large-scale population-based study (GENESiS; Sham *et al*. 2000). Of the 9000 families contacted through GENESiS, a total of 3600 (40%) participated either in this study (of adolescents) or in another study on childhood hyperactivity. Participants were only eligible for one or other study on the basis of their age (full details are given elsewhere; Eley *et al*. 2004). Of the 3600 responses received, a total of 1818 (20%) adolescents from 1294 families took part in G1219, the remainder participating in the hyperactivity study. The G1219Twins are

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Table 1. *Mean, standard deviations and twin pair correlations for externalizing behavior, maternal punitive discipline and negative life events split by sibling type (MZ, DZ and full siblings) and sex*

			DZ				Full sibling			
	MZ				Opposite sex				Opposite sex	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
n individuals	309	386	246	376	323	334	120	200	124	144
n complete pairs	153	192	122	187	323		52	90	118	
n incomplete pairs	3	2	2	2	11	0	16	20	26	6
Externalizing behavior										
Mean	11.89	10.73	12.83	12.51	12.96	12.13	13.45	13.04	13.93	12.75
S.D.	7.91	6.19	8.08	7.27	7.77	6.61	7.42	7.44	7.82	6.55
Correlation	0.47	0.61	0.24	0.44	0.24		0.37	0.20	0.20	
Maternal punitive discipline										
Mean	7.10	6.73	6.51	7.39	6.67	6.84	5.84	6.09	5.94	6.69
S.D.	4.38	4.04	3.94	4.18	3.99	3.81	3.90	3.85	3.66	3.94
Correlation	0.50	0.38	0.15	0.35	0.22		0.32	0.31	0.26	
Paternal punitive discipline										
Mean	6.28	5.97	6.13	6.63	6.26	6.62	5.71	5.19	5.52	6.18
S.D.	4.96	4.41	4.94	4.92	4.67	4.43	4.49	3.85	4.24	4.12
Correlation	0.60	0.49	0.39	0.41	0.33		0.51	0.49	0.46	
Negative life events										
Mean	−0.12	−0.13	0.06	0.05	0.02	−0.04	0.04	0.07	0.07	0.07
S.D.	0.98	0.92	1.02	1.05	1.00	0.96	0.94	1.05	0.98	0.97
Correlation	0.48	0.43	0.31	0.36	0.21		0.06	0.37	0.23	

MZ, Monozygotic; DZ, dizygotic, S.D., standard deviation.

a random selection of twins, born between 1985 and 1988, identified by the UK Office of National Statistics. Health authorities and general practitioners contacted 2947 families, of whom 1381 (47%) participated. The siblings were combined with the twins to increase the power to detect shared environmental effects. Both samples were sent two reminders, and only respondents aged 12–19 years were included. The siblings were slightly older than the twins (mean recruitment age: siblings = 15.20 years, twins = 14.09 years, difference = 1.11 years, $t = 18.06$, df = 3166, $p < 0.001$) and significantly more likely to have mothers with at least one A-level (internationally recognized pre-university qualification, typically taken at age 18; percentage of mothers with A-level or higher: siblings = 46.4%, twins = 39.7%, $\chi^2 = 11.33$, df = 1, $p = 0.001$). Although significant, these differences were too small to represent meaningful differences. The samples do not differ significantly from one another for MPD or PPD or exposure to NLEs. Informed consent was obtained from parents of all adolescents under 16, and from the adolescents themselves when over 16. Ethical approval for this study was given by the Research Ethics Committee of the Institute of Psychiatry and South London and Maudsley National Health Service (NHS) Trust.

These analyses focus on the second wave of data collection ($n = 1820$), which took place approximately 8 months (S.D. = 5 months, range = 0.8–25 months) after initial contact. Zygosity was assessed by maternal report of physical similarity (Cohen *et al*. 1975). Total data were available for 2648 individuals. Eighty-four twin pairs were of unknown zygosity and consequently were excluded from all genetic analyses, resulting in a sample of 2562 individuals. Of these, 589 were from sibling pairs (120 male, 200 female, 268 opposite sex), 695 were monozygotic (MZ) twins (309 male, 386 female) and 1279 were dizygotic (DZ) twins (246 male, 376 female, 657 opposite-sex). A complete description of the numbers of complete and incomplete pairs (7%) is provided in Table 1. The mean age of the sample at wave 2 was 15.0 years (range 12–21 years); 48% were boys.

Levels of parental education were higher than observed in a nationally representative sample (39% *v*. 32% educated to A-level or above), and parents were more likely to own their own houses (82% *v*. 68%; Meltzer *et al*. 2000). To reduce the impact of possible

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response bias associated with educational level, the sample was reweighted to match the distribution of educational qualifications in the representative sample (Meltzer *et al*. 2000). Only 74% of the wave 1 sample (2651 individuals) participated in wave 2; females, families with higher parental qualifications, and owner-occupiers were more likely to continue to participate. A second weight was created and multiplied with the wave 1 weight to account for both initial response and later attrition biases (roughly 26%). Weights were family-general and thus did not incur any additional individual-specific effects between members of the same family.

Measures

Externalizing behavior was measured using the Youth Self-Report (Achenbach, 1991). Items are rated as not true to very true (scored 0 to 2) and summed to provide an externalizing behavior score. The scores were positively skewed (skew = 1.124, kurtosis = 1.664), but did not represent a J-shaped distribution.

Ratings of MPD and PPD were assessed using the Negative Sanctions subscale adapted from a previously well-validated parent–child relationship measure (Hetherington & Clingempeel, 1992; Dunn *et al*. 1998). This consists of five items such as 'Does your mother/father yell at you for what you did?' Internal consistency was acceptable (mother: $\alpha = 0.66$; father: $\alpha = 0.72$). The distribution of scores was not significantly skewed for maternal (skew = 0.556, kurtosis = 0.121) or paternal punitive discipline (skew = 0.529, kurtosis = 0.017).

NLEs were assessed with the Life Event Scale for Adolescents, a checklist of 50 events for which adequate reliability has been demonstrated (Coddington, 1984). As behavioral genetic analyses cannot test the heritability of items that are necessarily shared by siblings (Purcell & Koenen, 2005), only 12 items that were individual-specific, negative, and dependent upon the individual's own actions, such as 'failing to achieve things that you want', were used in these analyses (Rowe *et al*. 2006). Cronbach's α was 0.59; the scale was positively skewed (skew = 1.086; kurtosis = 0.864), although it did not present a J-shaped distribution.

Analyses

Means and correlations were calculated separately for males and females, by sibling type, using SPSS (2004). Model-fitting was performed using the structural equation modeling package, Mx (Neale, 1997). The maximum likelihood function of Mx was applied to appropriately transformed raw data (i.e. regressed on age and sex) to avoid loss of information due to missing data, and the 'Weight' function of Mx was used to incorporate the sampling weights. This function controls for attrition by adjusting the log-likelihoods for each family by the sample weights described above.

We fit univariate models to our data to estimate the relative contribution of genetic (A), shared environment (environment that makes family members similar to one another; C) and non-shared environment (environment that acts to make family members different from one another; E) to the phenotypic variances. Behavioral genetic analyses make use of differences in the genetic relatedness of different sibling types to partition the phenotypic variance. For example, MZ twins share 100% of their genes and shared environment, but none of their non-shared environment. Therefore, similarities between them result from shared genes and shared environmental factors. DZ twins and full siblings share on average only half their genes, and any shared environmental factors. Consequently, if MZ twins are more similar to one another than DZ or full siblings, genes must play a role. We also tested for sex-effects on the magnitude of A, C and E, by first fitting a full sex-limitation model, allowing for A, C and E estimates to differ for males and females and the genetic correlation between opposite sex twins to differ from 0.5 (the genetic correlation between same-sex full sibling and DZ twin pairs as described above). Following this, we fit a scalar sex-limitation model in which the A, C and E parameters were constrained to be the same across sex, and the opposite sex genetic correlation was fixed to 0.5, but the male and female total variances were allowed to differ, to establish if this resulted in a significant deterioration in fit.

The fit of the full models was compared with that obtained from saturated models in which there are no constraints and all possible parameters (means, variances and covariances of/between variables) are estimated. These are descriptive models that fit the data perfectly, and thus serve as a comparative model for nested models. Comparing the -2 log likelihood (LL) fit of the ACE models with the saturated model produces a χ^2 statistic, which, along with the degrees of freedom (df) of the full model, calculated as the difference in the number of parameters estimated in the two models, makes it possible to determine how well the full model explains the observed data. A low, non-significant χ^2 statistic indicates that the model explains the observed data well. Detection of a significant genetic influence on the 'environmental' variable would provide evidence for rGE.

In addition, we estimated the extent to which genes and environment contribute to covariation between externalizing behavior and each of the

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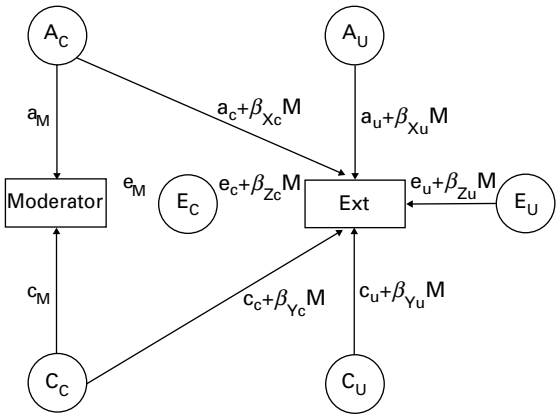

Fig. 1. Gene–environment correlation and interaction model. A_C, genetic influence common to the moderator and externalizing behavior (Ext); A_U, genetic influence unique to externalizing behavior (Ext); C, shared environment influence; E, non-shared environment influence; a_M, the influence of A_C on the moderator variable; a_C, the influence of A_C on externalizing behavior; a_U, the influence of A_U on externalizing behavior; M, mean; β, moderation term.

'environmental' risk measures using a bivariate Cholesky decomposition. This model partitions the variances and covariances into A, C and E. All variables that contribute to the covariance and are shared in common by both phenotypes are referred to as 'common' effects, and all those that contribute to the variance of externalizing behavior but are not shared with the 'environmental' variable are referred to as 'unique', as they are unique to externalizing behavior. To avoid confusion, the term 'shared' will be used to refer to environmental variance that is shared by family members and makes them similar to one another, although this is frequently referred to as 'common environment' in the literature. A significant common genetic pathway indicates that the genetic factor contributing to exposure to the 'environment' also contributes to the predisposition for the behavioral outcome, and thus controls for gene–environment correlation.

The final set of models examined whether the genetic and environmental influences on externalizing behavior are moderated by levels of exposure to each of the putative environmental risks, while simultaneously accounting for genetic and environmental correlations between each with externalizing behavior (Purcell, 2002; see Fig. 1), as described earlier. The potential moderator is modeled both as an outcome and as a moderating variable on all the path coefficients to the externalizing behavior, which are expressed as linear functions of the moderator. As a result, the variance of externalizing behavior is the consequence of the main effects from the common genetic, shared environmental and non-shared environmental influences that are shared with the moderating variables (e.g. a_C), and those variables unique to externalizing behavior (e.g. a_U), and the interaction between each of these terms with the moderator variable (e.g. β_{XC}). The significance of each interaction term was tested by dropping the moderation effect and comparing the fit of these nested models with the full model. Interaction terms that could be dropped without a significant change in χ^2 were excluded from the full model to determine the most parsimonious model. A significant interaction term provides evidence for environmental moderation of the levels of the variance components for externalizing behavior, while accounting for the presence of any gene–environment correlation identified from the univariate and bivariate models.

Results

Summary results

The means, standard deviations, sample size and twin pair correlations for externalizing behavior, MPD, PPD and NLEs are presented in Table 1. Males had significantly higher mean levels of externalizing behavior than females ($t = 2.040$, $p = 0.042$), but there were no significant sex differences in the mean levels of MPD ($t = 1.765$, $p = 0.078$), PPD ($t = 1.066$, $p = 0.287$) or NLEs ($t = 0.501$, $p = 0.617$). There were small correlations with age for all three variables (externalizing behavior: $r = 0.041$, $p < 0.05$, MPD: $r = -0.148$, $p < 0.01$; PPD: $r = -0.111$, $p < 0.01$; and NLE: $r = 0.022$, $p =$ N.S.). All scores were regressed for age and sex, and standardized residuals were used for the genetic analyses.

Genetic model-fitting results

The results of the univariate analyses are presented in Table 2. The full univariate externalizing behavior model fit the data significantly worse than the saturated model. Inspection of the model showed that this resulted from a lower variance for externalizing behavior in the female MZ twins (0.72) compared with the other female sibling types (0.92). However, comparison of both the Akaike information criterion (AIC) and the Bayes information criterion (BIC) of the two models demonstrate lower AIC and BIC fit statistics for the full model, indicating that the models do fit better than the saturated model. All other univariate models fit the data well, according to the $\Delta\chi^2$ between the full and saturated models, There were no significant sex effects on A, C and E for any of the variables. There were significant genetic and non-shared environmental influences on all variables. Shared environmental influences were significant for the PPD measure.

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Table 2. *Univariate model fit statistics for saturated and full models, and parameter estimates for genetic (A), shared environment (C) and non-shared environment (E) influences on externalizing behavior, maternal and paternal punitive discipline and negative life events*

	−2LL (df)	χ^2 (df)	*p*	AIC	BIC	A	C	E
Externalizing behavior								
Saturated model	6056.552 (2420)			1216.552	−5618.365			
Full sex-limitation	6107.701 (2452)	51.149 (32)[a]	0.017	1203.701	−5703.126	M = 44 (7–57)	3 (0–32)	54 (43–67)
						F = 66 (46–72)	0 (0–16)	34 (28–42)
Scalar sex-limitation	6117.038 (2455)	60.552 (35)[a]	0.005	1207.038	−5713.177	57 (42–62)	0 (0–11)	43 (38–50)
		9.337 (3)[b]	0.025					
Drop A	6152.258 (2456)	35.220 (1)[c]	0	1240.258	−5699.140	0	35 (30–40)	65 (60–70)
Drop C	**6117.038 (2456)**	**0 (1)[c]**	**0**	**1205.038**	**−5716.750**	**57 (50–62)**	**0**	**43 (38–50)**
Maternal punitive discipline								
Saturated model	6166.894 (2420)			1326.894	−5563.194			
Full sex-limitation	6198.535 (2452)	31.641 (32)[a]	0.484	1294.535	−5661.709	46 (20–56)	0 (0–22)	54 (44–65)
						06 (00–35)	31 (8–43)	63 (52–72)
Scalar sex-limitation	6203.173 (2455)	36.279 (35)[a]	0.409	1293.173	−5670.109	29 (09–48)	13 (0–27)	58 (51–66)
		4.638 (3)[b]	0.200					
Drop A	6211.053 (2456)	7.880 (1)[c]	0.005	1299.053	−5669.742	0	32 (27–37)	68 (63–73)
Drop C	**6206.005 (2456)**	**2.832 (1)[c]**	**0.092**	**1294.005**	**−5672.266**	**45 (38–51)**	**0**	**55 (49–62)**
Paternal punitive discipline								
Saturated model	6056.151 (2420)			1216.151	−5618.566			
Full sex-limitation	6093.994 (2452)	37.843 (32)[a]	0.220	1189.994	−5713.980	37 (8–65)	21 (0–45)	42 (33–52)
						22 (0–47)	29 (8–48)	49 (40–60)
Scalar sex-limitation	**6095.278 (2455)**	**39.127 (35)[a]**	**0.290**	**1185.278**	**−5724.057**	**30 (12–47)**	**24 (11–37)**	**46 (39–53)**
		1.284 (3)[b]	**0.733**					
Drop A	6105.938 (2456)	10.661 (1)[c]	0.001	1193.938	−5722.300	0	44 (39–48)	56 (52–61)
Drop C	6108.543 (2456)	13.266 (1)[c]	0.000	1196.543	−5720.997	59 (53–64)	0	41 (36–47)
Negative life events								
Saturated model	6127.723 (2420)			1287.723	−5582.780			
Full sex-limitation	6166.684 (2452)	38.952 (32)[a]	0.185	1264.684	−5677.635	47 (12–58)	01 (0–29)	52 (42–64)
						10 (00–41)	26 (02–40)	64 (52–75)
Scalar sex-limitation	6170.170 (2455)	42.447 (35)[a]	0.181	1260.170	−5868.611	32 (10–50)	10 (00–25)	58 (50–67)
		3.486 (3)[b]	0.323					
Drop A[c]	6178.304 (2456)	8.134 (1)[c]	0.004	1266.304	−5686.117	0	30 (25–35)	70 (65–75)
Drop C[c]	**6171.886 (2456)**	**1.717 (1)[c]**	**0.190**	**1259.886**	**−5689.326**	**45 (38–52)**	**0**	**55 (48–62)**

AIC, Akaike information criterion; BIC, Bayes information criterion; −2LL, minus twice the log likelihood fit statistic; df, degrees of freedom; χ^2, chi-square fit statistic for the comparative fit of the full with the saturated model; M, male; F, female.

Best-fit model is in bold.

[a] Compared with the saturated model; [b] compared with the full model; [c] compared with the scalar model.

Externalizing behavior correlated significantly with MPD ($r = 0.33$, $p < 0.001$; male = 0.30; female = 0.36), PPD ($r = 0.27$, $p < 0.001$; male = 0.19; female = 0.27) and NLEs ($r = 0.47$, $p < 0.001$, male = 0.46, female = 0.48). Parameters could be constrained across sex for all bivariate models without significant reduction in the fit of the model (externalizing behavior and MPD: $\Delta\chi^2 = 11.067$, df = 9, $p = 0.271$; externalizing behavior and MPD: $\Delta\chi^2 = 16.241$, df = 9, $p = 0.062$; externalizing behavior and NLEs: $\Delta\chi^2 = 10.164$, df = 9, $p = 0.337$); therefore, all results are reported on data collapsed across sex. Standardized path coefficient estimates for each of the bivariate genetic models are presented in Fig. 2. The path coefficients were used to estimate the proportion of total covariance between MPD, PPD or NLEs and externalizing behavior due to genes, shared environment and non-shared environment by estimating each as a proportion of the total covariance. Thus, the covariance with MPD due to genes results from: genes (0.54×0.45)/total covariance (genes: 0.54×0.45) + (shared environment: 0.12×0.00) + (non-shared environment: 0.15×0.63), or 61% of the covariance. The shared environment contributes 1% of the phenotypic covariance and the remaining 28% is due to non-shared environmental factors. Finally, we calculated the genetic correlation (rA: the extent to

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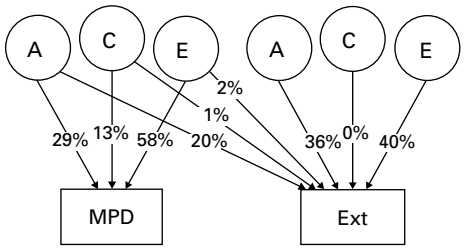
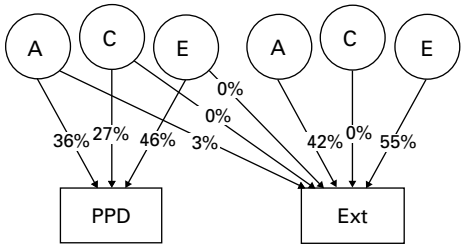
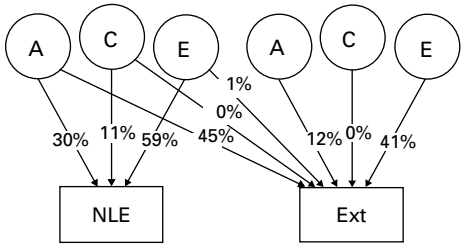


(*a*) Maternal punitive discipline and externalizing behaviour



(*b*) Paternal punitive discipline and externalizing behaviour



(*c*) Negative life events and externalizing behaviour

Fig. 2. Parameter estimates for the Cholesky models. MPD, Maternal punitive discipline; Ext, externalizing behavior; NLE, negative life events. Fit for model (*a*): −2LL = 13878.130, df = 5112, $\chi^2 = 43.954$, df = 58, $p = 0.914$. Fit for model (*b*): −2LL = 13310.417, df = 5112, $\chi^2 = 63.282$, df = 58, $p = 0.295$. Fit for model (*c*): −2LL = 13551.008, df = 5112, $\chi^2 = 60.564$, df = 58, $p = 0.383$.

which the two phenotypes share genetic variation), shared environmental correlation (rC) and non-shared environmental correlation (rE) to be 0.60 (95% CI 0.34–1.0), –1.0 (1.0–1.0) and 0.23 (0.14–0.32) respectively.

The covariance between PPD and externalizing behavior was almost entirely due to genetic covariation (98%), with a very small, non-significant, contribution (2%) from the non-shared environment. The genetic, shared and non-shared environmental correlations between externalizing behavior and PPD were 0.24 (95% CI −0.05 to 0.65), −1.0 (−1.0 to 1.0) and 0.00 (−0.11 to 0.10) respectively.

Finally, the covariance between NLEs and externalizing behavior was also largely due to genetic covariation (77%), with negligible shared (7%) and moderate non-shared environmental (21%) influence. The genetic, shared and non-shared environmental correlations between externalizing behavior and negative life events were 0.88 (95% CI 0.63–1.0), −1.0 (−1.0 to 1.0) and 0.19 (0.09–0.29) respectively.

Interaction effects

Fig. 3 illustrates how the changes in parameter estimates increased on exposure to MPD, PPD and NLEs [full model (panels *a*, *c*, *e*); best-fit model (panels *b*, *d*, *f*)]. The graphs in Fig. 3 show absolute levels of variance for A, C and E, as suggested by Purcell (2000), not the proportion of relative phenotypic variance due to A, C and E, and, as such, estimates for each do not depend on the size of the estimate for the other variance components. There was an interaction between MPD and genetic influences unique to externalizing behavior (β_{XU}; $\chi^2 = 4.64$, df = 1, $p = 0.031$; contact T.M.M.B. for fit statistics for dropping each pathway), and between MPD and both common (β_{ZC}; $\chi^2 = 8.32$, df = 1, $p = 0.004$) and unique (β_{ZU}; $\chi^2 = 37.13$, df = 1, $p = 0.000$) non-shared environment influences. Furthermore, there was an interaction between MPD and the shared environmental influences on externalizing behavior. It was not possible to ascertain whether this applied to the common shared environment (β_{YC}) or the unique shared environment (β_{YU}), because although each could be dropped individually, it was not possible to drop both simultaneously ($\chi^2 = 7.110$, df = 2, $p = 0.029$). For PPD, moderation of both shared and unique genetic effects was significant, while all other interactions were non-significant ($\chi^2 = 3.390$, df = 4, $p = 0.495$).

There was an interaction between NLEs and the non-shared environment path unique to externalizing behavior (β_{ZU}: $\chi^2 = 29.286$, df = 1, $p < 0.001$); all other interactions were non-significant ($\chi^2 = 6.866$, df = 5, $p = 0.231$).

Given that the full siblings appeared to differ significantly from the DZ twins pairs on some socio-demographic and personal characteristics, inclusion of them in the current analyses may have biased the results in some way. Therefore, we conducted all of the described analyses with the twin sample only and found that the results were comparable to those reported here for the complete dataset.

Discussion

Consistent with previous research we found that externalizing behavior was substantially heritable, with a small, non-significant shared environment effect. Although a substantial heritability and non-significant shared environment effect for externalizing behavior is consistent with some studies that use Child Behavior Checklist (CBCL) measures (e.g. Eaves *et al*. 1997), it is not consistent with results from all twin studies (e.g. Edelbrock *et al*. 1995).

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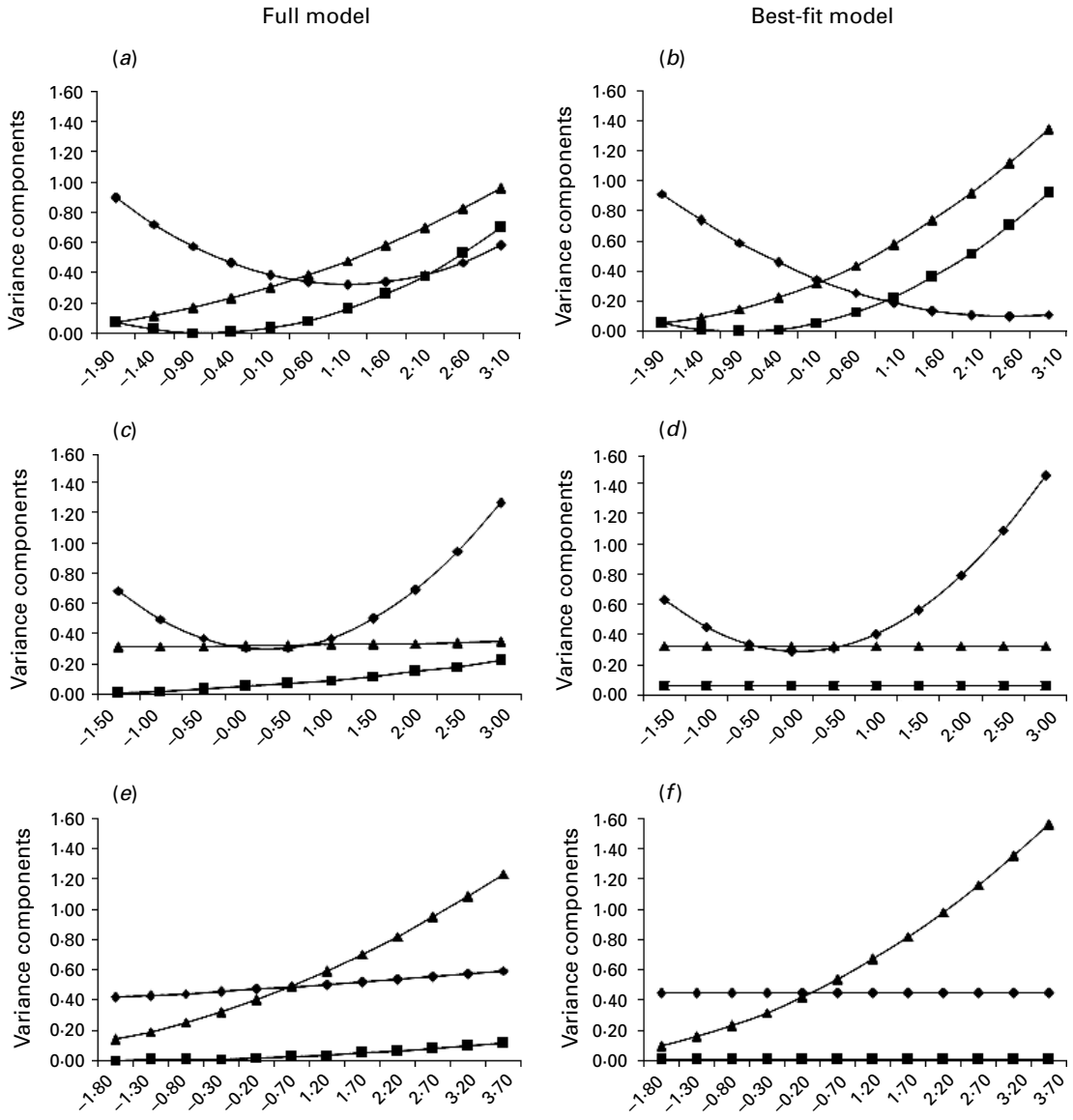

Fig. 3. Estimates of genetic (◆), shared environment (■) and non-shared environment (▲) variance at different levels of maternal punitive discipline (*a*, *b*), paternal punitive discipline (*c*, *d*), and negative life events (*e*, *f*). Left-hand panels show full models (*a*, *c*, *e*) and right-hand panels show best-fit models (*b*, *d*, *f*).

This may, in part, result from the use of self-report rather than parental report of externalizing behavior. Another contributing factor may have been the use of siblings of different ages rather than the sole use of twins, in which, if not controlled for, common age might act as a shared environment effect. Finally, we used the total externalizing scale in these analyses, whereas many other genetic studies use aggressive and delinquent subtypes. MPD, PPD and NLEs were also heritable, with modest (though, in the case of MPD and NLEs, non-significant) shared environment contributions, and the remainder due to non-shared environmental variance.

Parental punitive discipline

MPD and PPD were significantly heritable, indicating gene–environment correlation that may be passive (resulting from the parents providing both genes and the environment) or evocative (in which the externalizing child elicits harsh discipline from the parent; O'Connor *et al*. 1998). This is not to dismiss the possibility of direct environmental causality: 38% of the covariance of MPD with externalizing behavior was due to non-shared environmental influences, which could reflect direct causation of MPD on externalizing behavior. Although the genetic correlation between externalizing behavior and MPD was high, there was

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genetic variance unique to each phenotype. This may in part explain why many adolescents demonstrate externalizing behavior irrespective of levels of MPD. However, genes appeared to account entirely for the correlation between PPD and externalizing across the entire population, indicating that the association was due to common genetic risk.

The common genetic influences were not moderated by exposure to MPD, indicating that externalizing behavior was genetically influenced regardless of MPD levels. However, the unique genetic effects were moderated, implying that some of the genetic risk is contextually dependent, even after controlling for rGE. Genes were more salient in the development of externalizing behavior in those who were exposed to low MPD. These results are consistent with the social push perspective (Raine, 2002), as genes were more salient in the low-risk (low levels of MPD) environment. Furthermore, these results are in contrast to the bioecological model (Bronfenbrenner & Ceci, 1994), as the genetic risk in the current study appears to be greater in the environment least conducive to such behaviors (low levels of MPD), rather than in the environment most conducive to the behavior (high levels of MPD).

For PPD, the common and unique genetic risks for externalizing behavior were moderated by levels of exposure. In this example, genetic risk appears particularly salient in levels of higher exposure to PPD. This finding is consistent with the bioecological model as the genetic risk appears to be greater in the environment most conducive to such behaviors (high levels of PPD). The difference in moderating patterns of MPD and PPD on the genetic risk for externalizing behavior may be due to differences in the way adolescents react to mothers and fathers.

Negative life events

We also found evidence of gene–environment correlation for NLEs, and common genetic variance accounted for most of the covariation between NLEs and externalizing behavior. This may be an example of active rGE; that is, people with an antisocial predisposition are more likely to seek out or elicit environments that increase their exposure to negative events. This account is consistent with results from previous analyses, demonstrating that aspects of adults' personality explained genetic influence on controllable life events (Saudino *et al*. 1997). These findings are also consistent with studies that show how 'risk-taking' behavior contributes to the genetic variation of externalizing behavior (Krueger *et al*. 1994), as such behavior may increase the risk of exposure to NLEs.

These results provide evidence in support of our hypothesis that children's externalizing characteristics increase their risk of exposure to NLE. Although exposure did not moderate the extent to which genes play a role in externalizing behavior, it did moderate the contribution made by non-shared environmental influences, which increased as exposure to NLEs increased. As we had selected only those NLEs that were not necessarily shared by twins, NLEs may potentially be acting in these twins as non-shared environmental effects, accounting for some of the covariation above that explained by common genetic effects. Consequently, as the NLEs become more prevalent, they may be responsible for the apparent increased contribution of the non-shared environmental influences to the variance of externalizing behavior.

Although the absolute level of genetic risk remained unchanged in the rGE–G × E model, the proportion of the phenotypic variance attributable to the genetic risk did vary. Therefore, the increase in the variance of the non-shared environment, resulting from moderation of the NLE levels, will necessarily reduce the proportion of variance accounted for by genetic risk. This might be explained by the social push perspective (Raine, 2002) because the relative genetic risk decreases as the 'environment' becomes more severe, despite the absolute level of genetic risk remaining constant.

Limitations

These findings should be considered in the context of a number of limitations. The age range of the participants is fairly large (12–21 years) and encompasses a broad developmental range. Consequently, we might expect different etiological influences or mechanisms for the association between phenotypic outcomes and their correlates at different ages. At age 12, for example, adolescents with externalizing problems may have fewer opportunities to expose themselves to situations where negative events occur than older adolescents, and are also more likely to be reprimanded within the family. Although we did our best to account for age effects on the means of our measured variables, future research might benefit from looking at differences over age. Another limitation of this study was the exclusive use of self-report data, which may have led to an inflation of associations between measures. In addition, no paternity analyses have been performed in the G1219 study, and thus it is possible that some half-sibling pairs have been included as full pairs, which may have resulted in inflated estimates of genetic variances for each outcome. Furthermore, non-paternity might be associated with a parenting style that could result in greater

parenting problems in the full siblings than the identical or even DZ twins, for which shared paternity is highly likely. However, it is unclear what effect such an association might have on the reported results, and there is no evidence in the current study to suggest that full siblings receive more punitive discipline than DZ pairs.

Another concern of this study is that the variance of externalizing behavior for female MZ twin pairs was slightly, but significantly, lower than the variance for the other females in the sample, resulting in a bad fit of the full univariate externalizing behavior model. However, in large samples there is often power to detect small differences that may not be meaningful. As the AIC and BIC were lower in the full model compared with the saturated model, it appeared that full model could not be rejected. Furthermore, the difference in variance for MZ female twins compared with the other female pairs may indicate that there are sibling interaction or competition effects for female externalizing behavior. However, it was not possible to test this explicitly in the current model.

Finally, the externalizing behavior scale and the NLEs were somewhat skewed; skewed data may mimic G × E effects (Purcell, 2002), although the effects are reduced when the moderator is also included in the model. Transforming data can remove true interaction effects (Falconer & Mackay, 1996). Given the sensitivity of G × E analyses to scale, we opted *a priori* to use the scales in their regressed form, rather than transforming them. However, non-normal distribution of data is a possible limitation of the current study.

Although the results of this study show that variations in the levels of MPD and PPD are associated with changes in the genetic variance for externalizing behavior, we cannot directly compare these result with the adoption and molecular studies reported previously (Cadoret *et al*. 1995; Caspi *et al*. 2002). The previous G × E analyses assessed the interactive contribution of genetic and environmental risk to changes in mean levels of behavior, whereas the current analyses assess changes in the variance components of externalizing behavior as a function of MPD and PPD.

Despite these caveats, these results highlight the complex relationship between people and their surroundings in the development of behavior. We can no longer just assume that the environment influences the way people act 'above and beyond' genetic influences. Instead, exposure to certain environment may result from a genetic risk, and the magnitude of the genetic risk may in turn be dependent on the level of environment exposure.

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Declaration of Interest

None.

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