Disentangling Child and Family Influences on Maternal Expressed Emotion toward Children with Attention-Deficit/Hyperactivity Disorder

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ABSTRACT

Objective: We used multi-level modelling of sib-pair data to disentangle the influence of child-specific and shared family influences on maternal expressed emotion (MEE) towards children and adolescents with attention-deficit/hyperactivity disorder (ADHD). **Method:** MEE was measured for 60 ADHD proband-unaffected sibling pairs aged 6-17 years using the Five Minute Speech Sample. Questionnaire measures of conduct and emotional problems were collected for children and measures of depression and ADHD were collected for mothers. Multi-level models partitioned the effects of 5 MEE components (Initial Statement –IS, Relationship – REL, Warmth – WAR, Critical Comments – CC and Positive Comments – PC) into (i) child-specific and (ii) shared family effects. **Results:** Significant child-specific influences were confirmed for all MEE components, with higher levels of MEE expressed toward probands than siblings. For REL, PC, and CC, this effect was explained by comorbid conduct problems rather than ADHD. Only low WAR was associated with ADHD itself. Furthermore, only low WAR showed shared familial influences which were in part accounted for by maternal depression. **Conclusions:** MEE towards ADHD children was influenced by child-specific factors. For most components these were driven by comorbid symptoms of conduct problems rather than ADHD itself. WAR was different – it was influenced by both child-specific and shared familial effects and the child effects showed no relation to conduct problems. Further studies utilising a longitudinal design are required to establish the direction of causation and extend our understanding of the relationship between EE components and ADHD.

INTRODUCTION

Variations in patterns of family interaction moderate the course and outcome of ADHD.[1] Expressed emotion (EE) is a measure of the level of criticism and/or emotional involvement expressed by parents (or family members) toward their children (or relatives) that may be implicated in some of these effects.[2] Traditionally, individuals are classified as high in EE if they make more than a specified threshold number of critical comments or show any signs of hostility or marked emotional overinvolvement[2]. In general, parental EE shows both concurrent [3-5] and longitudinal[6] associations with internalizing and externalizing problems in child and adolescent community and clinical samples. More specifically, parental EE (especially criticism) is associated with their children's ADHD.[7,8] For example, criticism was strongly associated with both subtypes of ADHD in both clinic- and community-based samples[6]. The association between child ADHD and parental EE might be important clinically. This is because EE may drive some aspects of negative developmental trajectories associated with ADHD. Low levels of warmth and high levels of criticism at age seven were found to predict the extent to which children with ADHD developed conduct disorder.[9] Therefore, if the causes of high EE could be targeted then, in principle, developmental outcomes for ADHD children could be improved. Unfortunately, little is known about what causes high EE in ADHD. For instance, it remains uncertain whether high levels of EE directed towards the ADHD child represents (a) a general tendency of the parents of ADHD children to be critical that affects all members of their family (whether ADHD or not) or (b) a child-specific response elicited by the characteristics of the individual ADHD child. Treatment trials suggest that parental negativity is at least in part a response to the behavior of the ADHD child: medication-induced reductions in symptoms are associated with reductions in parental negativity during interaction.[10-12] However, these studies do not measure the extent to which such short-term changes would be related to more general reductions in patterns of hostility and criticism indexed by EE or the extent to which family dynamics or parental characteristics influenced either initial levels of hostility or the treatment effects.

One way to address this question is to compare different parental responses to ADHD probands and unaffected siblings within the same family. EE is especially useful for this as it can be reliably estimated separately for each child.[13] Such a design allows the influences on parental EE associated with differences between families to be disentangled from child-specific effects within families. Multilevel modelling approaches to such proband-sibling data nested within families allow the relative contribution of each to be estimated. Furthermore, they provide estimates of the extent to which specific characteristics of the ADHD child and the family explain these effects. This is important because it remains uncertain whether it is ADHD per se or

other aspects of the ADHD child's make-up that are associated with high parental EE. A study using multiple informant categorical and continuous measures of ADHD and conduct disorder (CD)/oppositional defiant disorder (ODD) demonstrated that not only was the EE-ADHD association stronger than the EE-CD/ODD link, but the EE-CD/ODD relationship did not withstand control of ADHD.[6] These findings are supported by an earlier study in which comorbid CD/ODD was not related to high parental EE.[14] In contrast, another study reported that the association between ADHD and maternal high EE was due to co-occurring conduct and emotional problems.[8] In terms of putative shared familial influences on high EE, there are a number of obvious candidates to explore. There is a high prevalence of psychological problems in the parents of children with ADHD, especially adult ADHD itself[15], but also depression, anxiety[16] and aggression.[17] Maternal depression is associated with parental criticism and hostility[18] and aggression in parents is linked to poor parenting practices.[19]

The final question to be addressed relates to the extent to which different aspects of EE show a similar set of shared familial and child specific influences. Previous studies have suggested only moderate correlations between initial statement (IS), warmth (WAR), relationship (REL), critical comments (CC) and emotional over involvement (EOI)[7]: EE elements overlap to some degree, but each has the potential to afford discrete information. This leaves open the possibility that different components may be independently related to shared-familial and child-specific effects. For instance, it can be hypothesized that EE components most directly linked to a response to challenging behavior (e.g., CC) would be most strongly subject to child-specific influences while other aspects of EE (e.g., WAR) could be affected by shared familial influences. Maternal depression has been linked specifically with low parental WAR[20] and low WAR in depressed parents may be linked to increased rates of externalizing child behaviour.[21]

The current study had the following aims: (i) to estimate the size of shared familial and child-specific influences on maternal EE towards ADHD probands and their unaffected siblings; (ii) to identify whether any child-specific effects found were due to ADHD or to other child characteristics; (iii) to examine the role of parental mental health in any shared familial effects found; (iv) to explore whether different components of parental EE had different patterns of child-specific and shared familial influence.

METHOD

Participants

Participants were children/adolescents with a clinical diagnosis of DSM-IV combined subtype ADHD (ADHD-CT), their unaffected siblings and their parents from the Southampton arm of the International

Multicentre ADHD Genetics Project (IMAGE).[22] The identification of participants, recruitment procedures and entry criteria are described in detail elsewhere.[23] Families were excluded from the current analysis if siblings had ADHD-CT (7) or were twins (2) or if EE data were missing (3). The final sample comprised 60 proband-sibling pairs [mean proband age 12.22 years (range 7.58-16.91 years) and sibling age 11.54 years (range 5.83-17.08 years); 52 probands and 28 siblings were male].

Measures

DSM-IV diagnoses. ADHD-CT diagnosis was made using the Parental Account of Childhood Symptoms interview (PACS)[24] and the ADHD subscale of the Conners Teacher Rating Scale Revised Long Version (CTRS-R: L),[25] using a procedure which has been described in full previously[23].

Maternal expressed emotion (MEE). This was measured using the Five Minute Speech Sample (FMSS).[26] Parents are instructed to talk uninterrupted for 5 minutes about each child, including their thoughts and feelings toward, and their relationship with, their child. Speech samples were transcribed verbatim and coded by a trained rater using a modified coding scheme designed for child populations.[7] Ratings were made on five components – Initial Statement (IS) and Relationship (REL) were rated as positive, neutral or negative, and Warmth (WAR) was rated high, moderate or low. Critical Comments (CC) and Positive Comments (PC) were frequency counts. The coding scheme has good code-recode, inter-rater and test-reliability, in addition to satisfactory construct and discriminant validity.[7]

Child conduct and emotional problems. Conduct problems (CP) were measured using the relevant subscales of the parent form of the Conners Rating Scales-Revised Long Version (CRS-R: L)[25] including ODD and CD-related symptoms each rated on a 4-point Likert scale. Emotional problems (EMO) were measured using the parent form of the Strengths and Difficulties Questionnaire (SDQ)[27] including 5 items scored on a 3-point Likert scale. Both measures have good internal reliability coefficients, test-retest reliability, and discriminatory power.[28,29]

Maternal ADHD and depression symptoms. ADHD was measured using mothers' self-reports on the Current Symptoms Scale Self-Report Form (CSS-SR),[30] an 18-item scale is based on the DSM-IV. It has also been shown to be correlated with spousal, parental and cohabiting partner ratings of symptoms.[31] Depression was measured with the General Health Questionnaire 12-item version (GHQ-12)[32] which has been extensively used as a short screening instrument to identify depression. This version contains 12 items with a 4-point scale. Both measures have sound psychometric properties.[33,34]

Procedure

Informed consent and assent were obtained from parents and children/adolescents respectively prior to participation. The research assessment was conducted by two trained researchers at child and adolescent mental health service outpatient clinics or at the homes of families. Mothers completed two separate FMSS; one for the ADHD proband and one for the sibling. FMSS was completed prior to administration of the PACS. Questionnaires were completed by the mother for each child and herself. Ethics approval was obtained from university and National Health Service ethics committees.

DATA ANALYSIS

Separate multilevel models were run for each of the five EE components using HLM 6 Hierarchical Linear and Nonlinear Modelling.[35] Variance in EE was partitioned into Level 1, child-specific effects, and Level 2, shared family effects. Level 1 (child-specific) effects represent the extent to which differences between children in their characteristics can explain levels of MEE expressed by an individual mother. Given the design of the current study, this relates to the effect of ADHD on MEE within families (as in each sibling dyad included in the analysis, there is one ADHD child and one unaffected sibling) and at the same time other child factors which may differentiate the ADHD child from the non-affected sibling. Level 2 effects represent the extent to which differences between families can explain the levels of MEE expressed by different mothers. In other words, this relates to how factors that differentiate families from one another can explain variance in average MEE levels expressed by mothers to their two siblings within families. Clearly, such between-family Level 2 effects cannot be due to child ADHD (as all families on average have the same levels of ADHD because of the proband-unaffected sibling design (.5 of a case). However, these between-family effects could be related to other child factors (e.g., the average level of child comorbidities in each family) or maternal characteristics such as mothers' depression. Predictor variables were also categorized into child-specific (Level 1) and family-related (Level 2) predictors depending on whether the unit of analysis was the individual child (e.g., EP, EMO) or the family (maternal depression, sibship gender composition, averaged child comorbidities across siblings). For child-specific predictors, each child within the family had a unique score. For family-related predictors, both children in the family have the same score. We adopted a three stage approach.

First, unconditional models in which no individual child or family predictors were included in the model were run to test whether maternal EE was related to within-family child-specific effects or shared family effects in general. These models provide information on the total variance in MEE within families (σ^2) and between

families (τ_{00}). Intra-class or "intra-family" correlation coefficients ($\rho = \tau_{00} / (\tau_{00} + \sigma^2)$) were calculated for each MEE component. The intra-class correlation coefficient (ICC) represents the degree of resemblance of levels of MEE for children in the same family as compared with levels of MEE from different families. In other words, it is the proportion of the total variability in MEE that is attributable to the families.

Second, for those EE components with a within-family effect, suggesting child-specific influences, we fit a series of models to which child-specific predictor variables (ADHD diagnostic status, CP and EMO) were added as fixed effects to test whether maternal EE was associated with comorbid problems rather than ADHD per se. In particular, we compared the model with ADHD status entered as the only predictor and the full model with all predictors entered. Child-specific dichotomous predictor variables, namely ADHD diagnostic status and gender were uncentred and continuous predictor variables; age, EMO and CP were added group mean centred.

Third, for those EE components for which between-family differences were found (suggesting shared familial effects for the two siblings), we added family-related predictor variables (maternal ADHD and depression and averaged child characteristics such as comorbidities across siblings). Sibship gender composition (a family-related predictor variable) was coded into dummy variables when entered into the model at the family-level (Level 2) with all boy sibships categorized as the control group. Categorical family-related predictor variables (i.e., sibling-dyad gender composition) were added to the models uncentred and continuous family-related predictor variables (i.e., mean sibship EMO, mean sibship CP, maternal ADHD and maternal depression) were entered grand mean centred.

RESULTS

EE components were all significantly though moderately associated with one another in the expected direction (r's >.25 - .54; P<0.001). The relative proportion of variance accounted for by within-family (i.e., child-specific) and between-family differences (i.e., shared family effects) differed for EE components (refer to Tables 1, 2 and 3 – M-0). For REL, PC, CC and IS there was little or no contribution of shared family effects marked by differences between families and a strong contribution of child-specific effects (variance accounted for by child-specific effects was: 100% - REL; 93% - PC; 94% - CC; 91% - IS). In contrast, for WAR the shared family effects predominated, although there were significant child-specific effects too (70% of WAR variance accounted for by shared family effects and 30% by child-specific effects). Initial ADHD status was associated with IS, REL, CC, and PC, however, this effect was no longer significant when all other predictors were entered (refer to Table 1 and 2 – M-2). In the final model, CP was related to CC and REL, with a marginal effect of this

factor on PC. For these components children with high CP had more critical parents. Altering the order of entry of the variables had no effect on the results.

<< Insert Table 1 and Table 2 >>

Given the joint influence of within- and between-family effects for WAR, both child-specific (as above) and family-based predictors (e.g., sibship gender composition, mean levels of sibship comorbidities and maternal mental health) were entered into the model. As for the other components, child ADHD status had a significant effect on WAR (refer to Table 3 – M-1). Gender and age also had significant effects. The effect of ADHD on WAR remained significant when controlling for gender, age, EMO and CP (refer to Table 3 – M-2). Of the between-family factors (shared by the two sibs), there was no effect of sibship gender composition; significant predictors included mean sibship CP and maternal depression (refer to Table 3 – M-3). Maternal ADHD was marginally associated with lower WAR. Adding these effects did not alter the effects of child-specific measures of ADHD.

<< Insert Table 3 >>

DISCUSSION

Previous studies have highlighted the potential importance of parental EE (low warmth and hostility/criticism) as a putative driver of negative developmental outcomes in ADHD.[9] In the reported study, we attempted to gain further insights into the causes of parental EE in ADHD by using a sib-pair design and multilevel modelling to disentangle the influences of child-specific influences and shared family effects. The analysis provided a number of new and important insights.

First, for the majority of EE components child-specific effects predominated – with little or no role for shared family influences on IS, REL, PC and CC. Levels of hostility and criticism were much higher for ADHD probands than their unaffected siblings within families while there were few systematic differences in average MEE between families. These patterns of results are consistent with the notion that high MEE is predominantly a "child effect" and are congruent with previous research[8] including treatment trials research.[10-12] Also consistent with this view, Lifford and colleagues[36] found that boys' ADHD symptoms impacted on mother-son hostility both concurrently and longitudinally, but there were no effects in the other direction.

Second, when we looked specifically at which child characteristics appeared to be driving the effects for these components it appeared that it was CP rather than ADHD. While when ADHD was entered alone it was significantly associated with these components, this effect was lost when other predictors were added. In fact in

the final models, high EE was more likely to be predicted by CP than ADHD. These results add to a rather mixed picture with regard to the relative roles of ADHD and CP in "driving" high EE. Marshall and colleagues[14] found that parental EE status was not related to CP in samples of ADHD and ODD patients. In addition, a more recent study showed that EE was more strongly associated with ADHD than CD/ODD in that the EE-ADHD relationship held when CD/ODD was controlled, but the opposite was true for the EE-CD/ODD link – i.e., this association did not withhold control of ADHD.[6] In contrast, another study found that parental criticism was unrelated to ADHD when CP was taken into account[8]. More general studies of parenting are also mixed. In some, CP exacerbates and is more strongly associated with less effective parenting practices than ADHD,[37, 38] while others find little support for this trend.[39] Disentangling these child-specific effects is made difficult due to the strong association found between ADHD and CP. Longitudinal studies suggest the relationship between EE, ADHD and CP may be more complex and transactional. For instance, Taylor[9] has suggested that ADHD symptoms elicit hostility from parents which in turn leads to the development of CD/ODD later on. Recent data suggests that certain ADHD children might be especially genetically vulnerable to these effects.[40]

Third, WAR could be differentiated from the other components in terms of both the relative importance of between- and within-family effects and the specific predictors that accounted for these effects. WAR differed markedly both between families and within families. This is consistent with the idea that although parent/family effects may have a more influential role in predicting WAR than they do on other EE components, child-specific factors associated with ADHD also make a salient contribution. This novel finding was possible because of the methodological perspective taken in the study – i.e., including WAR in our FMSS measure and using a proband-sib design. Consistent with our results, some studies using parenting scales have shown that the parents of ADHD children show less warmth and involvement with their children compared to control children.[41] WAR also differed from the other components in as much as ADHD status was the key child-specific correlate instead even when other factors including CP were added to the models. The notion that while hostility and criticism (as measured by REL and CC) are related to ODD/CD but WAR is related to ADHD specifically was a surprising finding.

Maternal depression symptoms significantly predicted low WAR accounting for differences in average WAR between families, a finding consistent with the literature on EE and parenting in general. Parental depression is associated with decreased warmth expressed toward children and negative parent-child relationships.[20,21] More generally, maternal depression is a strong predictor of EE in families of children with

disruptive behaviour disorder and obsessive compulsive disorder.[42] In contrast, one study found that associations between ADHD and depression in mothers did not remain significant when child behaviour was entered into the regression models.[8] Children in ADHD families in which mothers are depressed may be more vulnerable to low levels of warmth. Brennan and colleagues [43] found that children were more resilient to negative outcomes if they perceived their depressed mothers as being warmer. There was also a marginal effect of maternal ADHD on WAR that was independent of depression with high ADHD scorers having higher WAR; what seems like a counter-intuitive effect. ADHD in the past has been shown to be related to increased negligence and tolerance, lower levels of involvement and positive parenting and increased levels of inconsistent and permissive parenting.[19,39,44,45] These findings were echoed by Psychogiou and colleagues[46]. However, Psychogiou and colleagues[46] also showed that high levels of ADHD symptoms in mothers of children with ADHD led to higher WAR. The authors speculated that high-ADHD mothers may have increased empathy and tolerance for the high-ADHD child.

The study had a number of limitations. First, the data were cross-sectional in nature which impedes the examination of cause-effect relationships. Future studies employing a longitudinal design are required to enable concrete causal inferences to be made. Second, EE was assessed in mothers only. In dual-parent households, maternal EE alone may not reflect *parental or family EE* as it does not take into account fathers' EE and the similarities and differences in maternal and paternal EE. Furthermore, the inclusion of only one parent's EE and the exclusion of all other family members' EE is also unlikely to capture EE as it was intended, that is, as a broader index of the family environment. Third, although teacher ratings of child EMO and EP were obtained for the original sample, these ratings were not used in the multi-level analysis due to the considerable amount of missing data for these variables. In addition, mothers reported on their own ADHD and mood symptoms. Mothers may have over-reported their own and their child's symptoms. Fourth, no measure of personality was included in the analysis. Findings in relation to EE in adult psychiatric patients have found that personality is an important factor contributing to high-EE, more so than psychopathology of the relative or parent. High-EE relatives tend to have a more internal locus of control for their own behaviour than low-EE relatives and therefore prefer to take control in managing their own life problems and difficulties.[47]

Even with these limitations, the findings of the study may have important therapeutic implications. First, it may be beneficial to routinely screen parents of ADHD children for high EE. Second, interventions that aim to reduce parental criticism and hostility may benefit from including a combination of behavioral strategies that enable both child and parent to more effectively manage ADHD symptoms (and comorbid

oppositional/aggressive symptoms) and techniques specifically aimed to reduce criticism. Introduced at an early age, interventions that reduce criticism and improve parent-child interactions may prevent or reduce the risk of the development or escalation of ODD/CD later on.[9] Treatment studies have demonstrated significant improvements in clinical outcomes of adult patients with anorexia nervosa, bipolar disorder, and schizophrenia when families received interventions designed to reduce aspects of high-EE behaviour.[48-50] By and large, such family-based interventions typically involved education about the illness, improving communication skills and problem solving. Our findings suggest that interventions that target parental WAR may warrant a different therapeutic approach. However, further research is called for to better understand the different domains of EE. Future studies need to assess the impact of the different components of high EE on the course of ADHD and its co-occuring psychopathology.

In conclusion, our results suggest that maternal EE is an important marker of parental response to ADHD. However, it may be important to differentiate WAR from other EE components (IS, REL, CC, PC) in terms of the relative role of child-specific and shared family effects and in terms of the specific role played by ADHD as opposed to CP. Further studies utilising a longitudinal design, however, are required to establish the direction of causation between high EE, ADHD and CP as well as maternal mental health.

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Table 1: Fixed effects and variance estimates for the prediction of IS and REL for model zero with no specific predictors, model-1 with ADHD as a specific predictor and the final model.

	IS			REL		
	M-0	M-1	M-2	M-0	M-1	M-2
Intercept	1.88 *.05*	1.73 *.07*	1.88 *.11*	1.75 *.06*	1.47 *.08*	1.76 *.13*
Child-specific level						
ADHD		.28** *.10*	.14 *.17*		.57** *.12*	.09 *.19*
Gender			-.23 *.12*			-.16 *.13*
Age			-.00 *.03*			.03 *.03*
EMO			-.05 *.03*			.01 *.04*
CP			.01 *.01*			.02* *.01*
Variance estimates						
Child effects	.29** *.05*	.31** *.04*	.29 ** *.04*	.48** *.09*	.41** *.05*	.38** *.05*
Shared-family effects	.03 *.04*	-	-	.00 *.06*	-	-
-2* log likelihood	205.4	198.4	193.3	254.3	232.7	223.3

Note. Standard errors are in italics. M-0 – model with no child-specific characteristics estimated; M-1 is model 0 plus ADHD status; M-2 is the final model with all child-specific predictors added. No shared family estimates are given for Models 1-2 as random effects were not included in these models because the unconditional model revealed that between-family variance was not significant – the EE component did not differ significantly from one family to another. IS = initial statement; REL = relationship; EMO = emotional problems; CP = conduct problems. *p < .05, **p < .01, † marginally significant.

Table 2: Fixed effects and variance estimates for the prediction of PC and CC for model zero with no specific predictors, model-1 with ADHD as a specific predictor and the final model.

	PC			CC		
	M-0	M-1	M-2	M-0	M- 1	M-2
Intercept	3.15 *.28*	4.38 *.35*	3.38 *.55*	2.20 *.25*	1.07 *.30*	1.83 *.47*
Child-specific level						
ADHD		-2.5** *.49*	-.67 .82		2.3** *.43*	.68 *.71*
Gender			.31 *.57*			.09 *.49*
Age			-.08 *.14*			.05 *.12*
EMO			-.15 *.16*			-.10 *.14*
CP			-*.06*† *.03*			.08***.03*
Variance estimates						
Child effects	8.2** *1.50*	7.3** *.95*	6.9** *.88*	6.4** *1.17*	5.5** *.71*	5.2** *.66*
Shared-family effects	.66 *1.15*	-	-	.39 *.88*	-	-
-2* log likelihood	602.0	579.7	571.5	570.6	545.7	537.2

Note. Standard errors are in italics. See Table 1 for description of M-0 – M-2. CC = critical comments; PC = positive comments. *p < .05, **p < .01, † marginally significant.

Table 3: Fixed effects and variance estimates for the prediction of WAR

Parameter	M-0	M-1	M-2	M-3
Fixed effects				
Intercept	1.76 *.09*	1.65 *.09*	1.58 *.11*	1.66 *.12*
Child-specific level				
Child ADHD		.22* *.07*	.21† *.11*	.22* *.11*
Child gender			.20* *.10*	.23† *.13*
Child age			.04* *.02*	.04* *.02*
Child EMO			.02 *.02*	.02 *.02*
Child CP			.00 *.00*	.00 *.00*
Family-specific level				
Mixed-gender sibship				-.15 *.17*
All-girl sibship				-.16 *.39*
Mean sibship EMO				-.01 *.04*
Mean sibship CP				.02* *.01*
Maternal depression				.85* *.37*
Maternal ADHD				-.01† *.01*
Variance estimates				
Child-effects	.16** *.03*	.13** *.02*	.12** *.02*	.12** *.02*
Shared-family effects	.36** *.08*	.37** *.08*	.38** *.08*	.31** *.07*
-2* log likelihood	221.9	212.3	204.2	192.7

Note. Standard errors are in italics. M-0 – model with no child-specific characteristics estimated; M-1 is model 0 plus ADHD status; M-2 is model 1 plus other child-specific predictors; M-3 is the final model with all child-specific predictors and shared family predictors added. WAR = warmth; *p < .05, **p < .01, † marginally significant.