

## Family settings and children's adjustment: differential adjustment within and across families<sup>†</sup>

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**Background** Children in stepfamilies and single-parent families exhibit elevated levels of behavioural and emotional problems compared with children in intact (biological) families, but there is variation within and across these family types.

**Aims** To examine the sources of variation in children's behavioural and emotional problems across diverse family settings.

**Method** Levels of behavioural and emotional problems in children from diverse stepfamilies and single-parent families were compared with children living with both biological parents. Psychosocial risks were measured at the individual child and family levels.

**Results** Behavioural and emotional problems were elevated in children in stepmother/complex stepfamilies and single-parent families, but not in simple stepfather families, relative to 'biological' families. Psychopathology associated with family type was explained by compromised quality of the parent-child relationship, parental depression and socio-economic adversity. Sibling similarity in behavioural and emotional problems was most pronounced in high-risk family settings.

**Conclusions** Family type is a proxy for exposure to psychosocial risks; the extent of family-wide influence on children's development may be strongest in high-stress settings.

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Membership of a single-parent family or stepfamily is a robust correlate of psychopathology in children (Hetherington *et al*, 1998). To date, the central focus of research has been to explain between-family variation, that is, why children in different families exhibit such diverse outcomes according to family type membership. Studies of this kind suggest that variation in children's behavioural and emotional problems can be attributed, in large measure, to such factors as parent-child relationships, parental mental illness and socio-economic status (Amato & Keith, 1991; Hetherington & Clingempeel, 1992; Dunn *et al*, 1998). Less well understood are the sources of within-family variation: why are children in the same family so different from one another? Findings from behavioural genetics alerted researchers to the substantial differences in sibling adjustment (Plomin & Daniels, 1987). Highlighting sibling differences or within-family variation in children's behavioural and emotional problems was important for several reasons. First, it was clear that an important source of variation in children's adjustment had been ignored in prior research. Second, it forced re-consideration of the assumption that many psychosocial risks operated on a family-wide basis (and therefore affected siblings similarly). Third, it opened up new avenues to the study of resilience, because there was no certainty that risk processes explaining between-family variation in children's behavioural and emotional problems were the same as those that explained within-family variation.

A study of diverse family types provides an ideal context for assessing both the magnitude and the sources of between- and within-family variation in child psychopathology. The research design and data

analyses allowed us to distinguish two opposing views on the impact of risks on children's psychopathology. On one hand, we examined the degree to which psychosocial risks, including family type, operated on a family-wide basis. On the other, we examined the hypothesis that psychosocial risks operated in a child-specific manner and, furthermore, that there would be significant within-family variation in children's behavioural and emotional problems across family types. The key conceptual and methodological point here is that a risk that is presumably 'shared' by siblings – membership of a non-traditional family – may have systematic yet differential effects on siblings.

## METHOD

### Subjects

The Avon Brothers and Sisters Study (ABSS), a sub-study within the community-sample-based Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC; Golding, 1996), is a longitudinal investigation of children's adjustment in different family types. The intention of the ABSS sampling design was to select 50 families of different types from the larger community sample. Data for the current study are based on the first wave of data from the ABSS, including 50 non-step or biological families ( $n=113$  children), 49 stepfather families ( $n=122$  children), 45 stepmother or complex families ( $n=123$  children), and 48 single-parent families ( $n=106$  children); the sample size in analyses below, 453, differs slightly from the total sample size because of missing data.

We included families with two or more children in the sampling procedure because we were especially interested in the differences between siblings in adjusting to family transitions. The representativeness of families within each of the four family types in ABSS was suggested by the finding that ABSS families did not differ from families of the same type in the large community sample in terms of maternal education, income and child adjustment. Overall, 83% of families who were eligible (based on the above criteria) agreed to participate. The age of the youngest child was 4.8 (s.d. 0.38) years and the age range of the siblings was 6–17 years (mean age 10.2 years, s.d. 2.9). The sample included a range of socio-economic levels.

<sup>†</sup>See editorial, pp. 93–94, this issue.

## Procedures and measures

The ABSS study began when the target child was approximately 4.5 years old. Parents, step-parents and children over the age of 7 years (i.e. older siblings of the target child) completed questionnaire and interview measures in the family home; interviews were conducted by trained research assistants.

### Children's psychopathology

Parents completed the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), a revised version of a widely used measure of behavioural problems for young children (Elander & Rutter, 1996). The SDQ has several behavioural problem sub-scales, but these sub-scales were combined in a 'total difficulties' score because of the similarity in results at the sub-scale level and because the scales are moderately to highly intercorrelated.

### Socio-economic status

The highest level of education achieved by mothers was categorised into three commonly used distinctions (no/minimal qualifications, school-leaving qualifications and university degree). Income was measured on a five-point scale (1, less than £100 per week; 5, over £400 per week).

### Maternal depression

Emotional well-being was reported by mothers using a modified version of the Malaise Index (Rutter *et al*, 1970), a self-report measure of depressive symptoms ( $\alpha=0.81$ ). This measure has been used extensively in previous research.

### Parent-child relationship

Parent-child relationship measures were adapted from previously validated scales (Hetherington & Clingempeel, 1992; Dunn *et al*, 1998). Frequency of parent-child conflict was measured using a modified version of the Daily Routines scale ( $\alpha=0.86$ ). Frequency of talking to each child about what he or she had done wrong, explaining rules and compromising, was measured using a modified version of the Communication About Discipline scale ( $\alpha=0.68$ ). Frequency of different types of punitive discipline techniques, such as

yelling at or ridiculing the child, was measured using a modified version of the Negative Sanctions scale ( $\alpha=0.66$ ). Expressive and instrumental involvement with each child was measured using the Expression of Affection scale ( $\alpha=0.83$ ). Reports of enjoyment of spending time with each child were assessed from the positive factor of the Parent-Child Relationship scale ( $\alpha=0.90$ ); parental criticism and nagging of the child were assessed using the negative factor from the Parent-Child Relationship scale ( $\alpha=0.82$ ).

These six parenting scales were subjected to principal components analyses (varimax rotation). Two factors emerged with eigenvalues greater than one, explaining 59% of the total variance. The negativity factor (coercion, hostility, punitiveness) was composed of the negative scale from the Parent-Child Relationship measure, the Negative Sanctions scale and the Daily Routines scale ( $\alpha$  for the factor scales=0.76). The positivity factor (warmth, supportiveness, enjoyment of the child) included the Expression of Affection scale, the positive scale from the Parent-Child Relationship measure and the Communication About Discipline measure ( $\alpha$  for the factor scales=0.68).

### Family type and biological relatedness of parents and children

Four family types were defined:

- (a) *Biological families* are those in which both parents are biologically related to all children.
- (b) *Simple stepfather families* are those in which the father is not biologically related to at least one child but the mother is biologically related to all children.
- (c) *Stepmother/complex stepfamilies* are those in which the mother is not biologically related to at least one child in the family or both the mother and father are not biologically related to at least one child in the family.
- (d) *Single-mother families* are those in which the family is headed by a non-married, non-cohabiting woman.

The rationale for using this coding of family type was based on several considerations. First, most research on stepfamilies to date has considered simple stepfather families, and we wished to have a comparable group of stepfamilies to compare with previous studies. Second, simple stepfather

families constitute the vast majority of stepfamilies (Haskey, 1994). Third, there are important empirical differences between simple stepfather and other stepfamily types (see below). In addition to measuring family type, we also included a measure of biological relatedness between parent and child. This measure is not redundant with family type, because within stepfamilies children varied in their biological relatedness to the father.

### Data analyses

An important methodological limitation of most studies of the risk and protective factors for children's psychopathology is that only one child per family is selected. The consequence of this is that the effects attributable to family-level factors (e.g. parental psychopathology), individual child-level factors (e.g. age, gender) and the interaction between the two are completely confounded. A novel feature of the study was the use of multi-level modelling (Bryk & Raudenbush, 1992; Goldstein, 1995; Goldstein *et al*, 1998), an analytic approach that capitalises on the nested or hierarchical structure of family data. This approach partitions variation attributable to each 'level' in the data structure, which in the current case are the family level (which we refer to as *between-family* variation) and the individual child level (which we refer to as *within-family* variation).

Three features of the multi-level model results are highlighted in Table 1. In the top section, we present the fixed effects associated with the predictor variables. These estimates and standard errors are interpreted as in a regression model; an estimate that is approximately twice its standard error has a significant ( $P < 0.05$ ) association with child psychopathology. In the 'random effects' section, error variance is decomposed into family-level (between-family) and individual child-level (within-family) variability. Estimates for the fixed and random effects are simultaneously calculated using a maximum likelihood procedure, the value of which is also presented. It is important to note that the estimates included in the random effects section of the table are not interpreted in the same way as the estimates for the fixed effects. The estimates in the random effects section are estimates of variance.

For the fixed and random effects parameters, family type was dummy coded with biological (or non-step) families as the

**Table 1** Prediction of child psychopathology: fixed and random effects from multi-level analysis (standard errors in parentheses)

	Model 1	Model 2	Model 3	Model 4	Model 5
<b>Fixed effects</b>					
<i>Family-level variables</i>					
Simple stepfather	0.22 (0.15)	0.22 (0.13)	0.09 (0.22)	-0.01 (0.13)	-0.03 (0.12)
Stepmother/complex	0.52 (0.15)	0.52 (0.14)	0.47 (0.13)	0.19 (0.15)	0.26 (0.14)
Single-parent	0.48 (0.15)	0.48 (0.14)	0.31 (0.15)	0.18 (0.13)	0.15 (0.13)
Income			-0.06 (0.04)	-0.06 (0.04)	-0.05 (0.04)
Maternal education			-0.11 (0.09)	-0.10 (0.08)	-0.10 (0.08)
Maternal depression			1.96 (0.33)	1.17 (0.30)	1.18 (0.29)
<i>Child-level variables</i>					
Gender				-0.17 (0.06)	-0.18 (0.06)
Age				-0.02 (0.01)	-0.03 (0.01)
Non-biological relatedness: father				0.26 (0.12)	0.31 (0.12)
Non-biological relatedness: mother				0.34 (0.17)	0.26 (0.18)
Maternal negativity				0.43 (0.04)	0.24 (0.11)
Maternal positivity				-0.22 (0.05)	-0.21 (0.5)
<i>Interactions</i>					
<i>Between-level interactions</i>					
Negativity × simple stepfather					0.06 (0.12)
Negativity × stepmother/complex					-0.24 (0.11)
Negativity × single-parent					0.10 (0.12)
<i>Within-level interactions</i>					
Negativity × child age					0.03 (0.01)
<b>Random effects</b>					
<i>Family level</i>					
Control	0.31	0.17 (0.07)	0.15 (0.07)	0.13 (0.06)	0.13 (0.05)
Simple stepfather	-	0.07 (0.14)	-0.03 (0.11)	-0.07 (0.08)	-0.07 (0.08)
Stepmother/complex	-	0.21 (0.15)	0.09 (0.12)	0.25 (0.13)	0.19 (0.11)
Single-parent	-	0.28 (0.15)	0.15 (0.13)	0.10 (0.09)	0.10 (0.09)
<i>Individual child level</i>					
Control	0.51	0.35 (0.06)	0.35 (0.06)	0.27 (0.05)	0.27 (0.05)
Simple stepfather	-	0.34 (0.13)	0.34 (0.13)	0.19 (0.09)	0.18 (0.09)
Stepmother/complex	-	0.19 (0.11)	0.19 (0.11)	0.09 (0.08)	0.08 (0.08)
Single-parent	-	0.05 (0.10)	0.06 (0.10)	-0.01 (0.07)	-0.02 (0.07)
-2(log likelihood)	1143	1129	1088	942	924

For the fixed and random effects parameters, family type was dummy coded, with biological families as the control condition. Thus, the regression coefficients (for fixed effects) and variance estimates (for random effects) indicate the effect relative to the biological families. For example, beginning in model 2, family-level and child-level variances are allowed to differ across family type. Model 2 indicates that there is significantly more child-level variance in stepfather families compared with biological families because the variance estimate, 0.34, is greater than twice its standard error, 0.13. Thus, the total child-level variance in stepfather families is equal to 0.35 (for biological families)+0.34 (the additional variance in stepfather families)=0.69.  $n=453$ .

control condition. Thus, in Table 1, the regression coefficients (for fixed effects) and variance estimates (for random effects) indicate the relative difference compared with biological families. For example, in model 2, the family-level variance for stepmother/complex stepfamilies is the sum of the variances for biological (control) families plus the estimate for stepmother/complex stepfamilies, 0.17+0.21 (Table 1).

Finally, in order to include children in single-parent families and to avoid data loss from non-response by fathers, we used only mothers' parenting in the analyses below. For each measure listed above the percentage of missing data was slight, but this rate was exaggerated when accumulated across many variables. In order to avoid problems with missing data in the final multivariate analysis, we used a mean

substitution method for dealing with missing data.

## RESULTS

### Within-family and between-family variance

In the first, baseline analysis, variation in child psychopathology was decomposed

into family-level and individual child-level sources. Of the total variance in child psychopathology, 41% (0.354/0.858) was attributable to family-level factors and the remainder, 59% (0.504/0.858), to individual child level factors ( $\chi^2$  for base model=1159). This finding is important in providing one of the first direct empirical demonstrations that the differences between children within the same family are as great as, and even slightly greater than, differences between children in different families. We then added the fixed effects of family type, entered as a dummy-coded variable (Table 1, model 1). It will be evident from the coefficients for the family type variables that children in stepmother/complex stepfamilies scored 0.52 of a scale point higher than children in intact families. Children in single-parent families scored 0.48 of a scale point higher than children in intact families (for both,  $P < 0.05$ ). Living with a stepfather was not associated with a significantly increased risk of emotional and behavioural problems (the estimate of 0.22 is less than twice its standard error). The change in fit function when the family type variables were entered into the model was a significant improvement over the baseline model (change  $\chi^2(3)=16$ ,  $P < 0.01$ ). The significant reduction in family-level variance from the baseline model to model 1 signifies that some of the differences that exist between families in children's emotional and behavioural symptomatology can be accounted for by the type of family in which the child lives.

Allowing the family type coefficients to vary at the family and individual level (model 2 in Table 1) was associated with an improved fit of the model (change,  $\chi^2(6)=14$ ,  $P < 0.05$ ). An examination of the random effects parameters indicated that children in stepfather families showed significantly more individual child-level (or within-family) variability than children in biological families. The within-family variance for children in stepmother/complex or single families was not significantly different from intact families. Stepfather, stepmother/complex and single families did not show significantly greater family-level variability compared with biological families. This means that the mean level of emotional and behavioural problems shown by children in these different family types is not more varied than the mean level of problems shown by children in biological families.

### Mediators of behavioural and emotional problems

Models 2 to 5 display the estimates of fixed and random effects as more explanatory variables are added to the equation. In model 3, key family-level risks are added: two indices of socio-economic adversity, and maternal depression. In model 4, child-level variables of age, gender and biological relatedness to mother and father are included. Model 4 also includes the central child-level variable: quality of parent-child relationship. Model 5 adds interactions between and within levels. At each stage, the increase in model fit is significant, based on the change in value with the decrease in degrees of freedom (distributed as a  $\chi^2$ ).

Three features stand out from the models in Table 1. First, the impact of the explanatory variables diminished as variables were added to the equation, demonstrating that these risks do not act in isolation. In particular, family type *per se* did not explain variability in child psychopathology once hypothesised risks were statistically controlled. This can be seen by examining the way in which the coefficients for stepmother/complex and single-parent families became non-significant between model 3 and model 4 when parenting variables were entered into the equation. The interaction suggested that family type moderates proximal risks: parent-child negativity had a greater effect in biological families than in stepmother/complex stepfamilies. The age  $\times$  parent-child negativity interaction indicates that the strength of the connection between negativity and behavioural/emotional problems increases with age.

Second, it is possible to calculate intraclass correlations according to family type. Intraclass correlation, defined as the percentage of family-level variance to total variance, indexes the relative similarity of the two or more siblings within a family, compared with two children chosen randomly. From the results in model 5, the intraclass correlation for biological (control) families is  $[0.13/(0.13+0.27)]=0.33$ ; for stepfather families the intraclass correlation is  $(0.13-0.07)/[(0.13-0.07)+(0.27+0.18)]=0.12$ ; for stepmother/complex stepfamilies it is  $(0.13+0.19)/[(0.13+0.19)+(0.27+0.08)]=0.48$ ; for single-parent families it is  $(0.13+0.10)/[(0.13+0.10)+(0.27-0.02)]=0.48$ . The implication is that family membership may have more of an impact on psychopathology in stepmother/complex and single-parent families than in stepfather and biological families. However, because there were no significant family type differences in family-level variance and only one significant difference in child-level variance across family types, differences in intraclass correlations should be considered as trends rather than as reliable differences.

Third, a novel feature of this approach to studying families is that it allows us to evaluate changes in family-level and child-level effects as variables measured at these levels are entered into the model. The pattern of between-family and within-family variance continued to differ according to family type once explanatory variables were included. As explanatory variables enter the model, the intraclass correlation changes, and it may change differentially for different family types. These effects can be seen by comparing Figs 1 and 2.

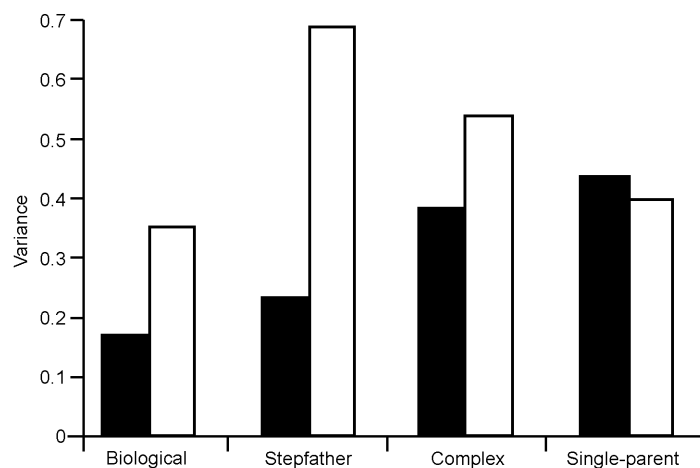
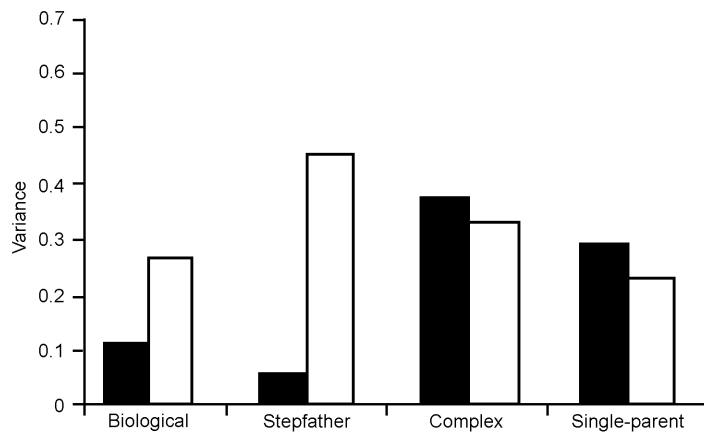


Fig. 1 Family-level (■) and individual child-level (□) variance by family type.





**Fig. 2** Family-level (■) and individual child-level (□) variance by family type: net of predictor variables.

## DISCUSSION

Theories of family influence on children's psychological development have been criticised for failing to account for differences in sibling adjustment and the apparent absence of 'shared' or family-wide effects. The current study, which distinguished between individual child-level and family-level risks, provides clarification of this debate and offers new directions for family research on risk and resilience. The central findings were:

- stepmother/complex stepfamily and single-parent family settings were associated with increased levels of behavioural problems in children, although proximal risks mediated the effect of family type;
- there was substantially more within-family variation in children's outcomes in stepfather families than in biological families; stepmother/complex and single-parent families did not show increased within-family variance compared with biological families;
- siblings in stepmother/complex stepfamilies and single-parent families may be more alike than children in stepfather and biological families.

Documenting variability in children's behavioural/emotional problems following exposure to stress – and understanding who is vulnerable – is a basic requirement for research on risk and resilience (Cicchetti & Cohen, 1995). The current findings extend previous observations about the tremendous variability in children's adjustment to marital transitions in two ways. First,

increased variation in child psychopathology was observed at both the individual child level (increased within-family variation) and the family level (increased between-family variation). We draw attention to this overall pattern, although the specific comparisons were significant in only a minority of cases. Second, although both individual child-level and family-level variance were reduced substantially once key mediating risks were included in the model, the differences between family types were retained (but much reduced). That is, although we were able to account for mean differences in behavioural and emotional problems according to family type, we were unable to account entirely for variance differences. The implication is that there may be risk factors that accentuate variability in psychopathology in non-traditional families without necessarily increasing mean level of disturbance.

### Do risk factors operate on an individual or family-wide basis?

A challenge for family researchers has been to document not only the risks for children's behavioural and emotional problems, but also the mechanisms by which they operate. Recent debates have focused particularly on whether identified risk factors operate on an individual or on a family-wide basis (Deal *et al*, 1994; Plomin, 1994; Rutter *et al*, 1999). By separating family-level from individual child-level risks, the multi-level model approach provides clarity on the nature of risk processes for children's

psychopathology. Results in Table 1 reveal that child-specific risks explained child-level or within-family variation, whereas family-wide or between-family risks explained between-family variation.

What is a particularly novel finding is that a risk measured at the family level – family type – could affect children in the same family differently. This was illustrated by the increased child-level variance across family types, particularly in the stepfather families. Similarly, the interaction between a family-level variable – family type – and a child-level variable – parent-child negativity – highlights the need to consider specific factors that shape siblings' different responses to a 'shared' risk. Further research using this conceptual and methodological approach may explain why children in the same family are not similarly vulnerable to stresses presumed to affect the whole family. An important lesson from these analyses is that identifying risks provides no necessary clues as to their mode of operation. Risks measured at the family level may not operate on a family-wide basis, and risks measured at the individual child level may have family-wide effects.

In addition to highlighting the importance of within-family variation in children's behavioural and emotional problems, these findings also demonstrate the equally important – and more novel – point that some risks do appear to operate in a family-wide manner. The finding that there is a 'shared' or family-wide effect is most clearly demonstrated by the patterns of intraclass correlations. Sibling resemblance (i.e. the magnitude of intraclass correlation) was greatest in family types that appear to be experiencing the most adverse circumstances: single-parent families and stepmother/complex stepfamilies. The implication is that a stressful environment may have family-wide effects, increasing the level of psychopathology for all children rather than (or in addition to) specific children within the family. It seems unlikely that genetic similarity among siblings could entirely account for the family-wide effect. If genetic relatedness were a major factor, then we would expect proportionately greater family-level variance in families with the highest degree of biological relatedness. In fact, stepmother/complex families, which had the greatest intraclass correlation, included the highest percentage of half-siblings and unrelated siblings.

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## CLINICAL IMPLICATIONS

- Researchers and clinicians need to consider the wide variation in children's behavioural and emotional problems following family transitions, and the likelihood that even children in the same family may adapt differently to a parental separation or re-partnering.
- Stresses within the family are more important than family type *per se* in accounting for children's psychopathology. Parental psychopathology and impaired parent–child relationship quality are particularly associated with poor adjustment.
- Individual child-level risks are important in explaining variation in psychopathology; family-level risks may be especially important at high levels of family stress.

## LIMITATIONS

- Reports of children's psychopathology were based only on parent report.
- Analyses are based on a cross-sectional design; data from the prospective longitudinal aspect of the study are forthcoming.
- Only family risks were assessed; additional research is needed on extrafamilial risks that explain different sibling outcomes.

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