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**Child hyperactivity, mother-child negativity, and sibling dyad negativity:  
A transactional family systems approach**

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### Abstract

ADHD is among the most common mental disorders in children and adolescents. While most studies in this field have focused on the genetic and neurobiological underpinnings of the disorder, research focusing on the family environment as a critical context contributing towards the manifestation and maintenance of child ADHD symptoms is still less extensive. Therefore, the aim of this study was to examine longitudinal and bidirectional associations between child hyperactivity, mother-child negativity, and sibling dyad negativity. Data were analyzed of up to 4,429 children from the Avon Longitudinal Study of Parents and Children (ALSPAC), a nationally representative prospective birth cohort study in the United Kingdom, at three time points (T1-T3, children aged 4, 7, and 8 years). At baseline (T1,  $n = 4,063$ ) the child sample (98.8% White ethnic background) comprised of 51.6% males. Child hyperactivity symptoms, mother-child negativity, and sibling dyad negativity were examined based on maternal report. The random intercepts cross-lagged panel model was adopted to parcel out between-family differences from within-family fluctuations and test bidirectional associations. On the between-family level, families with higher child hyperactivity reported higher mother-child and sibling dyad negativity. On the within-family level, unidirectional spillover processes between sibling dyad negativity and mother-child negativity as well as between mother-child negativity and child hyperactivity emerged. Future work in the area of child hyperactivity should implement a transactional family systems approach incorporating both parent-child and sibling subsystems. Interventions to reduce negative interactions between parents and hyperactive children may offer promising improvements in child symptoms and thus alleviate family burden.

Keywords: mother-child; siblings; child hyperactivity; family systems; Avon Longitudinal Study of Parents and Children

**Child hyperactivity, mother-child negativity, and sibling dyad negativity:****A transactional family systems approach**

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental childhood-onset disorder characterized by a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with child development in several domains. With prevalence estimates ranging from 2.2–7.2%, ADHD is among the most common mental disorders in children and adolescents (Sayal et al., 2018). ADHD is associated with a significant economic burden, as illustrated by a recent longitudinal study demonstrating that hyperactivity in childhood was related to a 17-fold increase in overall costs associated with mental health, social, educational, and criminal justice service use by early adulthood compared to controls (Chorozoglou et al., 2015). Considering the high heritability of ADHD, it is unsurprising that a large majority of studies in this field have focused on exploring possible genetic and neurobiological underpinnings of the disorder (Faraone & Larsson, 2019). In contrast, research on the family relationships in families of children with ADHD appears less extensive (Claussen et al., 2022; Johnston & Mash, 2001; Weyers et al., 2019). To understand the developmental course of the disorder and intervene appropriately, an emphasis on family dynamics is equally important (Deault, 2010). Therefore, this study aims to examine bidirectional between- and within-family associations between mother-reported child hyperactivity symptoms and mother-child as well as sibling relationships across early childhood.

**A Transactional Family Systems Approach to Developmental Psychopathology**

Family systems theory (FST; Cox & Paley, 1997; Minuchin, 1974) postulates that families function as an interdependent system, in which different family subsystems and relationships (e.g., interparental, parent-child, and sibling) mutually influence one another. Accordingly, child development should be considered within the context of the whole family system, incorporating multiple levels of influence within families and across subsystems.

Transactional models support this notion, further arguing that child development is a product of the bidirectional interaction between the child and their developmental context (e.g., family and social context; Sameroff & Mackenzie, 2003). To gain a holistic understanding of developmental psychopathology, a family systems-informed transactional model has been highlighted as crucial (Deater-Deckard, 2017). As such, the quality of family relationships may influence the well-being and functioning of children, and vice versa. Numerous studies have reported on the spillover between parent-child and sibling relationship negativity (e.g., Yu & Gamble, 2008). Moreover, there is robust meta-analytic evidence suggesting a link between child externalizing problems and parent-child (Pinquart, 2017) or sibling negativity (Buist et al., 2013). Previous work further suggests a reciprocal relationship between child externalizing behavior and maternal negativity (Zadeh et al., 2010) as well as sibling dyad negativity (Pike & Oliver, 2017). Researchers argue that this bidirectionality may extend to symptoms of mental disorders that are considered as primarily neurodevelopmental in origin, such as ADHD (Lifford et al., 2009). Indeed, emerging evidence has illustrated such bidirectional processes between mothers' parenting behavior and child ADHD symptoms in a genetically sensitive longitudinal adoption-at-birth sample (Harold et al., 2013).

### **Family Relationships of Children with ADHD**

The family environment has long been proposed as a critical context contributing to the manifestation, maintenance, and exacerbation of the symptoms and developmental outcomes of child ADHD (Claussen et al., 2022; Johnston & Mash, 2001; Weyers et al., 2019). The strain caused by this disorder likely disrupts family relationships, thereby impairing family functioning (Deault, 2010). For instance, there is robust evidence that parents of children with ADHD experience higher parenting stress compared to parents of healthy controls, with symptom severity being directly related to increased levels of parenting stress (Theule et al., 2013). It has further been found that families of children diagnosed with ADHD display increased interparental conflict (Weyers et al., 2019) and parent-child conflict

(Markel & Wiener, 2014). Families of children with subclinical ADHD symptoms have similarly shown impaired family functioning, including increased mother-child hostility and conflict (Heckel et al., 2013; Lifford et al., 2009). Symptoms of hyperactivity/impulsivity, but not inattention, were found to be particularly associated with increased parenting stress and family conflicts (Graziano et al., 2011). Therefore, the current study focuses on child hyperactivity as a cardinal symptom of ADHD that is strongly associated with family relationships.

### **Sibling Relationships of Hyperactive Children**

While the majority of studies addressing family relationships of hyperactive children have focused primarily on parent-child and interparental relationships, few studies have also considered sibling relationships (Anastopoulos et al., 2009; Paidipati & Deatrick, 2015). These studies found that children diagnosed with ADHD experienced their sibling relationships as more negative and conflict-laden compared to healthy control groups (Kouvava & Antonopoulou, 2020; Mikami & Pfiffner, 2008). A recent study exploring the impact of child ADHD on family stress and well-being in everyday life further identified the sibling domain as a key source of daily family strain, with higher levels of sibling negativity being related to increased hyperactive-impulsive symptoms (Bauer et al., 2019). Analogous to the parent-child relationship, studies examining sibling dyads in which one child suffers from ADHD indicate that the association with sibling conflict is particularly strong in the context of elevated hyperactivity-impulsivity symptoms, as opposed to inattention (Mash & Johnston, 1983). Accordingly, children involved in sibling bullying perpetration were at increased risk of presenting clinically elevated hyperactivity problems (Wolke & Samara, 2004).

To our knowledge, only one previous study has examined the link between ADHD and mother-child and sibling relationship quality simultaneously (Heckel et al., 2013). The study focused on a sample of 479 children and adolescents aged 6-18 years who had been referred to a pediatric practice for an ADHD assessment. The findings suggested that children

diagnosed with ADHD had poorer relationships with their mothers, with stronger effects in those diagnosed with the combined (hyperactive-impulsive and inattentive) subtype than those with the pure inattentive subtype. Moreover, the authors reported a link between ADHD diagnosis and poor sibling relationships, although this was specific to the relationship between boys diagnosed with the combined subtype and their sisters. Once the quality of family relationships (mother, father, and sibling) was controlled for in the same model, the severity of ADHD symptoms was associated with a more disrupted relationship with the child's sisters only. However, the generalizability of the findings is limited due to the clinical sample and the cross-sectional study design.

### **Gaps in the Current Literature**

In sum, there is strong evidence that families of children with high levels of hyperactivity are characterized by increased negativity in parent-child and sibling dyad relationships. Nevertheless, four major gaps in the current literature can be identified: First, studies have primarily considered either the parent-child or (more rarely) the sibling relationship (Anastopoulos et al., 2009). It is important to study multiple relationships in the family simultaneously, as they are inextricably and reciprocally interconnected (e.g., Zemp et al., 2021).

Second, as most previous studies used cross-sectional designs or were descriptive in nature, the direction of effects between family negativity and child hyperactivity symptoms remains unclear (Johnston & Mash, 2001). There is thus a pressing need for longitudinal data allowing us to explore the temporal order of these constructs across child development.

Third, the vast majority of studies did not systematically distinguish between-family differences from within-family fluctuations. Hence, they provide no insight into the associations between child hyperactivity and family negativity as they unfold over time *within a given family*. To advance our understanding of how changes within families are interrelated, statistical models that partition the two different sources of variance (within- and between-

level) are essential (Curran & Bauer, 2011). Recent family research disentangling between- and within-family variances reported that these processes do not necessarily operate in the same way (Zemp et al., 2018). We deem the untangling of within-family changes from between-family differences as a particularly powerful means to test mutual influences between child hyperactivity and negativity in the mother-child and the sibling relationship, and how they fluctuate over time within families.

Fourth, many studies have focused on clinical samples of children and adolescents with a previous diagnosis of ADHD. Given important drawbacks associated with a categorical approach (e.g., risk of an artificial demarcation between disturbed and ‘normal’ phenomena that insufficiently reflects individual cases), researchers have argued that ADHD is best viewed as the extreme end of a continuous trait rather than as a binary manifested disorder, particularly when exploring the development and course of symptoms in the normal population (Larsson et al., 2012). Moreover, studies in young children indicate that hyperactive-impulsive symptoms are temporally unstable and should thus be treated as a continuum rather than a nominal diagnostic label (Lahey et al., 2005).

### **The Current Study**

The aim of this study was to examine the longitudinal and bidirectional associations between mother-reported child hyperactivity and mother-child as well as sibling dyad negativity, while statistically differentiating between-family differences from within-family fluctuations. For this purpose, we analyzed data of over 4,000 children from a nationally representative prospective birth cohort study from the UK, the Avon Longitudinal Study of Parents and Children using three time points (T1, T2, and T3, when the children were 4, 7, and 8 years old, respectively). The data were based on maternal reports focusing on the cohort child and their older sibling. Thus, the quality of the mother-child (cohort child and mother) and sibling dyad (cohort child and older sibling) relationships was assessed from the mother’s perspective. A novel statistical approach was adopted, namely the random intercepts cross-



lagged panel model

(RI-CLPM; Hamaker et al., 2015), in order to parcel out between- and within-persons (or families, respectively) variances. We focused on the dimension of hyperactivity as a continuum of symptom severity within a community sample.

On the between-family level, we anticipated intercorrelations between child hyperactivity symptoms, mother-child negativity, and sibling dyad negativity. On the within-family level, we expected bidirectional longitudinal associations between child hyperactivity symptoms and negativity in family relationships across the three time points of child development. Specifically, we hypothesized that higher child hyperactivity levels would predict greater negativity in the mother-child and sibling dyad relationships at the subsequent time point within families. Conversely, we assumed that higher levels of family negativity in the mother-child and sibling dyad relationships would be prospectively linked to greater child hyperactivity symptoms within families.

## **Method**

### **Study Design**

This study used data from the UK representative prospective birth cohort study ALSPAC. Pregnant women resident in Avon, UK, with expected dates of delivery from 1st April 1991 to 31st December 1992 were invited to participate. The initial number of pregnancies enrolled was 14,541. Of these, 14,062 resulted in live births and 13,988 children who were alive at 1 year of age. From 1991 onwards, ALSPAC data have been collected annually and include a broad range of sociodemographic, psychological, and physiological outcomes. Detailed reports on the recruitment and enrolment processes of the mother and the cohort child are available in the cohort profiles (Boyd et al., 2013; Fraser et al., 2013). Ethical approval for the study was obtained from the fromtheALSPAC Ethics and LawCommittee (Institutional Review Board No. 00003312) and the local research ethics committees (Bristol and Weston Health Authority, Southmead Health Authority and Frenchay Health Authority).

### **Transparency and Openness**

We report how we determined our sample size, all data exclusions, and all measures in the study. Please note that the study website contains details of all available data through a fully searchable data dictionary and variable search tool (<http://www.bristol.ac.uk/alspac/researchers/our-data/>). The analysis code for this study is openly available at <https://osf.io/e3vxq/>. This report's design and its analysis were not pre-registered.

### **Sample**

The current sample includes all cohort children whose mothers reported on child hyperactivity symptoms as well as mother-child and sibling dyad negativity when cohort children were aged four (T1), seven (T2), and eight (T3) years. Due to our specific interest in the sibling relationships, we selected an initial starting sample comprising all cohort children who grew up in a household with at least one older sibling ( $n = 4,429$ ). Information about the sibling relationships was limited to the sibling dyad consisting of the cohort child and their older sibling closest in age. Our final sample included 4,063 mother-child dyads at T1, 4,415 mother-child dyads at T2, and 3,582 mother-child dyads at T3. Data points across all time points were available for 3,406 mother-child dyads. Data from simulation studies have led to the recommendation that longitudinal studies with three time points should employ a sample size of  $N > 2,500$  (Hecht & Zitzmann, 2021) to ensure very good model performance; thus, our sample size was deemed sufficiently large.

Demographic characteristics of mothers and cohort children at baseline (T1) were as follows: The child sample comprised 2,098 (51.6%) males. The mothers' age ranged from 16 to 44 years ( $M = 29.99$ ,  $SD = 4.27$ ). The majority of families were from a White ethnic background ( $n = 3,923$ ; 98.8%). A total of 2,957 (74.3%) of mothers had at least ordinary-level qualifications (35% ordinary-level qualifications; 24.2% advanced-level qualifications; 14.2% university degree), and 3,155 (78.6%) of mothers reported being married.

## Measures

**Child hyperactivity.** Child hyperactivity symptoms were assessed using five items taken from the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) according to maternal reports at T1, T2, and T3. Mothers were asked to think about their child's behavior over the last six months and indicate to what extent each statement applied to their child. Hyperactivity symptoms included: (1) "she/he has been restless, overactive, cannot stay still for long"; (2) "she/he is constantly fidgeting or squirming"; (3) "she/he is easily distracted, her/his concentration wanders"; (4) "she/he thinks things out before acting" (reverse-coded); (5) "she/he sees tasks through to the end, has good attention" (reverse-coded). Responses were given on a 3-point Likert scale, with 0 = *doesn't apply/not true*, 1 = *applies somewhat/somewhat true*, and 2 = *certainly applies/certainly true*. Child hyperactivity was treated as a continuous construct, thus a sum score was computed to reflect child hyperactivity symptoms on a continuum from 0 to 10, with higher scores indicating greater symptom severity. Cronbach's  $\alpha$  ranged from .78 to .82 across the three waves (T1 through T3) in the current study.

**Mother-child negativity.** Items assessing mother-child negativity were adapted from a previously published measure reflecting mother-child relationship quality (comprising two subscales: mother-child positivity and mother-child negativity), which has demonstrated high face and predictive validity (Dunn et al., 1999; Oliver & Pike, 2018). The present study exclusively focused on mother-child negativity. Mothers were asked to report on the negative aspects of their relationship with the cohort child using four items at each of the three time points (T1, T2, T3): (1) "I often get very irritated with this child"; (2) "I dislike the mess and noise that surrounds this child"; (3) "I have frequent battles of will with this child"; (4) "this child gets on my nerves". Items were rated dichotomously with 0 = *no* and 1 = *yes*. A mean score from 0-1 was computed across the time points, with a higher score reflecting greater

mother-child negativity. Cronbach's  $\alpha$  ranged from .64 to .70 across the three waves (T1 through T3) in the current study.

***Sibling dyad negativity.*** Items pertaining to sibling relationship negativity were adapted from a previously validated measure to assess sibling relationship quality (Dunn et al., 1999).

Previous studies have identified two underlying factors within this scale: sibling positivity and sibling negativity (Dunn et al., 1999; Pike & Oliver, 2017). The present study exclusively focused on sibling negativity. Sibling relationship negativity was assessed using eight items according to maternal reports at T1, T2, and T3. Mothers were asked to report on negative aspects of the dyadic relationship between the cohort child and their older sibling (nearest in age to the cohort child). Mothers reported how frequently the cohort child: (1) "quarrels with this older child"; (2) "is unhappy/jealous if [they] do things just with this older child"; (3) "is unhappy/jealous if [their] partner does things just with this child"; (4) "teases/needles this older child". Subsequently, mothers rated the same four items for the cohort child's sibling. Items were rated on a 3-point Likert scale: 0 = *rarely/never*; 1 = *sometimes*; 2 = *frequently*. A collective mean score reflecting maternal reports of the dyadic relationship was computed (encompassing ratings of the cohort child and of the older sibling) ranging from 0-2 across all time points, with higher scores reflecting greater sibling dyad negativity. Cronbach's  $\alpha$  ranged from .83 to .85 across the three waves (T1 through T3) in the current study.

***Control variable:*** As boys are consistently found to present higher levels of hyperactivity than girls (Sayal et al., 2018), child gender is a potential confounder to consider when studying child hyperactivity. Therefore, we included child gender as a control variable.

### **Missing Data**

Longitudinal designs are prone to missing data due to attrition over time. A total sample size of 3,406 (76.9% of the total starting sample) was retained across the four-year span of this study. We performed a dropout analysis to compare non-completers (mother-child dyads who did not participate across all three time points;  $n = 1,023$ ; 23,1%) to those who

completed all three time points (completers;  $n = 3,406$ ) regarding demographic characteristics and the main study variables using independent sample  $t$ -tests for continuous variables and  $\chi^2$ -tests for categorical variables (see supplemental material; Table S1). Compared to the non-completers, the mothers in the completer group were significantly older. Moreover, the completers were more likely to be in their first marriage and to have a white ethnic background as well as a higher educational status. Completers further reported lower levels of child hyperactivity, mother-child negativity, and sibling dyad negativity at T1 compared to non-completers.

To account for the missing data due to attrition across the three time points, we used full information maximum likelihood (FIML) estimation in Mplus (Muthén & Muthén, 1998–2017) and were thus able to use the complete starting sample size throughout our analyses. FIML uses all available information in the variance/covariance matrix to compute model parameters and is therefore able to produce less biased estimates compared to listwise or pairwise deletion or listwise substitution (Johnson & Young, 2011).

### **Statistical Analysis**

Preliminary data analysis was conducted using IBM SPSS Statistics 25.0. All statistical modeling was conducted with Mplus 8.1. (Muthén & Muthén, 1998–2017) using FIML estimation to handle missing data. Global model fit was evaluated using the  $\chi^2$ -test, the root mean square error of approximation (RMSEA), the comparative fit index (CFI), the Tucker–Lewis Index (TLI), and the standardized root mean square residual (SRMR). A non-significant  $\chi^2$ , CFI and TLI values greater than .95, and RMSEA and SRMR values smaller than .06 and .08, respectively, are common criteria indicating good model fit (Little, 2013).

To disentangle between- and within-family fluctuations, we utilized the random intercept cross-lagged panel model (RI-CLPM) to analyze our data (Hamaker et al., 2015). Stable between-person variance is captured by a random intercept that is regressed on the construct at all time points. Residual variance in the construct not captured by the random

intercept reflects within-family fluctuations. Figure 1 provides a conceptual illustration of this model as applied to the current study. On the between-family level, associations between intercept variables reflect between-person differences (e.g., children display greater mother-reported hyperactivity in families with higher mother-child negativity compared to families with lower mother-child negativity). On the within-family level, cross-lagged relationships reflect how within-family deviations from a stable level of one construct (e.g., elevated mother-child negativity at one time point, controlling for the typical level of this particular family) is linked to subsequent within-family deviations in another construct (e.g., more severe child hyperactivity symptoms at the next time point, compared to the given child's usual symptom level). By examining cross-lagged relationships at the within-family level, while controlling for differences at the between-family level and autoregressive paths across time points, the model provides a particularly powerful basis for studying temporal order in longitudinal data (Zemp et al., 2018).

[Figure 1]

## **Results**

Descriptive statistics and the correlation matrix across the main study variables are illustrated in Table 1. Table 2 depicts the results of our RI-CLPM. The RI-CLPM fit the data well, as indicated by the model fit indices presented in the respective table.

[Table 1]

[Table 2]

### **Between-Family Associations**

Associations between the random intercept variables reflect how stable between-family differences in one construct are associated with stable between-family differences in another construct. Mothers who reported higher child hyperactivity also reported higher levels of both mother-child and sibling dyad negativity (see Table 2). Moreover, mothers who reported higher mother-child negativity overall reported higher sibling dyad negativity.

### **Within-Family Associations**

Autoregressive effects provide information about how prior deviations from a family's or child's typical level of one construct are associated with fluctuations in that same construct at the next time point. We found that mothers reporting higher than average mother-child negativity or sibling dyad negativity at T2 reported higher than usual levels of mother-child negativity and sibling dyad negativity at T3, respectively (see Table 2). Similarly, children who showed higher than average levels of child hyperactivity at T2 were also more likely to show higher than average levels of child hyperactivity at T3. There were no significant autoregressive effects from T1 to T2.

Cross-lagged pathways capture how fluctuations in a family's or a child's typical level of one construct are associated with future fluctuations in another construct in this given family or child. The findings revealed that higher than usual sibling dyad negativity at T2 predicted higher than average mother-child negativity at T3. Moreover, the findings showed that elevated mother-child negativity at T2 (controlling for the usual level of this particular family) predicted higher than average child hyperactivity at T3 (but not at T1 and T2). We found no bidirectional within-family associations between child hyperactivity and mother-child as well as sibling dyad negativity at any time point.

### **Control Variable**

We included the cohort child's gender in the analysis by regressing it onto the random intercepts of our three main constructs. Mothers of boys reported higher child hyperactivity and lower sibling dyad negativity as compared to mothers of girls (Table 2).

## **Discussion**

There is consistent evidence suggesting that family relationships represent an important developmental context for the manifestation and maintenance of child ADHD symptoms (Johnston & Mash, 2001). However, existing studies have primarily focused exclusively on aspects of the parent-child (Lifford et al., 2009) or the interparental

relationship (Weyers et al., 2019), thus neglecting the potential additional influence of the sibling relationship. Moreover, a large majority of previous studies were based on cross-sectional data, rendering it difficult to draw temporal conclusions. The current study aimed to address these shortcomings by examining the longitudinal and bidirectional associations between child hyperactivity symptoms, mother-child negativity, and sibling dyad negativity across early childhood. Additionally, our study is the first to parcel out between-family differences from within-family fluctuations. In line with previous studies (Zemp et al., 2018), we found that separating between- and within-family variances resulted in contrasting and in part unexpected results pertaining to the intricate dynamics between child hyperactivity and mother-child and sibling dyad interactions.

On the between-family level, we found that all of our constructs were intercorrelated with one another. Specifically, mothers reporting higher levels of child hyperactivity also reported higher levels of mother-child and sibling dyad negativity compared to mothers reporting lower levels of child hyperactivity. Additionally, mothers reporting higher mother-child negativity also reported more sibling dyad negativity compared to those with lower mother-child negativity. Our findings correspond to previous studies reporting positive links between parent-child conflict or negativity and child hyperactivity symptoms in families of children with subclinical manifestations of ADHD (Lifford et al., 2009), as well as in families of children diagnosed with ADHD (Harold et al., 2013). They further resonate with sibling research demonstrating that children with ADHD grow up with more negative and conflict-laden sibling relationships (Kouvava & Antonopoulou, 2020; Mikami & Pfiffner, 2008).

Turning to within-family fluctuations, unlike past research (Heckel et al., 2013), we did not find any bidirectional links between child hyperactivity and mother-child negativity. Thus, higher than usual levels of child hyperactivity did not predict increases in mother-child negativity within families. We did however find unidirectional spillover processes between mother-child negativity and child hyperactivity. Specifically, families with higher than usual



mother-child negativity within a given family at the child's age of seven predicted increases in child hyperactivity at age eight. These findings are partially in line with a transactional model of developmental psychopathology (Sameroff & Mackenzie, 2003), which postulates that child behavior is the product of the dynamic and reciprocal interaction between the child and their developmental (e.g., family and social) context. In the quest to unravel how the gene-environment interaction may fuel the development of child ADHD, researchers have called for the integration of a transactional family systems approach (Deater-Deckard, 2017). According to a diathesis-stress model of ADHD, the family environment is often not considered a primary etiological factor but is rather seen as a secondary driver involved in the manifestation, maintenance, and exacerbation of ADHD symptoms in genetically vulnerable children (Zemp, 2018). Our results resonate with this theoretical perspective, as we assume that negative family interactions play a crucial role in this vicious cycle of symptom aggravation.

We may speculate why we did not find any spillover from child hyperactivity onto mother-child negativity. Our study employed a continuous assessment of ADHD symptoms, thereby neglecting the categorical approach inherent to the clinical diagnosis of ADHD. It may be that bidirectional linkages are especially pronounced in a subgroup of families most burdened with children on the severe end of the symptom spectrum. The presentation of clinically severe child ADHD symptoms has, for instance, been shown to evoke higher levels of parenting stress and to especially distress family relationships (e.g., Theule et al., 2013), thus maintaining or enhancing children's symptoms and problem behavior (Johnston & Mash, 2001). Future studies including a clinical diagnosis of ADHD, in addition to a continuous assessment of ADHD symptoms, may allow conclusions to be drawn about possible bidirectional relationships between child ADHD symptoms and family relationships in the presence of a confirmed diagnosis.

Furthermore, the current study revealed a within-family association between sibling dyad negativity at age seven and mother-child negativity at age eight, suggesting spillover effects from sibling dyad negativity onto mother-child negativity. Importantly, however, the significant cross-lagged effects consistently emerged only between the time points T2 and T3 (when children were seven and eight years of age) and not between the first two time points (when children were four and seven). These differences suggest that in order to truly capture developmental processes as they unfold over time within a given family, it may be necessary to incorporate shorter time lags. Indeed, simulation studies exploring the selection of optimal time lags in panel studies argue that time lags of several months or years may be too long, and instead recommend supplementing research with “shortitudinal” and intensive longitudinal data (e.g., diary studies; Dormann & Griffin, 2015).

Surprisingly, there were no associations between sibling dyad negativity and child hyperactivity at any time point, in either direction of effect. Taken together, our pattern of results suggest that mother-child interactions may be more central to the manifestation or maintenance of child hyperactivity symptoms than sibling dyad interactions on the within-family level. The question thus arises of why, within a given family, the role of sibling dyad negativity might be less important for future child hyperactivity, or vice versa, compared to mother-child negativity. Several potential explanations may be put forward. First, it is important to consider the age of the cohort children in the current sample. According to attachment theory, the parent-child relationship is seen as central for child development across early childhood (Bowlby, 1969). Parents, and in particular mothers, are found to spend proportionately higher amounts of time with their children in the early years, with general declines observed across the course of development from middle to late childhood (Lam et al., 2012). While siblings too play an important role in early childhood, they are found to take on an especially salient role in children’s lives around middle childhood; indeed, time spent with siblings has been found to surpass time spent with parents during this period (McHale &

Crouter, 1996). Thus, the stronger influence of mother-child negativity on child hyperactivity in this sample of young children is plausible. Second, sibling dyad negativity was assessed by mother-reports only. Mothers may be more reliable raters of and more perceptive towards their own interaction with their child, as opposed to the child's interaction with the siblings. This methodological circumstance could have amplified the effects regarding mother-child negativity at the expense of sibling dyad effects. Third, although aspects of the mother-child and sibling dyad subsystem were considered simultaneously, it was not possible to test for possible mediation effects across the key variables. As suggested by FST (Cox & Paley, 1997; Minuchin, 1974), it is crucial to understand family relationships as interdependent subsystems that are reciprocally interlinked. Considerable evidence points at a spillover mechanism across sibling dyad and mother-child negativity (Yu & Gamble, 2008), a finding which was also mirrored in our results. Thus, it is possible that mothers exhibit more negativity in response to increased sibling dyad negativity, in turn enhancing child hyperactivity (Buist et al., 2013), or vice versa. Lastly, it should be noted that sibling dyad negativity was correlated with both mother-child negativity and child hyperactivity. It is therefore possible that there was not enough variance for effects to emerge (i.e., due to collinearity) or that a suppression effect may have occurred (i.e., sibling dyad negativity acting as a suppressor variable, increasing the predictive power of mother-child negativity). Future studies should therefore incorporate mother-child negativity as a possible mediator between child hyperactivity and sibling dyad negativity, as this may help elucidate the complexity of their interplay.

### **Practical Implications**

Our findings have several clinical and practical implications. The central role of mother-child negativity as a temporal precursor of child hyperactivity underlines the potential for interventions tailored at improving parenting practices with the goal of alleviating child ADHD symptoms. As such, parents might be trained to better identify early child behavior that evokes child-directed negativity, thus preventing an increase in child hyperactivity and

consequently alleviating the family burden associated with the child's symptoms (Harold et al., 2013). There is considerable evidence to support the effectiveness of parenting training in the treatment of ADHD, especially in young children (e.g., Rimestad et al., 2019). Studies have further found that beginning the treatment with behavioral parenting training, as opposed to medication, may yield better treatment outcomes overall (Pelham et al., 2016). Hence, an emphasis on enhancing family relationships by reducing negative interactions between parents and hyperactive children, as the current study revealed, can be seen as important and highly promising.

Moreover, although this study did not reveal any within-family effects of sibling dyad negativity on child hyperactivity, we did find associations on the between-family level. Our findings thus emphasize the importance of considering sibling relationships in the treatment of children's ADHD symptoms. Along these lines, a recent pilot study found that sibling dyad behavioral interventions for children with ADHD may be effective in reducing sibling dyad negativity (Rosenthal et al., 2022). Thus, future work should incorporate the important developmental context of the sibling relationship in order to enhance our understanding of the reciprocal associations between child hyperactivity and multiple family relationships (Heckel et al., 2013).

### **Limitations**

The present findings should be interpreted with some limitations in mind. First, as all measures were based on maternal reports only, a possible bias due to common-method variance cannot be excluded. Future work should thus employ a multi-rater approach. Incorporating child self-reports of sibling dyad negativity may be particularly important given that sibling bullying usually occurs without the parents' knowledge (Dantchev & Zemp, 2022). Moreover, the inclusion of paternal or other parental figures' reports could complement a transactional family systems approach. The addition of observational data, which are among the most valid methods for studying aspects of family negativity, would also

strengthen respective study designs. Furthermore, the current sample lacked information regarding the sibling dyad composition (in terms of age and gender). Other studies have reported differential effects between sibling negativity and ADHD once age and gender were accounted for (Heckel et al., 2013).

Second, the time points included in our analysis were unevenly spaced and our time lags were long (i.e., three years and one year, respectively). Unfortunately, no data were available at other time points, and we were therefore unable to capture family processes as they unfold on a shorter (e.g., daily) basis. Due to this issue, it is also difficult to draw causal conclusions from our data, because numerous factors occurring between the time points may have influenced the examined associations. Future work should incorporate daily diary studies using ambulatory assessment. This approach minimizes retrospective biases and allows for the collection of ecologically valid data on daily experiences as they occur in real time of everyday family life (Trull & Ebner-Priemer, 2013).

Third, we were unable to control for parental ADHD. Meta-analytic evidence has revealed that genetic factors of ADHD account for around 70% of the variance in symptoms (Faraone & Larsson, 2019), reflecting the high heritability of the disorder. Given that parental negativity is particularly pronounced in families with parental ADHD (Park et al., 2017), and as parental ADHD may further lead to reporting bias of child ADHD symptoms (Faraone et al., 2003), the lack of assessment of and control for parental ADHD is an important weakness of this study.

Fourth, we assessed ADHD symptoms using a dimensional approach and we did not test group differences using a confirmed ADHD diagnosis due to the low prevalence in our sample. Thus, we cannot make any specific claims about family interactions of children diagnosed with ADHD. Although it has been argued that dimensional approaches are especially useful in normal populations (Larsson et al., 2012) and young children (Lahey et al., 2005), it might be important for future studies to additionally employ a categorical

approach, as this may shed light on specific processes observed in families most burdened by child hyperactivity symptoms. Moreover, the exclusive reliance on mother-reported hyperactivity based on the SDQ does not adequately reflect clinical practice. It would be useful for future work to include independent raters outside the family (e.g., clinicians or teachers), considering the importance of integrating ratings from multiple informants in the clinical presentation of ADHD.

Finally, it is important to note that we examined an ethnically homogeneous sample with low diversity, thus limiting the generalizability of the current findings. Future work should seek to replicate our findings in more diverse samples representing ethnic minorities, sexual or gender minorities, families with low socioeconomic status, or otherwise at-risk populations.

### **Conclusion**

ADHD is a family affair. Our study contributes to this notion by adopting a novel statistical approach to parcel out between-family differences from within-family fluctuations, allowing for a particularly powerful methodology to test mutual influences of family interactions. In summary, the current findings suggest that supporting families to reduce negative interactions between mothers and hyperactive children holds promise for reducing the expression of hyperactivity in children. This, in turn, would likely alleviate the burden associated with the child's symptoms for all family members. It is of central importance to incorporate the developmental context of the whole family system – including the parental, parent-child, and sibling relationships – both in future research and in clinical practice.

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**Table 1***Correlations, means, and standard deviations of focal study variables*

Variable (time point)	<i>M</i>	<i>SD</i>	Bivariate correlations									
			1	2	3	4	5	6	7	8	9	
1. Child hyperactivity (T1)	3.99	2.39	-									
2. Child hyperactivity (T2)	3.29	2.36	.59*	-								
3. Child hyperactivity (T3)	3.22	2.49	.57*	.73*	-							
4. Mother-child negativity (T1)	0.30	0.30	.39*	.29*	.30*	-						
5. Mother-child negativity (T2)	0.23	0.30	.32*	.36*	.36*	.52*	-					
6. Mother-child negativity (T3)	0.23	0.30	.31*	.33*	.41*	.53*	.66*	-				
7. Sibling dyad negativity (T1)	2.01	0.42	.26*	.21*	.22*	.35*	.30*	.32*	-			
8. Sibling dyad negativity (T2)	2.05	0.42	.23*	.25*	.25*	.31*	.40*	.37*	.58*	-		
9. Sibling dyad negativity (T3)	1.96	0.42	.23*	.22*	.25*	.30*	.34*	.39*	.57*	.68*	-	

*Note:* Child hyperactivity scores ranged from 0-10. Mother-child negativity scores ranged from 0-1. Sibling dyad negativity scores ranged from 0-2. \* $p < .01$  (two-tailed).

**Table 2***Saturated RI-CLPM for child hyperactivity, mother-child negativity, and sibling dyad negativity (n = 4,423)*

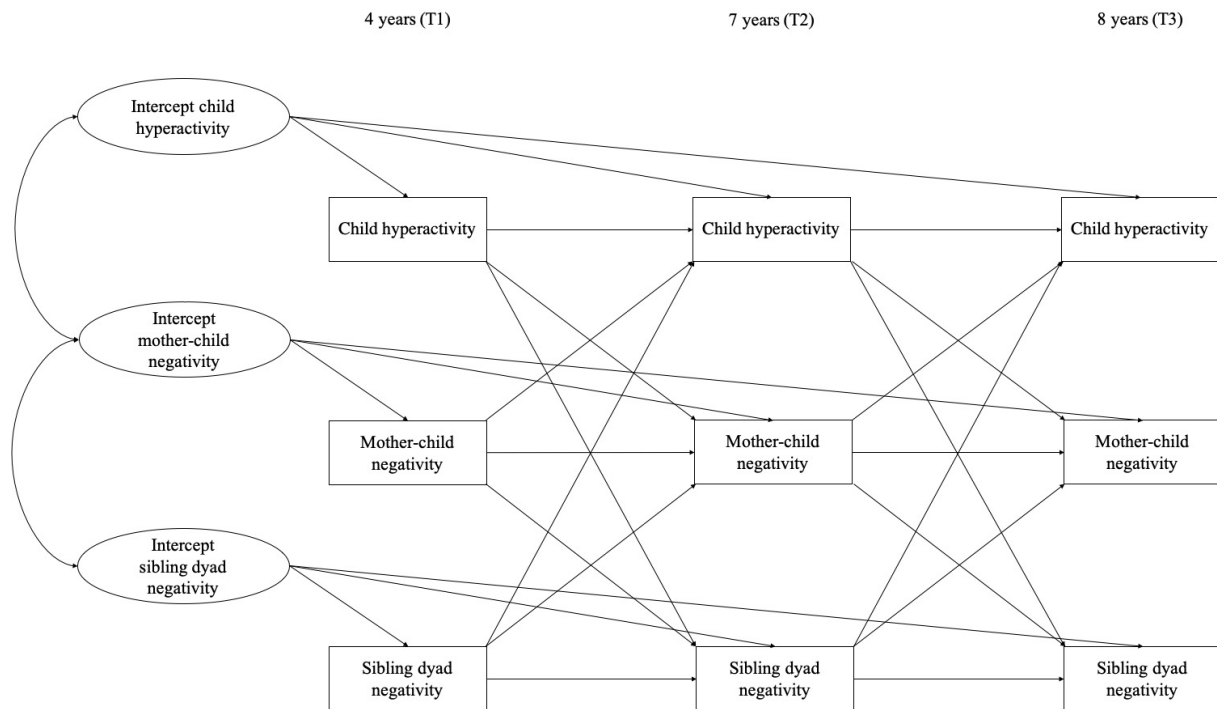
Intercept associations ( $\beta$ )	Unstandardized Coefficient		<i>SE</i>		<i>p</i>	
Hyperactivity ↔ Mother-child	.223*		.012		< .001	
Hyperactivity ↔ Sibling dyad	.241*		.016		< .001	
Mother-child ↔ Sibling dyad	.039*		.002		< .001	
Control variables	Unstandardized Coefficient		<i>SE</i>		<i>p</i>	
Gender → Intercept of hyperactivity	.818*		.062		< .001	
Gender → Intercept of mother-child	.007		.008		.356	
Gender → Intercept of sibling dyad	-.024*		.011		.035	
Autoregressive paths ( $\beta$ )	T 1—2	<i>SE</i>	<i>p</i>	T 2—3	<i>SE</i>	<i>p</i>
Hyperactivity	-.058	.050	.254	.361*	.031	< .001
Mother-child	-.032	.039	.408	.236*	.031	< .001
Sibling dyad	.035	.044	.421	.256*	.033	< .001
Cross-lagged effects ( $\beta$ )	T 1—2	<i>SE</i>	<i>p</i>	T 2—3	<i>SE</i>	<i>p</i>
Hyperactivity → Mother-child	.001	.005	.823	.003	.004	.481
Hyperactivity → Sibling dyad	-.003	.007	.653	-.009	.006	.106
Mother-child → Hyperactivity	-.305	.270	.258	.711*	.215	.001
Mother-child → Sibling dyad	.040	.046	.387	.039	.043	.363
Sibling dyad → Hyperactivity	-.415	.222	.062	.173	.154	.261
Sibling dyad → Mother-child	-.031	.028	.270	.055*	.055	.009
Model Fit-Indices	$\chi^2(9) = 33.83$ ; RMSEA = .025 (.016-.034); CFI = .998; TLI = .991; SRMR = .009					

*Note:* Reported coefficients reflect the unstandardized model results. \* $p < .05$  (two-tailed).



**Figure 1**

*Conceptual depiction of the RI-CLPM examining associations between child hyperactivity, mother-child negativity, and sibling dyad negativity*



*Note:* Within-time covariances among construct residuals were included in the model, as were covariances between residuals of child hyperactivity, mother-child negativity, and sibling dyad negativity within and across time, but are not depicted for the sake of clarity. The autoregressive cross-lagged paths were computed using the residual variance variables for each construct but are not depicted for the sake of clarity. Factor loadings for the intercept variables were set to one. Additional details on the computation of the RI-CLPM can be found in Hamaker et al. (2015).

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Although these data are available and have been used by many different people and labs, our study is the first, as far as we are aware, that has investigated bidirectional associations between child hyperactivity and mother-child as well as sibling dyad negativity, as they unfold over time within a given family. Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee (Institutional Review Board No. 00003312) and the local research ethics committees (Bristol and Weston Health Authority, Southmead Health Authority, and Frenchay Health Authority).

Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time. The informed consent obtained from ALSPAC participants does not allow the data to be made freely available through any third party-maintained public repository. However, data used for this submission can be made available on request to the ALSPAC executive. The ALSPAC data management plan describes in detail the policy

regarding data sharing, which is through a system of managed open access. Full instructions for applying for data access can be found here: <http://www.bristol.ac.uk/alspac/researchers/access/>. The ALSPAC study website contains details of all the data that are available (<http://www.bristol.ac.uk/alspac/researchers/our-data/>).

This study's design and its analysis were not preregistered. The analysis code for this study is openly available at <https://osf.io/e3vxq/>

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HYPERACTIVITY, MOTHER-CHILD AND SIBLING NEGATIVITY

**Table S1**

*Dropout analysis comparing completers to non-completers across the key demographic and focal sturdy variables*

	Completers <sup>a</sup> <i>n</i> (%) or <i>M</i> ( <i>SD</i> )	Non-completers <i>n</i> (%) or <i>M</i> ( <i>SD</i> )	<i>X</i> <sup>2</sup> -value or <i>t</i> -value	df	<i>p</i>
Maternal age	28.85 (4.56)	30.33 (4.18)	-9.68	4427	.007
White ethnic background	922 (21.8)	3312 (78.2)	22.94	1	<.001
Maternal highest education	619 (19.5)	2554 (80.5)	51.84	1	<.001
1 <sup>st</sup> Marriage	729 (22.7)	2690 (77.3)	15.92	1	<.001
Child hyperactivity T1	0.63 (0.48)	0.99 (0.06)	-42.74	4427	<.001
Mother-child negativity T1	0.59 (0.49)	0.94 (0.23)	-31.68	4427	<.001
Sibling dyad negativity T1	0.59 (0.49)	0.95 (0.21)	-33.85	4427	<.001

*Note:* <sup>a</sup>Completers: Mother-child dyads who did not participate across all three time points. <sup>b</sup>Non-completers: Mother-child dyads who participated across all three time points.