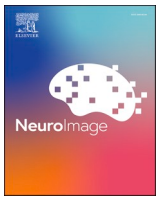
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Parental mental illness and child brain structure: A diffusion MRI study of emotion regulation related pathways

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ABSTRACT

Background: Children of parents with mental illness (at-risk) face an increased risk for developing mental health issues themselves than children of healthy parents (controls), yet the neurobiological underpinnings remain unclear. This study explored differences in white matter (WM) between at-risk children and controls, focusing on brain pathways related to emotion regulation and the associations between parenting behaviours, stress, and WM microstructural properties.

Methods: WM properties were assessed via diffusion-weighted imaging in 64 children (28 at-risk, 36 control) across five emotion-related pathways. Multiple regression analyses examined pathway alterations between groups and associations between parenting behaviours, parenting stress, emotion regulation and WM pathways across all participants beyond effects of group membership, controlling for socioeconomic status, age, sex, scanning site and global WM volume.

Results: At-risk children showed alterations in the right inferior longitudinal fasciculus (ILF), indicated by higher fractional anisotropy and lower mean diffusivity. Across groups, dysfunctional parenting was associated with widespread WM alterations, including the corpus callosum (CC) genu, bilateral dorsal cingulum, bilateral ILF, in parts of the superior longitudinal fasciculus and left uncinate fasciculus. Additionally, lower partnership support was specifically linked to alterations in the CC genu. No significant associations were found between WM microstructure and children's self-reported emotion regulation.

Conclusions: These findings show potential evidence of altered WM pathways in children at-risk for mental illness, particularly in emotion regulation related pathways. Additionally, parenting behaviour and stress were associated with WM microstructure in children, underscoring potential influence of early environmental factors on brain development. Longitudinal and replication studies are needed to clarify causal mechanisms and long-term effects.

1. Introduction

In recent years, the transmission of mental illnesses among generations and its prevention have increasingly become subject of research (e.g. Döhnert and Wiegand-Grefe, 2021; Duffy et al., 2023; Lannes et al., 2021; Sandstrom et al., 2020; Zhang et al., 2020). One

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reason for this interest is the accumulation of mental disorders in families (e.g. Plass-Christl et al., 2018). A cross-national WHO study demonstrated that parental mental illness significantly predicts offspring mental disorders, with children being 1.8 to 4.6 times more likely to develop a disorder (McLaughlin et al., 2012). These children not only face an increased risk of developing the same disorders as their parents (van Santvoort et al., 2015) or mental disorders in general (Mattejat and Remschmidt, 2008) but also experience reduced health-related quality of life (Radicke et al., 2021) and generate up to threefold higher psychosocial social costs compared to healthy children of healthy parents (Waldmann et al., 2021). Understanding the neurobiological and neurophysiological basis of this transmission could help identify protective factors and thus improve affected families' well-being and reduce health service costs.

Emotion regulation (ER) is a transdiagnostic process implicated in several mental disorders, including major depressive disorder, anxiety, bipolar disorder, eating disorder, and alcohol-related disorders (Aldao et al., 2010; Fernandez et al., 2016). Recent research has highlighted its role in both the development and maintenance of psychopathology (e.g. Brenning et al., 2022; Cludius et al., 2020; Kraft et al., 2023, 2024; Lincoln et al., 2022). Neurobiological studies reveal that ER is associated with brain activity in prefrontal, temporal and limbic areas (reviews Morawetz et al., 2025; Ochsner et al., 2012; Picó-Pérez et al., 2017) and structural properties of grey matter (Ahmed et al., 2015; Fortea et al., 2024). Beyond functional and morphological correlates, ER is related to white matter (WM) pathways – the anatomical basis of brain connectivity (Bullock et al., 2022; Fields, 2008). WM pathways consist of axons bundles that connect distinct brain areas, enabling efficient and fast signal transmission (e.g. Catani and Thiebaut de Schotten, 2008; Fields, 2010, 2015). Neuroplasticity research has shown that functional activity influences the myelination of axons (Ishibashi et al., 2006; Stevens et al., 2004; Wake et al., 2011; Yang et al., 2012), suggesting a dynamic interplay between brain function, grey matter (Neufeld et al., 2022; Smith et al., 2023) and WM microstructural properties (Fields, 2015). Transdiagnostic meta-analyses have identified consistent WM alterations across mental disorders, particularly in fronto-temporal and fronto-parietal tracts (Jenkins et al., 2016). Five key pathways have been consistently described in relation to mental disorders and ER: First, the uncinate fasciculus (UF) connects the anterior temporal lobe (including the amygdala) with the prefrontal cortex and is involved in ER, particularly reappraisal (Zuurbier et al., 2013). Altered UF microstructural properties have been linked to impaired emotional empathy (Oishi et al., 2015) as well as impaired emotional expression recognition (Coad et al., 2020). Alterations are also associated with ER in paediatric mental disorders (Versace et al., 2015; for a review see von der Heide et al., 2013). Second, the corpus callosum (CC) connects the left and right prefrontal regions and is essential for coordinating cognition, emotion and behaviour (e.g. Versace et al., 2015). Alterations in the CC, particularly the forceps minor, are associated with suicidal behaviour (e.g. Lischke et al., 2017; Zhang et al., 2019) and a range of mental disorders including major depressive disorder, bipolar disorder, obsessive-compulsive disorder (e.g. Koshiyama et al., 2020; Piras et al., 2021; Sagarwala and Nasrallah, 2020). Third, the superior longitudinal fasciculus (SLF) connects the frontal to the parietal lobe and is anatomically close to the arcuate fasciculus and the cingulum (Janelle et al., 2022). SLF alterations have been observed in social anxiety disorder (Tükel et al., 2017) and are associated with individual differences in reappraisal use (Vitolo et al., 2022). Fourth, the inferior longitudinal fasciculus (ILF) connects the occipital cortex with the temporal lobe (Latini et al., 2017) facilitating the integration of visual and emotional information, for example in face memory and face recognition tasks (Unger et al., 2016). Alterations in ILF were found in generalized anxiety disorder (Liao et al., 2014) and are associated with the severity of social anxiety symptoms (Parsaei et al., 2024; note that this review included the study from Tükel et al., 2017). Finally, the cingulum as part of the limbic system plays a key role in emotion processing and regulation and was found to be associated with ER in paediatric mental disorders (Hung et al., 2020; Versace et al., 2015). Together, these pathways – the SLF, UF, CC forceps minor, ILF and cingulum – appear essential for adaptive ER, though the direction of alterations varies across studies. Importantly, these tracts are not exclusive to ER but also support broader cognitive and affective functions: language and attention (SLF; Catani et al., 2005; De Schotten et al., 2011), semantic memory (UF; von der Heide et al., 2013), pain processing and conflict monitoring (cingulum; Shackman et al., 2011), interhemispheric communication (CC; Gazzaniga, 2000), and visual-spatial integration (ILF; Zemmoura et al., 2021). This multifunctionality implies that WM alterations in these pathways may not be ER-specific but could reflect broader neurobiological adaptations or vulnerabilities across cognitive domains.

Notably, studies in at-risk children – primarily offspring of parents with depression or bipolar disorder – have identified alterations in these same pathways despite methodological variability (e.g., tract-based spatial statistics vs. pathway reconstruction) and wide age ranges (infancy to adulthood), (Cattarinussi et al., 2021). For example, studies of offspring of bipolar parents consistently report alterations in SLF (Frazier et al., 2007; Ganzola et al., 2018; Versace et al., 2018), cingulum (Ganzola et al., 2018; Roberts et al., 2022; Rozovsky et al., 2021) and CC (Ganzola et al., 2018) and show delayed developmental processes among healthy offspring of bipolar parents in the CC and ILF (Versace et al., 2010). A transdiagnostic comparison of offspring of parents with bipolar disorder, depression, and ADHD revealed common WM alterations in the forceps minor, cingulum, UF, and ILF (Versace et al., 2018).

However, the direction of these alterations is mixed: some studies report reduced fractional anisotropy (FA) in at-risk groups (e.g. Frazier et al., 2007; Ganzola et al., 2018; Hung et al., 2017; Versace et al., 2010), while others find increased FA (e.g. Roberts et al., 2022; Rozovsky et al., 2021; Sullivan-Toole et al., 2023; Winter et al., 2022). This bidirectional pattern of FA alterations may reflect diverse neural mechanisms in response to parental mental illness influenced by factors such as developmental stage, environmental context, and individual differences in emotional processing. Reduced FA has been associated with disturbed myelination or white matter properties, such as in controls individuals who experienced early-life adversity (Frodl et al., 2012), while increased FA (in at-risk for depression and early traumatization) has been hypothesised to reflect potential compensatory plasticity in response to adversity (Fields, 2015; Frodl et al., 2012). For example, a study of adults with familial risk for major depression showed higher FA values of at-risk healthy participants in forceps minor and SLF compared to healthy controls (Winter et al., 2022), probably representing a compensatory mechanism associated with resilience. Similarly, Sullivan-Toole and colleagues (2023) found a steeper age-related FA increase in the cingulum, SLF and CC among youth with a maternal depression history, potentially reflecting adaptive plasticity. In contrast, Hung and colleagues (2017) observed age-related FA decreases in the frontal-limbic pathways (including CC and cingulum) in children at risk for major depression, possibly indicating delayed or disrupted development.

A complementary explanation for contradictory FA findings emerges from functional neuroimaging: Kjærstad et al. (2025) identified two subgroups among first-degree relatives of bipolar individuals, with one subgroup showing hyperactivation and the other hypoactivation in ER-related brain regions. This functional heterogeneity suggests that children of parents with mental illness may develop divergent emotional responses – either hyper- or hypoactive – depending on factors such as age at adversity onset, genetic predisposition, or environmental influences. Such variability in functional might contribute to the mixed WM findings observed in structural studies. For instance, hyperactive emotional processing has been hypothesized to correlate with increased FA, potentially reflecting enhanced connectivity due to heightened neural demand, while hypoactive processing could be associated with reduced FA, possibly indicating less coherent fibre organization.

Importantly, these associations are correlational, and the directional interpretation of FA remains ambiguous without convergent evidence from multimodal imaging or histological validation (Jones et al., 2013). The direction of FA changes may thus reflect diverse adaptive or maladaptive processes rather than a uniform improvement or impairment of fibre integrity. Together, these structural and functional insights highlight the need to consider individual differences in emotional reactivity and adaptive mechanisms when investigating WM alterations in at-risk populations.

The relationship between parental mental illness and children's WM development is critically mediated by parental stress, parenting behaviour, and children's emotion regulation (ER) strategies, which interact in complex ways to shape neurobiological outcomes (e.g. reviews Belsky and De Haan, 2011; Rutherford et al., 2015). Prolonged parental stress – defined as aversive psychological and physiological responses to parenting demands (Deater-Deckard, 2008) – negatively affects parenting quality, parent-child interactions and ultimately children's emotional and cognitive development (e.g. Crnic and Greenherg, 1990; Deater-Deckard, 2008; Guajardo et al., 2009). For example, increased parental stress is associated with insecure attachment (Anthony et al., 2005) and behavioural problems in children (Barroso et al., 2018). On a neuronal level, parental stress influences children's brain development through multiple pathways. Epigenetic mechanisms triggered by perinatal stress can alter gene expression, thereby impacting brain maturation (Chan et al., 2018). Further, prenatal exposure to maternal stress or early-life adversity is associated with altered microstructure of fronto-limbic pathways, such as the UF and cingulum, in preterm neonates (Lautarescu et al., 2020) and even in healthy term-born neonates (Lean et al., 2022; Lugo-Candelas et al., 2023). These alterations may reflect accelerated maturation – a potential adaptive response preparing the child for challenging rearing environments. Longitudinal studies further highlight the lasting impact of early parenting: Harsh parenting at age 3 predicts reduced cerebral WM volume at age 10 (Hidalgo et al., 2022), whereas positive parenting can buffer stress effects: highly stress-reactive children who receive greater positive parental affect show WM structures more similar to low stress-reactive peers (Sheikh et al., 2014).

A key aspect linking parental stress to children's brain development is the child's own ER, which is profoundly shaped by parent-child interactions (Silvers, 2022). Dysfunctional parental ER – common in mental illness – often leads to maladaptive ER strategies in children, such as withdrawal, rumination, or aggression (Halligan et al., 2013; Hosman et al., 2009; Lin et al., 2023; Morelen et al., 2016; Peris and Miklowitz, 2015; Silk et al., 2006; Zitzmann et al., 2023) that increase vulnerability to mental health problems (e.g. Aldao, 2013; Aldao et al., 2010; Grob et al., 2006; Sheppes et al., 2015). These strategies are also associated with altered WM microstructural properties: For example, childhood maltreatment and parental verbal abuse have been linked to altered WM in ER-related pathways (Choi et al., 2009; Lim et al., 2019). Conversely, supportive parenting promotes adaptive ER processes, which are reflected in cortical thinning and reduced activation in regions the salience and implicit ER networks in youth (Tan et al., 2020). Higher maternal attachment security, for instance, correlates with enhanced microstructural properties of the child's inferior fronto-occipital fasciculus, UF, SLF, and cingulum (Serra et al., 2015).

To sum up, parental mental illness may impair children's ER directly, i.e. through genetic predispositions or exposure to dysfunctional parenting, and indirectly, by altering brain development via chronic stress. Accordingly, findings underscore the intricate interplay between parental stress, ER, and children's WM development. However, previous studies have primarily focused on offspring of depressed or bipolar parents, and are often constrained by methodological limitations, such as relying on tract-based spatial statistics or adult templates for child data. Moreover, the period of late childhood and adolescence remains understudied though it is a critical window for both brain maturation and the emergence of psychopathology.

To address these gaps, the present study measures WM microstructural properties in five ER-related pathways (SLF, ILF, UF, cingulum, and CC forceps minor) in healthy children aged 7–16 years. We compare children of acutely ill parents (at-risk group) with children of healthy parents (controls) to explore associations between parental stress and behaviour, children's emotion regulation (ER) strategies, and WM development across parental disorders. To assess WM microstructure, we use FA and mean diffusivity (MD) as key metrics. FA quantifies the degree of anisotropic diffusion, reflecting the coherence of water movement along fibre tracts, while MD indicates the overall magnitude of water diffusion, with higher values suggesting less restricted diffusion (O'Donnell and Westin, 2011; Soares et al., 2013). FA and MD have a complementary sensitivity to different tissue properties and altered FA/MD ratios are often interpreted as indicators of disrupted fibre integrity. Nevertheless, given the reported heterogeneity in FA findings across at-risk populations, and the potential for overinterpretation (see e.g. Figley et al., 2022), directional predictions and specific interpretations of underlying tissue properties based on FA and MD are avoided.

Considering these challenges, this study pursues three key objectives: First, it seeks to clarify the direction and underlying mechanisms of WM alterations in at-risk children, while accounting for the heterogeneity observed in previous research. We hypothesize that children in the at-risk group show altered WM microstructure in SLF, ILF, UF, cingulum and CC forceps minor compared to controls (H1). Second, it intends to disentangle the distinct contributions of parental behaviour and stress to children's WM microstructural properties while controlling for group effects, i.e. investigating the effect of parental behaviour and stress in such a way that the parental disorder adds to the variance, but the direct influence of the parental disorder is controlled for. We hypothesize that dysfunctional self-reported parental behaviour (H2) and stress (H3) are associated with altered microstructural properties of the children's WM pathways. Third, it explores how children's adaptive and maladaptive ER strategies relate to WM microstructure (H4), potentially explaining individual differences in resilience or vulnerability. We expect that the children's emotion regulation strategies, as reported by themselves and their parents, are associated with microstructural properties of WM pathways across all children.

2. Method

2.1. Sample

The data collection was part of the *children of mentally ill parents at-risk evaluation* project (COMPARE; # 01GL1748C/01GL1748E). In this multicentre study, several subprojects investigated the mechanisms of familial transmission of mental disorders. A detailed description of the general project methodology can be found in Christiansen et al. (2019). MRI data was collected at the Ruhr University Bochum (Germany) and the Bender Institute of Neuroimaging Giessen (BION, Germany) as part of the COMPARE emotion sub-project carried out by the Technical University Dortmund and the Justus Liebig University Giessen. The control group was recruited using flyers, newspaper articles and university email distribution lists. The at-risk group was recruited through cognitive behavioural outpatient centres, namely Behavioural Therapy Outpatient Clinic Giessen and Research and Treatment Centre for Mental Health (FBZ) in Bochum.

Several inclusion and exclusion criteria were set for the at-risk and control groups: Firstly, in the at-risk group, children had to be between 7 and 16 years old and one parent had to have at least one current DSM-5 (APA, 2013) mental disorder diagnosis (assessed via a semi-structured diagnostic interview – DIPS; Margraf et al., 2017), as assessed in an initial interview at the project-related outpatient clinics by qualified psychologists, who were in training to become licensed psychotherapists. Secondly, the affected parent was seeking treatment as part of the COMPARE study; however, no treatment had yet begun at the time of

data collection, including medical treatment. Thirdly, the children were excluded if they themselves were undergoing treatment at the time of inclusion, but not if they began treatment during the project. Additionally, in the at-risk group, one parent was interviewed on behalf of the child using the parent version of the semi-structured diagnostic interview (children-DIPS; Schneider et al., 2017) to ensure that the child was free from a mental disorder.

In the control group, firstly, care was taken to ensure that only children (aged 7–16) and parents without a mental disorder participated. This was achieved by having both parents report that they had not received psychiatric or psychotherapeutic treatment in the past and had never been diagnosed with a mental disorder. Secondly, both parents (or only one if separated) completed the Brief Symptom Inventory (BSI; Kliem and Brahler, 2017) to screen for possible undiagnosed mental health problems. If one parent exceeded a threshold value for the BSI (Global Severity Index T-score $\geq$ 63; Kliem and Brahler, 2017), the families were excluded. Similarly, to screen for possible undiagnosed mental health problem in the child, one parent completed the child behaviour checklist (CBCL; Döpfner et al., 2014) for their participating child; if a threshold value was exceeded (CBCL T_{sum}-Score $\geq$ 63), the parent was interviewed using the parent version of the children-DIPS and the family was excluded in case of a probable diagnosis of the child.

Further exclusion criteria for the two groups were inadequate German language skills and standard MRI exclusion criteria, such as pacemakers, prostheses and braces.

A total of 80 children were invited to participate in the scanning session as they met the inclusion criteria. Of these children, six withdrew from scanning before a diffusion-weighted sequence could be obtained. Data from an additional ten children were excluded due to quality concerns (see the Quality Control section), resulting in data from 64 children, the final sample. Multiple children of a family were allowed to participate; in the at-risk group, four families each had two children participating, while in the control group, five families had two children each participating. Among the mentally ill parents in the at-risk group, the majority of the primary diagnoses according to DSM-5 (APA, 2013) were depressive disorders (49.99 %), followed by trauma- and stress-related disorders (28.57 %), anxiety disorders (17.85 %), and one parent with a somatic symptom disorder (3.57 %). Regarding comorbidity within the affected parents: Fifteen parents had one diagnosis, seven parents had two diagnoses and six parents had between three to five diagnoses. Comparable to the primary diagnoses, additional diagnoses were predominantly depressive disorders, specific phobia, anxiety disorder and panic disorder. The characteristics of the children for each group in the final sample are shown in Table 1. Differences in age, CBCL T-Scores (Döpfner et al., 2014) and parental socioeconomic status (Lampert et al., 2018) between the at-risk group and the control group were calculated using Welchś *t*-test. There was a significant difference in socioeconomic status between the at-risk and control group (95 % - CI [−5.20, −2.22], $t(42.73) = -5.03$, $p < .001$). Differences for sex between the at-risk and the control group were calculated using Pearson Chi^2-Test. Differences in education status were tested using a non-conditional permutation test based on 100.000 Monte Carlo resamples to handle small cell frequencies (see Supplements Table 4). The analysis revealed no significant association between group and education level ($\chi^2(4) = 4.13$, $p = .40$), suggesting the groups did not differ in their educational status.

Importantly, the research questions examining associations between the WM microstructure of the five predefined tracts and parental behaviour, parental stress and ER were analysed across all children. However, questionnaire data were missing for 16 families in the at-risk group and one family in the control group, resulting in complete data for 47 participants. The characteristics of this correlation-hypotheses sample are presented in Table 2.

Table 1
Description of the sample of participating children in the two groups "at-risk" and "control". Children of the at-risk group means that at least one parent had a mental health disorder. None of the parents of the children in the control group had a mental disorder.

	at-risk	control	*p*-value	effect size
N (female)	28 (9)	36 (17)	.336[1]	0.15[3]
Age (*M, SD*)	11.20 (2.91)	11.34 (2.35)	.848[2]	0.05[4]
CBCL T_{sum}-Score (*M, SD*)	49.04 (10.47)[5]	46.17 (15.55)	.396[2]	0.21[4]
SES of parents (*M, SD*)	14.89 (3.42)	18.59 (2.13)	< .001[2]	1.34[4]

Note. CBCL = Child Behaviour Checklist (Döpfner et al., 2014), SES = socioeconomic status of the parents (Lampert et al., 2018), *M* = mean, SD = standard deviation

[1] Pearson's Chi-squared for sex distribution
[2] Welchś *t*-test.
[3] Cohenś w.
[4] Cohenś d.
[5] four values in the at-risk group are missing from CBCL T_{sum} calculation.

2.2. Procedure and measures

The study protocol was approved by the local Ethics Committees of the Justus Liebig University Giessen and the Technical University Dortmund. All participants provided written informed consent in accordance with the guidelines of the ethical standards of the Declaration of Helsinki and its later amendments. Parents were informed in detail about the content and procedure of the study by telephone and information letter. Accordingly, the children were informed about the content and procedure in a developmentally appropriate way.

The measurement was divided into two sessions that took place within one week. At the first session, a mock scanning procedure was applied to accommodate the children with the scanner and scanning procedure. The children took part in a handedness and reading ability test; the procedure was explained in detail in a developmentally appropriate way. At the second session, the actual measurement took place. First, parents and children signed the consent form and the children were prepared for the MRI scan. The children entered the scanner and an experimenter stayed with the children in the scanner room to make sure they were comfortable and to remind them to move as little as possible, if necessary. A structural image was then acquired (approx. 04:30 min), followed by functional sequences in which the children performed an emotion labelling task (approx. 23 min), and finally a diffusion-weighted sequence (approx. 07:30 min). The emotion labelling

Table 2
Description of the total sample used for associations between the WM properties of the five predefined tracts and parental behaviour, parental stress and emotion regulation.

	Dimension	total sample
N (female)	-	47 (18)
Age (*M, SD*)	-	11.00 (2.40)
SES of parents (*M, SD*)	-	17.64 (2.67)
CBCL T_{sum}- Score (*M, SD*)	-	46.58 (13.64)
EFB (*M, SD*)	total score	2.45 (0.50)
ESF (*M, SD*)	parental stress	18.23 (11.96)
	role restriction	8.19 (6.21)
	social support	11.96 (5.06)
	partnership	15.96 (4.45)
FEEL Adaptive (*M, SD*)	parent-report	34.09 (10.27)
	self-report	37.26 (11.54)
FEEL Maladaptive (*M, SD*)	parent-report	19.58 (8.30)
	self-report	20.26 (7.42)

Note. CBCL = Child Behavior Checklist (Döpfner et al., 2014), SES = socioeconomic status of the parents (Lampert et al., 2018), *M* = mean, SD = standard deviation, EFB = parenting skill questionnaire (Naumann et al., 2010), ESF = parental stress questionnaire (Domsch and Lohaus, 2010), FEEL = emotion regulation in children (Greuel and Heinrichs, 2014).

paradigm was adapted from Lieberman et al. (2007) to fit our study sample; those results will be reported in a separate publication.

Complementing the MRI measurements, all participants filled out several questionnaires as part of their participation in the COMPARE emotion project. A questionnaire assessing ER in children and adolescents (FEEL-KJ; Grob et al., 2006; Grob and Smolenski, 2009) was completed by both the child and the parents providing self- and parent-report of the children's ER, divided into adaptive and maladaptive strategies. We used the short version (Greuel and Heinrichs, 2014), which consists of 30 items with a 5-point Likert scale and combines the subscales of emotions into the dimension of adaptive and maladaptive strategies (Cronbach's alpha = 0.69 – 0.91).

Additionally, parents completed two online questionnaires: the parenting skills questionnaire (EFB; Naumann et al., 2010) and the parental stress questionnaire (ESF; Domsch and Lohaus, 2010). The EFB is the German adaption of the English parenting scale (Arnold et al., 1993), consisting of 35 items with a 7-point Likert scale, which can be evaluated on the scales Overreaction, Laxness, and Verbosity as well as on a total score (Cronbach's alpha = 0.59 – 0.80) with higher values indicating higher extent of dysfunctional parenting behaviour. The ESF is a German self-report questionnaire, consisting of 38 items with a 4-point Likert scale, which can be rated on the four scales parental stress (perceived parenting competence, difficulties in interaction with the child), role restriction (perceived limitations due to raising the child), social support (perceived support from social environment, e.g. relatives and friends) and partnership (perceived support from the partner, agreement on parenting issues; note that all participating parents had a partner living with them in the same household at least half of the week; Cronbach's alpha = 0.76 – 0.92).

The parents' socioeconomic status was calculated according to Lampert et al. (2018), using education, occupation, and net household income data, coded according to a scheme resulting in a range from 3 to 21. Socio-demographic information about the children, such as age, sex, whether siblings participated, and education level, was also collected.

To assess children's psychopathology, and to check exclusion criteria in the control group, the German parent-report version of the Child Behaviour Checklist 6–18R (Döpfner et al., 2014) with 113 items on a 3-point Likert scale was used in both groups. Items are aggregated into three superordinate scales: externalizing problems, internalizing problems, and total problems (Cronbach's alpha = 0.82 – 0.93; Döpfner et al., 2014). The German self-report version of the Brief Symptom Inventory (BSI; Kliem and Brahler, 2017) with 53 items on a 5-point Likert scale was used to assess parental psychopathology. For the control group a T-Score above 63 leads to the exclusion of the family. Items were aggregated into a Global Severity Index (Cronbach's alpha = 0.97). The semi-structured diagnostic interview DIPS (Margraf et al., 2017) was used to assess mental disorders according to DSM-5 (APA, 2013). Additionally, the parent version of the child semi-structured diagnostic interview (child-DIPS; Schneider et al., 2017) was used. Previous studies report high interrater reliability for both interviews around $0.68 < \kappa < 0.92$ (e.g. Kröber et al., 2023; Neuschwander et al., 2013). All data from questionnaire were collected before the scanning sessions.

2.3. Image acquisition and preprocessing

MRI measurements from the Technical University Dortmund were carried out at the Ruhr University Bochum using Phillips Achieva 3T Data from BION at the Justus Liebig University Giessen was collected using Siemens Prisma 3T Using the Achieva the DTI protocol consisted of two measurements, anterior–posterior (AP) and posterior–anterior (PA). The AP DTI image protocol was measured with the following settings: TR/TE = 8530 ms/ 89 ms, 70 slices, slice thickness = 2 mm, field of view (FoV) = 248 mm, number of excitations = 1, and spatial resolution = 2 × 2 × 2 mm, diffusion gradient (b value) = 1000 s/mm^2, duration = 5 min 24 s. Regarding the diffusion gradient, a DTI diffusion mode with 33 directions and 2 weightings was used. The same settings were used for the reference images (b value = 0 s/mm^2) in the PA direction (duration = 0 min 38 s). Using the Prisma the DTI protocol consisted of two measurements, anterior–posterior (AP) and posterior–anterior (PA). The AP DTI image protocol was measured with the following settings: TR/TE = 8500 ms/ 83 ms, 70 slices, slice thickness = 2 mm, field of view (FoV) = 248 mm, number of excitations = 1, and spatial resolution = 2 × 2 × 2 mm, diffusion gradient (b value) = 1000 s/mm^2, duration = 5 min 40 s. Regarding the diffusion gradient, the Free Diffusion Mode with 33 directions and 2 weightings was used. The Free Diffusion Mode was chosen because the directions were set to be as identical as possible between the scanners, which has been tested and verified (see supplements comparison of dwi directions). The same settings were used for the reference images (b value = 0 s/mm^2) in the PA direction (duration 1 min 59 s).

2.3.1. DTI pre-processing & quality control

The two-dimensional DICOM files of each brain were organized into volumetric three-dimensional NIfTI files using dcm2niix converter (Li et al., 2016). Tracts constrained by underlying anatomy (Tracula; FreeSurfer version 7.4.1; Maffei et al., 2021; Yendiki et al., 2011, 2014) was used with default configuration parameters (if not otherwise noted) to automatically reconstruct all 42 major WM pathways including: SLF, ILF, UF, cingulum and CC forceps minor (hereafter referred to as the CC genu, as labelled in the TRACULA atlas). Tracula is one of the well-validated approaches that automate white matter bundle segmentation with an end-to-end pipeline and has been successfully used for various clinical applications (Andica et al., 2023). The following steps comprise the preprocessing in a nutshell: image corrections (FSL topup & eddy correction), intra-subject registration (freesurfer bbregister), tensor fitting (FSL dtifit), inter-subject registration (ANTs symmetric normalization), estimation of anatomical neighbourhood priors for the trajectories of interest (freesurfer Tracula), ball and stick model fitting (FSL bedpostx), reconstruction of the trajectories of interest (freesurfer Tracula).

Quality control was performed based on the guidelines from He et al. (2021). In addition, any images that exceeded two standard deviations for any of the four quality measures (average translation, average rotation, percent of bad slices, average dropout score) were excluded (7 subjects). Most of the images flagged visually also had quality measure values exceeding two standard deviations. Three additional participants were excluded due to severe artifacts in both the raw and eddy-corrected data. Therefore, a total of 10 subjects were excluded due to poor quality: four from the at-risk group and six from the control group. A more detailed description of the pre-processing, quality control process and metrics, software packages and correlation heatmaps can be found in the supplement. Because data were acquired at two scanner sites, acquisition protocols were aligned across sites to achieve comparable diffusion MRI measurements. To complement protocol harmonization, mean white-matter SNR from Tracula (i.e. SNR of dwi intensities within WM only) was averaged across each subject and then compared between sites. Mean SNR was descriptively similar but significantly different (site Giessen: $M = 3.45$, $SD = 0.13$, $N = 41$; site Bochum: $M = 3.38$, $SD = 0.13$, $N = 23$; Welch's $t = 2.04$, $p = .047$, $d = 0.53$), so scanner site was included as a covariate in all analyses. To evaluate whether more elaborate harmonization (e.g. neurocombat; Fortin et al., 2017) was necessary beyond including scanner site as a covariate, we followed previous methodological work that provides practical guidelines on when and how to apply ComBat-based harmonization (Orlhac et al., 2022). We visually inspected the cumulative distribution functions and probability density functions of FA and MD for each scanner site and tract. In addition, we applied two-sample Kolmogorov–Smirnov tests, which assess whether two samples are drawn from the same underlying distribution. Both the visual inspection and the Kolmogorov–Smirnov tests indicated no site-related differences in the distributions of FA and MD. Therefore, modelling scanner site as a covariate was deemed sufficient, and no further harmonization beyond this covariate was

performed (see supplements scanner site comparison).

Next to the measures FA and MD, axial and radial diffusivity (AD and RD) can provide additional clues for interpreting (i.e. along and perpendicular to the main fibre orientation) and are therefore reported as metrics in the supplement.

2.4. Second level analysis

The sample included sibling pairs (four in the at-risk group, five in the control group), as noted above. Methods that could account for this clustering, such as multilevel models, are not feasible because the number of families is too small to obtain reliable family-level variance estimates (e.g. Maas and Hox, 2005). Removing siblings by randomly retaining only one child per family or creating matched subsamples would have further reduced the sample and thus substantially weakened statistical power (see supplements section power analysis). This limitation is discussed when interpreting the findings.

The following Tracula derived tracts (right and left hemisphere except for CC genu) were included in the analysis: SLF1, SLF2, SLF3, ILF, UF, CC genu, Cingulum (dorsal and ventral), totaling 15 WM pathways also called region of interests (ROIs). For each pathway, separate multiple regression analyses tested differences in mean FA and mean MD between the Groups controlled for covariates of no interest to ensure that these covariates do not account for any of the findings. Mean FA and mean MD values across each region of interest served as dependent variables, with Group (at-risk vs. control) as the predictor. As covariates of no interest age, socioeconomic status, sex (male vs. female), scanner sites (Giessen vs. Bochum) and global WM volume was used. The covariates were chosen since they were previously reported to have an influence on WM pathways (e.g. Kochunov et al., 2012; Koirala et al., 2021; Lebel et al., 2012; Simmonds et al., 2014; Ursache and Noble, 2016; Versace et al., 2018). Global WM volume was derived for each participant from the freesurfer segmentation output using the Total-WhiteMatterVolume measure. It was included as covariate of no interest to evaluate whether observed effects represent more localized changes of WM pathway properties, as hypothesised, or rather reflect unspecific global white matter differences between groups. As preliminary analyses revealed no significant differences between the groups across movement parameters (average translation, average rotation; all $p > .05$), motion parameters were not included as additional covariates in the second-level models. Data were z-transformed, except the categorical predictors (sex, Group) which were dummy coded (0,1). To control the family-wise error rate among correlated white matter tracts (see supplements section correlation heatmaps), the effective number of independent tests (M_{eff}) was determined following the method described by Nyholt (2004). This approach utilizes the spectral decomposition of the correlation matrix to quantify the degree of redundancy between variables based on the variance of the observed eigenvalues. This correction has been validated for psychological research (Derringer, 2018) and successfully applied in neuroimaging studies with similar difficulties (e.g. Grasby et al., 2020; Hibar et al., 2015) to account for multiple non-independent tests arising from correlated measures. Therefore, it provides a more biologically appropriate adjustment than a Bonferroni correction, which assumes independence and would be overly harsh for these data. The adjusted significance level was subsequently calculated by dividing the nominal alpha ($\alpha = 0.05$) by the estimated number M_{eff} of independent tests. In the present study, the spectral decomposition across all tracts and diffusion measures (FA, MD) resulted in an $M_{eff} = 10$ and therefore for an adjusted significance level $\alpha_{corrected}$ of 0.005. Because of the confound between Group effect and socioeconomic status, the calculations were again carried out without socioeconomic status as covariate of no interest (see Table 1).

For the correlations between parental behaviour, parental stress, ER and WM pathways (FA, MD), we used the same model, including age, socioeconomic status, sex, scanner site, global WM volume and Group as covariates of no interest, to examine if there was a correlation (i.e. partial correlation r_p) between the questionnaire (e.g. parenting skill total score) and mean FA or mean MD values of a specific pathway, beyond the potential effect of Group and the covariates of no interest. Following the M_{eff} approach (Derringer, 2018), the corrected $\alpha_{corrected}$ was defined as 0.05 / ($M_{eff\ tracts}$ * $M_{eff\ questionnaire}$) as not only tracts are correlated but also questionnaire dimensions. For parental behaviour (H2; $M_{eff\ questionnaire} = 1$ as total score is used), resulted in $\alpha_{corrected} = 0.005$. For parental stress (H3; four dimensions; parental stress, role restriction, social support and partnership support) and ER (H4; self-reported maladaptive and adaptive strategies, and maladaptive and adaptive strategies reported by the parents) $M_{eff\ questionniare} = 3$ for each questionnaire. This led to $\alpha_{corrected} = 0.0017$. The script containing computations, plots, verification of the assumptions of the statistical tests etc. can be found at github [1], the analysed data can be found on zenodo https://doi.org/10.5281/zenodo.17084310.

3. Results

Multiple regression models were used to examine variations in FA and MD across 5 different WM pathways, first comparing them between the Groups (H1), and second testing their relation to parenting behaviour (H2), parental stress perception (H3), and the children's ER strategies (H4), while controlling for covariates of no interest (SES, age, sex, scanner site and global WM volume). For comparing WM pathway properties between groups, the predictor of interest was Group (H1), while for testing the correlations hypotheses (H2, H3, H4), Group was added as a covariate to look at (partial) correlations across all participants beyond the effect of parental mental illness. All models were checked for statistical assumptions. For example, multicollinearity was assessed using Variance Inflation Factor (VIF). In all computations, all predictor VIFs were below 2.2, indicating that the variance of the estimated coefficients was not substantially inflated by correlations among predictors. Residuals vs. leverage plots were used to identify influential data points, defined as those outside Cook's distance in the plot ($D > 0.5$). These cases were then examined with respect to their externally studentized residuals and leverage (hat) value, as large values indicate that the model fits this observation poorly while simultaneously showing high influence on the regression estimates (e.g. Chatterjee and Hadi, 1986). If necessary, regression models were then additionally calculated without these data points. Only M_{eff} corrected results are reported, except for Table 3 where * indicates M_{eff} corrected results. The M_{eff} adjustment was computed jointly across WM tracts and both diffusion metrics (FA and MD). Note: *M* and *SE* refer to unstandardized estimated marginal means. As SES correlates with the parenting skills total score ($r(45) = -0.41$, $p = .005$), parenting stress in the partnership support dimension ($r(45) = 0.33$, $p = .024$), and maladaptive ER (self-assessed: $r(45) = -0.51$, $p < .001$; parent-assessed: $r(45) = -0.39$, $p = .007$), all calculations were re-run without SES as a covariate of no interest (all other covariates remained in the model).

3.1. Differences in MD and FA between the groups (H1)

No significant differences were found in the SLF, UF, cingulum and CC genu. In contrast, the right ILF showed significant differences in FA and MD: the at-risk group showed higher FA (right ILF FA: $\beta = 0.90$, 95 % CI [0.29, 1.50], $t(57) = 2.95$, $p = .005$, $M = 0.55$, $SE = 0.01$ vs. $M = 0.51$, $SE = 0.01$) and lower MD compared to the control group (right ILF MD: $\beta = -0.93$, 95 % CI [−1.57, −0.31], $t(57) = -2.97$, $p = .004$, $M = 0.00076$, $SE = 0.00002$ vs. $M = 0.00083$, $SE = 0.00001$), indicating higher FA and lower MD values in the right ILF in the at-risk group.

When SES was removed as a covariate of no interest no result survived correction.

Exploratory causal mediation analyses were conducted to examine whether SES accounted for the association between group effect and right ILF microstructure, using the same covariates as in the primary models and 10,000 nonparametric bootstrap samples. In the model with

Table 3
Partial correlations between fractional anisotropy / mean diffusivity and dysfunctional parenting total score, controlled for age, sex, scanner site, global white matter volume and group. Values are shown depending on inclusion of socioeconomic status as covariate and the removed outlier.

			dysfunctional parenting total score with SES covariate		dysfunctional parenting total score without SES covariate		dysfunctional parenting total score with SES - outlier removed		dysfunctional parenting total score without SES - outlier removed	
ROI	Location	Measure	$r_p(39)$	p-value	$r_p(40)$	p-value	$r_p(38)$	p-value	$r_p(39)$	p-value
SLF 1	Right	FA	−0.29	.107	−0.43	.009	−0.49	.009	−0.61	< .001*
		MD	0.36	.046	0.53	.001*	0.61	< .001*	0.76	< .001*
	Left	FA	−0.24	.188	−0.40	.016	−0.46	.010	−0.60	< .001*
		MD	0.29	.125	0.45	.009	0.55	.002*	0.68	< .001*
SLF 2	Right	FA	−0.36	.054	−0.47	.005*	−0.57	.002*	−0.66	< .001*
		MD	0.38	.034	0.53	.001*	0.62	< .001*	0.74	< .001*
	Left	FA	−0.30	.121	−0.40	.020	−0.52	.006	−0.59	< .001*
		MD	0.29	.110	0.47	.006	0.55	.002*	0.70	< .001*
SLF 3	Right	FA	−0.38	.036	−0.48	.003*	−0.63	< .001*	−0.69	< .001*
		MD	0.39	.032	0.52	.001*	0.63	< .001*	0.74	< .001*
	Left	FA	−0.36	.057	−0.46	.006	−0.58	.002*	−0.67	< .001*
		MD	0.33	.071	0.49	.003*	0.57	.001*	0.71	< .001*
ILF	Right	FA	−0.35	.038	−0.46	.003*	−0.59	< .001*	−0.66	< .001*
		MD	0.39	.025	0.56	.001*	0.64	< .001*	0.79	< .001*
	Left	FA	−0.43	.019	−0.52	.001*	−0.67	< .001*	−0.72	< .001*
		MD	0.30	.093	0.48	.004*	0.56	.001*	0.71	< .001*
UF	Right	FA	−0.32	.055	−0.53	.001*	−0.44	.016	−0.64	< .001*
		MD	0.39	.032	0.54	.001*	0.61	.001*	0.74	< .001*
	Left	FA	−0.41	.018	−0.51	.001*	−0.59	.001*	−0.67	< .001*
		MD	0.36	.048	0.53	.002*	0.61	.001*	0.75	< .001*
cingulum dorsal	Right	FA	−0.45	.012	−0.55	.001*	−0.64	< .001*	−0.71	< .001*
		MD	0.38	.036	0.54	.001*	0.64	< .001*	0.77	< .001*
	Left	FA	−0.36	.044	−0.47	.004*	−0.59	.001*	−0.67	< .001*
		MD	0.91	.063	0.50	.003*	0.61	< .001*	0.74	< .001*
cingulum ventral	Right	FA	−0.33	.071	−0.44	.007	−0.48	.012	−0.58	.001*
		MD	0.26	.125	0.47	.004*	0.51	.001*	0.69	< .001*
	Left	FA	−0.26	.115	−0.35	.019	−0.47	.006	−0.52	< .001*
		MD	0.25	.166	0.44	.009	0.54	.001*	0.70	< .001*
CC	Genu	FA	−0.56	.001*	−0.64	< .001*	−0.73	< .001*	−0.79	< .001*
		MD	0.45	.015	0.56	.001*	0.67	< .001*	0.75	< .001*

Note. ROI = Region of interest i.e. WM pathways; Location = right or left hemisphere; FA = fractional anisotropy; MD = mean diffusivity, $r_p()$= partial correlation coefficient with degrees of freedom, SLF = superior longitudinal fasciculus, ILF = inferior longitudinal fasciculus, UF = uncinate fasciculus, CC = corpus callosum; * survived M_{eff} correction: $\alpha_{corrected} = 0.005$; SES = socioeconomic status.

right ILF FA as the outcome, the average causal mediation effect (ACME) via SES was negative and not statistically significant (ACME = −0.41, 95 % CI [−0.80, 0.01], $p = .053$), while the average direct effect (ADE) of group remained significant (ADE = 0.89, 95 % CI [0.24, 1.46], $p = .010$). The total effect was not statistically significant (Total effect = 0.47, 95 % CI [−0.01, 0.99], $p = .053$). In the corresponding model with right ILF MD, the ACME via SES was positive and not significant (ACME = 0.48, 95 % CI [−0.06, 0.98], $p = .081$), whereas the ADE of group was significant and negative (ADE = −0.93, 95 % CI [−1.65, −0.07], $p = .030$). The total effect was not statistically significant (Total effect = −0.44, 95 % CI [−1.02, 0.12], $p = .124$). Taken together, these exploratory mediation analyses indicate that group effect exerts an effect on right ILF FA and MD that is not fully accounted for by SES. While SES is associated with both group effect and right ILF microstructure and thus may act as a confounding factor, the indirect effects via SES were not statistically significant and the direct group effect remained present. Further details of the mediation models, including graphical displays of the indirect and direct pathways, are provided in the supplements section mediation analysis.

In sum, there seems to be a difference in FA (at-risk > control) and MD (at-risk < control) between the groups in the right ILF, although these findings should be interpreted with caution considering SES confounding and the exploratory nature of the mediation analyses.

3.2. Correlation between parenting behaviour and WM pathways (H2)

In the full model a negative correlation was found between dysfunctional parenting total score and FA of the CC genu (Table 3). In the model without SES, dysfunctional parenting total scores were likewise associated with a broader pattern of reduced white matter microstructure. Specifically, significant correlations with lower FA and high MD were observed in the CC genu, bilateral dorsal cingulum, bilateral UF, bilateral ILF, right SFL3 and right SLF2. Moreover, dysfunctional parenting was related to increased MD in the right ventral cingulum right SLF1 and left SLF3.

One participant in the control group appeared to be an overly influential observation, as it fell outside the Cook's distance threshold ($D > 0.5$) in approximately half of the residuals vs. leverage plots, with an externally studentized residual greater than 4 and a leverage (hat) value above 0.20 in those models. This pattern can be interpreted as a very large residual combined with high influence on the regression estimates, justifying recomputing the regression models without this participant. In the full model but excluding this outlier, dysfunctional parenting total scores were associated with a more widespread pattern of reduced white matter microstructure. Specifically, significant negative correlations with FA and positive correlations with MD emerged not only in the CC genu, but also bilaterally in the dorsal cingulum, ILF and SLF3 as well as in FA and MD of the right SLF2 and left UF. Additionally, MD was associated with the left and right cingulum ventral, left and right SLF1, left SLF2 and right UF. Finally, as expected, an even broader pattern emerged in the model without SES and excluding the outlier, where all correlations between dysfunctional parenting and the examined tracts reached statistical significance.

In sum, across the SES adjusted and SES unadjusted models excluding the influential observation, the most consistent associations between dysfunctional parenting total score and white matter microstructure emerged in the CC genu, bilateral dorsal cingulum, bilateral ILF, bilateral SLF3, right SLF2 and left UF, all of which showed large

effect sizes. A comparison between these models shows that while SES acts as a confounding factor that partially attenuates the strength of the associations, the key findings remain robust. The persistence of these effects after accounting for SES indicates that the observed relationships cannot be explained by socioeconomic status alone, suggesting a unique contribution of parenting quality to children's white matter development.

3.3. Correlation between parenting stress and WM pathways (H3)

Taking a look at the four dimensions of the parental stress questionnaire, i.e., parental stress, role restriction, social support and partnership support, only the partnership support dimension showed associations with WM tracts. In the full model, higher partnership support was related to both higher FA and lower MD of the CC genu (FA / MD: $r_p(39) = 0.55$ / -0.54, both $p = .001$). Additionally, partnership support was related to higher FA in the left dorsal cingulum (rp(39) = 0.54, $p < .001$), the right SLF1 ($r_p(39) = 0.51$, $p = .001$) and the left ILF ($r_p(39) = 0.53$, $p = .001$). Moreover, it was associated with lower MD in the right UF ($r_p(39) = -0.58$, $p < .001$).

In the model without SES, the pattern of associations between partnership support and white matter microstructure became even more pronounced. Higher partnership support was again related to both higher FA and lower MD of the CC genu (FA/ MD: $r_p(40) = 0.63$ / -0.63, both $p < .001$) and with the bilateral dorsal cingulum (left FA / MD: $r_p(40) = 0.61$ / -0.55, both $p < .001$; right FA / MD: $r_p(40) = 0.57$ / -0.60, both $p < .001$), bilateral UF (left FA / MD: $r_p(40) = 0.57$ / -0.62, both $p < .001$; right FA / MD: $r_p(40) = 0.54$ / -0.68, $p < .001$ / $p = .001$) and bilateral ILF (left FA / MD: $r_p(40) = 0.60$ / -0.55, both $p < .001$; right FA / MD: $r_p(40) = 0.55$ / -0.61, both $p < .001$). Additionally, partnership support was related to higher FA and lower MD in the right SLF1 (FA / MD: $r_p(40) = 0.60$ / -0.58, both $p < .001$). Moreover, partnership support was associated with higher FA in the left ventral cingulum (FA: $r_p(40) = 0.50$, $p < .001$), left SLF1 (FA: $r_p(40) = 0.53$, $p < .001$), as well as with lower MD in the left and right SLF3 (left MD: $r_p(40) = -0.54$, $p < .001$; right MD: $r_p(40) = -0.60$, $p < .001$), and right SLF2 (right MD: $r_p(40) = -0.60$, $p < .001$).

Overall, partnership support showed the most robust association with white matter tracts in the CC genu, where higher partnership support was consistently related to higher FA and lower MD in models with and without SES. Beyond this finding, the full model including SES also revealed positive associations with FA in the left dorsal cingulum, right SLF1 and left ILF, and a negative association with MD in the right UF. When SES was removed from the model, these effects strengthened and extended such that both FA and MD of the left dorsal cingulum, right SLF1, left ILF and right UF were significantly associated with partnership support, consistent with SES attenuating but not explaining these associations.

3.4. Correlation between emotion regulation and WM pathways (H4)

Regarding the correlations between adaptive and maladaptive ER strategies of the participants themselves (self and parent-report) and WM pathways, no correlation survived the M_{eff} correction. This does not change when SES is removed from the model.

4. Discussion

This study examined differences in microstructural properties of WM pathways related to ER (SLF, ILF, UF, cingulum, and CC genu) between healthy children of parents with mental illness (at-risk group) and healthy children of parents without mental illness (control group) with strict inclusion and exclusion criteria (H1). Additionally, the study explored how (dys-)functional parenting behaviours (H2), parental stress (H3) and ER of the children (self and parent-report; H4) are associated with the microstructure of these WM pathways.

4.1. WM alterations in the at-risk group

Comparing the two groups, higher FA and lower MD in at- risk group, was found in the right ILF. The ILF is the major occipital-temporal association WM pathway, connecting the occipital extrastriate cortex with the temporal lobe and vice versa. As mentioned above, ILF alterations were found in various mental illnesses (e.g. Gorman et al., 2021; Tükel et al., 2017; Versace et al., 2018). Specifically, higher FA values in the right ILF of at-risk individuals compared to controls have been reported in previous studies (Versace et al., 2010, 2018) and predominantly right-sided tracts (e.g. ILF, UF; Versace et al., 2018). Crucially, those differences did not survive M_{eff} correction and exhibited smaller effect sizes when SES was not controlled for. However, exploratory mediation analysis revealed a direct effect of group on ILF FA/MD and no statistically significant mediation effect. These findings suggest that including SES as a covariate improves the precision of the group estimate by accounting for shared variance unspecific to group differences and thereby revealing the group effect. Further, multicollinearity was negligible as indicated by VIFs. However, given that SES differs significantly between groups, with at-risk children more likely to grow up in socioeconomically weaker environments, the specific interaction between SES and parental mental illness remains unclear. To disentangle these contributes to WM differences, longitudinal designs are needed, and current results must be replicated and interpreted with caution. After all, previous research has consistently shown a bidirectional interaction between SES and the mental disorders: Individuals with lower SES are at a higher risk of mental illness, possibly because they experience more stressful events in their lifetime and have less access to support and resources (Oliver-Parra et al., 2020; World Health Organization, 2014), while individuals with a mental disorder suffer from a higher risk of lower educational performance, job loss and lower income (e.g. Goodman et al., 2011; Miech et al., 1999; Whooley et al., 2002).

Interpreting our finding of higher FA and lower MD in the right ILF in currently healthy at-risk children despite the unclear SES-involvement raises a critical question: Do these alterations reflect a protective coping mechanism or an early sign of maldevelopment? On the one hand, the observed pattern may reflect a compensatory coping mechanism in response to environmental challenges (e.g. Baum et al., 2020; Deoni et al., 2016). As the ILF is important for integrating visual and emotional processes (e.g. Catani and Dawson, 2017), particularly recognizing facial emotions (Bauer, 1982; Fischer et al., 2016; Herbet et al., 2018), alterations could represent a neural adaption that allows children to more effectively monitor their parents' (or other caregivers') emotional states. On the other hand, this pattern could signify maldevelopment: a study on healthy and neglectful mothers indicates that alterations in the right ILF (reduced volume) predicts lower emotional availability for their children, suggesting that structural disruptions from early adversity impair the ability to appropriately "read" and respond to social-emotional signals (Rodrigo et al., 2016). Though this study was conducted in an adult sample, this converges with our cautious interpretation that ILF alterations could similarly indicate disrupted socio-emotional processing. Furthermore, deviations of ILF from typical maturation trajectories, such as the failure to show healthy age-related increases in WM pathways (Reynolds et al., 2019), were shown to point to a more general breakdown in coordinated neural development that limits long-term socio-emotional and cognitive adaptability (e.g. Rollins et al., 2009; Yeatman et al., 2012). As our sample included children aged 7 to 16 years, such a neurodevelopmental perspective is particularly relevant, because (pre-)adolescence represents a sensitive phase in WM development. During this period, WM is characterised by ongoing developmental changes and increased plasticity, which implies increased vulnerability to both positive and negative environmental influences (e.g. Barnea-Goraly et al., 2005; Fuhrmann et al., 2015; Kolb and Gibb, 2011; Muftuler et al., 2012; Schmithorst and Yuan, 2010). Importantly, we must acknowledge the interpretive boundaries of DTI metrics. FA and MD are summary indices

of microstructural properties, such as fibre density, coherence and myelination and cannot be uniquely attributed to any single biological feature. Therefore, while these findings are sensitive indicators of altered white matter microstructure in at-risk children, they should be viewed as evidence of altered developmental trajectories rather than direct measure of tissue quality.

4.2. Relation of WM with parental behaviour and children's emotion regulation

Strong negative correlations were observed between dysfunctional parenting and FA in the CC genu, and - after removal of an overly influential individual data point - between dysfunctional parenting and both lower FA and higher MD in the CC genu, bilateral dorsal cingulum, bilateral ILF, bilateral SLF3, right SLF2 and left UF, and in models with and without SES as covariate. As mentioned, the total score of the dysfunctional parental behaviour questionnaire comprises three subdimensions: overreaction (irritable, loud or angry reactions), laxness (compliance and difficulties in consistently enforcing rules) and verbosity (excessive explaining, negotiating or arguing with the child). Taken together, a high total score reflects parents who react emotionally charged, inconsistently, and with little focus in challenging situations. Their behaviour resembles patterns commonly associated with authoritarian or permissive parenting styles. Therefore, our results are in line with findings showing that extreme forms of parental dysfunctional behaviour are associated with childhood adversity (e.g. emotional abuse, neglect or exploitation) and alterations (e.g. reduced FA) in various white matter tracts including especially the CC (genu) as well as SLF, UF, ILF and cingulum (for a meta-analysis Lim et al., 2020; Lugo-Candelas et al., 2023; McCarthy-Jones et al., 2018). Less is known about the effects of more common – but still dysfunctional – parental behaviour; furthermore, most studies focused on mother-child dyads. A study investigating more typical dysfunctional parenting showed a negative relation of harsh maternal parenting with WM volume (Hidalgo et al., 2022). Further, studies show an association between unpredictable patterns of maternal care and imbalanced maturation of the UF and cingulum in childhood (Granger et al., 2021) and a positive relationship between maternal affective parenting (at 3 years) and secure mother-child attachment (at 1 years) with an increase of FA in the CC at age six (Kim et al., 2020). Therefore, our results extend previous finding by examining more normative dysfunctional parenting practices (rather than extreme adversity), showing associations between dysfunctional parenting behaviour and altered WM microstructure of emotion-related WM pathways in late childhood and adolescence.

Among the four parental stress dimensions, only partnership support showed associations with WM microstructure, whereas perceived parenting stress, role restriction, and social support did not. Lower scores on partnership support, reflecting greater dissatisfaction and difficulties in the parental relationship and less agreement on educational issues, were associated with FA and MD in the CC genu, as well as with FA in the left dorsal cingulum, right SLF1, left ILF and with MD in the right UF, with these associations becoming even more widespread across FA and MD when SES was not included as a covariate. Against the backdrop of a recent large-scale longitudinal study showing that associations between environmental risk factors, mental health, and brain structure are typically small – particularly in late childhood and adolescence, where developmental trajectories become more heterogeneous (e.g. Dall'aglio et al., 2023) – it seems plausible that only the stress measure closest to the child's daily family atmosphere – partnership support – shows clear links to diffusion metrics in our modestly sized sample, while broader stress scales do not. Prior research on parental stress and WM has predominantly focused on neonates and the perinatal period (e.g. Karlsson et al., 2020; Lautarescu et al., 2020; Lean et al., 2022), when children are most directly affected by maternal conditions. In contrast, the present sample comprised pre-adolescent children, and parental stress was expected to influence WM more indirectly, for example via its effects on children's ER or chronic stress exposure. However, child stress, peer support, and other buffering factors were not directly assessed, and the parental stress dimensions considered here may be generally more influential earlier in development. The link between partnership support and WM microstructure is consistent with the concept of expressed emotion, which states that an unfavourable family climate can adversely affect youth mental health (for a review see Peris and Miklowitz, 2015) and may, via experience-dependent plasticity of white matter, contribute to alterations in developing tracts (e.g. Benjamin et al., 2024; Binter et al., 2024; Carozza et al., 2025; Lebel and Deoni, 2018; Roy et al., 2024). Since various mental disorders have high affective components (APA, 2013), it is notable that partnership support was related to diffusion metrics in several emotion-related pathways, in line with recent longitudinal evidence linking early family functioning, but not mid-childhood functioning, to pre-adolescent white matter microstructure in large, socioeconomically diverse sample (global FA and global MD; Delaney et al., 2023).

Surprisingly, no correlation survived the multiple comparison correction between questionnaire-based ER strategies and WM microstructure in either self- or parent-reports. A closer look at the literature reveals that findings in this area seem to be highly dependent on the assessment method and the specific operationalization of the ER construct. Several studies employing experimental ER tasks, which typically operationalize isolated strategies or specific sub-components of ER (e.g. Coad et al., 2020; Oishi et al., 2015; Unger et al., 2016), have reported associations with WM microstructure. Questionnaire studies that found significant links often utilized the ERQ (e.g. Vitolo et al., 2022; Zuurbier et al., 2013), which assesses the habitual use of two specific strategies, reappraisal and suppression. However, even with the ERQ, results remain mixed (e.g. Seyedmirzaei et al., 2022). Our lack of findings with the FEEL-KJ may partly reflect the level of psychometric aggregation applied in our analyses. We used broad composite dimensions (e.g. maladaptive vs. adaptive strategies) that bundle a variety of behaviours into a small number of scores. Different ER strategies are supported by largely overlapping, yet partly distinct patterns of activation within fronto-limbic control networks (e.g. Goldin et al., 2008), and aggregating across such strategies may therefore mask or dilute strategy-specific associations with WM pathways. Collapsing heterogeneous behaviours into only two composite indices may reduce sensitivity to detect microstructural correlates. Finally, the FEEL-KJ questionnaire operationalizes ER primarily as the frequency of habitual strategy use (item examples: "When I am angry, I withdraw", "When I am afraid, I think it will pass"), whereas WM microstructure may relate more closely to the efficiency, flexibility, or context-sensitivity of emotional processing rather than mere frequency, as proposed in broader constructs such as ER flexibility or emotional competence (Bonanno and Burton, 2013; Nizielski and Rindermann, 2016; Rindermann, 2009). This is compounded by the fact that the correspondence between biological markers and emotion regulation is often characterized by small effect sizes and heterogeneity, as indicated by meta-analytic evidence on psychophysiological responses (e.g. Zaehringer et al., 2020), suggesting that our sample might have been underpowered to detect such subtle effects.

In summary, the observed correlations provide evidence that parental behaviour is linked to the development of white matter in children. Our findings extend previous research which found correlations between extreme dysfunctional behaviour and WM, showing that more common dysfunctional behaviours are also related to WM, even in late childhood and adolescence. Family environment (e.g. setting shared boundaries) also seems to be an important but under-researched variable in pre-adolescence (e.g., Delaney et al., 2023). Specifically, more directly investigating the quality of parental behaviour and family environment might help differentiate dysfunctional from compensatory neural alterations in children of parents with a mental illness. The WM microstructural difference in the right ILF between the groups, which is

confounded by SES (though a direct effect is suggested by exploratory mediation), further highlights the importance of assessing the broader family environment. This aligns with evidence that the ILF's maturation plateau occurs earlier than that of the SLF and cingulum, potentially making it more sensitive to stable, early environmental influences - such as chronic parental stress - while the prolonged development of the SLF and cingulum may obscure group differences in favour of continuous, subtle associations as found in our correlation analyses (Vanes et al., 2020).

4.3. Limitation and future directions

Despite its insights, several limitations warrant consideration. First, the cross-sectional design precludes causal conclusions and makes it difficult to disentangle genetic and environmental effects. We lacked detailed information on the duration and timing of parental symptoms, which are critical for identifying sensitive developmental windows. While we accounted for SES, unmeasured factors such as peer support or extracurricular activities might have served as protective buffers.

Second, the sample includes siblings, which share genetic and environmental background. As emphasized by recent research, mental illnesses and neurodevelopment are shaped by a complex interplay of genetic risk, environmental adversity, and biological processes such as inflammation (e.g. Cao et al., 2024; Cao et al., 2025, 2025; Sforzini et al., 2025). However, from a family-systems perspective, the similarity between siblings is not merely a nuisance variable, it is a theoretically meaningful reflection of the shared family environment we aimed to study.

Third, with a sample size of $N = 64$ and $N = 47$ for correlation analyses, the study is underpowered to detect small effects, and the correlations models are only on the borderline for effects in medium range. Further, the at-risk group ($N = 28$) was not large enough to perform a stratified analysis by specific parental diagnosis. Subdividing this cohort into several clinical groups would have compromised statistical power and reliability of the estimates, though future studies would ideally include comparisons of trans-diagnostic similarities and differences of mental disorders.

Fourth, the SES in both groups is relatively high, as reflected by a minimum SES score of 15 (correlation hypothesis around 18) out of 21, placing them in the upper 40 % (to 20 %) of the general population according to national norms (Lampert et al., 2018). This suggests that the participating families were particularly proactive, with parents who were both well-educated and may have sought help early on in the course of their difficulties. Further, all participating families had voluntarily engaged with an outpatient clinic, indicating a level of insight into their mental health challenges and a willingness to access care that might not be representative of the population.

Fifth, to characterize white matter microstructure, we primarily rely on the combination of FA and MD (axial and radial diffusivity are provided as explorative measures in the supplement). As mentioned above, we restrain of interpreting FA and MD as direct indicators of WM integrity, as FA and other standard metrics are highly sensitive to complex WM architecture like fibre crossings, present in up to 90 % of WM voxels (Jeurissen et al., 2013). In such areas, increased FA can either indicate high integrity of the primary tract or paradoxically reflect a reduction in fibre populations of a second crossing tract (Figley et al., 2022). However, the consistent increase in FA and reductions in MD observed across our identified tracts point to a possible increase in WM integrity related to parental mental illness, as MD is more robust to crossing-fibre confounds (Figley et al., 2022). Future screenings of at-risk populations could additionally benefit from emerging non-invasive approaches, which has recently shown promise in predicting WM pathology and brain health (Tong et al., 2025; Wu et al., 2025). Validating these cross-modal markers in developmental cohorts could offer a scalable, cost-effective alternative to MRI for identifying vulnerable children.

Finally, diffusion imaging was limited to 33 directions to keep the acquisition time under eight minutes and the overall scan time around half an hour as well as the sequence maintain comparability with previous studies in the same scanner system (Meissner et al., 2021). This deliberate compromise minimized head movement and ensured good data quality (e.g. Meissner et al., 2020), while at the same time reducing the overall burden on the children who voluntarily participated in the extensive COMPARE project (Christiansen et al., 2019) and the MRI measurements. Unfortunately, the reduced number of directions limited the use of more advanced modelling techniques for white matter.

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Data and code availability

The script containing computations, plots, verification of the assumptions of the statistical tests etc. can be found at github [1], the analysed data can be found on zenodo https://doi.org/10.5281/zenodo.17084310.

[1] https://github.com/TSchweikert/Paper-TransDTI.

CRediT authorship contribution statement

Timo Schweikert: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Klara Hagelweide:** Writing – review & editing, Methodology, Investigation, Data curation, Conceptualization. **Lena-Mareike Krüger:** Investigation, Data curation, Conceptualization. **Naomi Leona Werkmann:** Writing – review & editing, Investigation, Data curation, Conceptualization. **Vanessa Seipp:** Investigation, Data curation, Conceptualization. **Arleta Luczejko:** Investigation, Data curation, Conceptualization. **Sarah Weigelt:** Writing – review & editing, Resources, Project administration, Funding acquisition, Conceptualization. **Hanna Christiansen:** Resources, Project administration, Funding acquisition, Conceptualization. **Christina Schwenck:** Writing – review & editing, Resources, Project administration, Funding acquisition, Conceptualization. **Meinhard Kieser:** Resources, Project administration, Funding acquisition, Conceptualization. **Kathleen Otto:** Resources, Project administration, Funding acquisition, Conceptualization. **Corinna Reck:** Resources, Project administration, Funding acquisition, Conceptualization. **Ricarda Steinmayr:** Resources, Project administration, Funding acquisition, Conceptualization. **Linda Wirthwein:** Resources, Project administration, Funding acquisition, Conceptualization. **Anna-Lena Zietlow:** Resources, Project administration, Funding acquisition, Conceptualization. **Rudolf Stark:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.neuroimage.2026.121849.

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