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Maternal posttraumatic stress and *FKBP5 Genotype* interact to predict trauma-related symptoms in preschool-age offspring

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Abstract

Background: Children of parents with posttraumatic stress (PTS) face heightened risk for developing emotional and behavioral problems, regardless of whether they experience a traumatic event themselves. The current study investigates whether child *FKBP5*, a stress relevant gene shown to interact with child trauma exposure to increase risk for PTS, also moderates the well-established link between maternal PTS and child symptoms.

Methods: Data are derived from a longitudinal lab-based study for which 205 dyads of trauma-exposed mothers and their preschool-age children from a sample enriched for violence exposure provided DNA samples and completed measures of maternal and child trauma-related symptoms. Hypotheses tested whether child *FKBP5* rs1360780 SNP genotype interacts with child trauma exposure and maternal PTS to predict child trauma-related symptoms.

Results: Hypotheses were partially supported, with maternal PTS predicting increased child symptoms for children carrying the minor T-allele (CT/TT), but not those homozygous for the major C-allele.

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Contributors

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Conflicts of interest

We have no conflicts of interest to disclose.

Supplementary materials

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Limitations: Study results may not generalize to lower-risk or non-clinical populations, did not assess between-group differences in race/ethnicity, and do not consider other genes that may interact with FKBP5 or contribute to genetic risk for trauma-related impairment.

Conclusions: These findings provide the first evidence that the robust gene x environment interaction involving *FKBP5* and child trauma exposure extends to other environmental perturbations, including maternal PTS. Our results highlight the importance of efforts to address trauma-related psychopathology in caregivers, which may disrupt intergenerational risk processes and improve outcomes for children.

Keywords

FKBP5; Intergenerational trauma; Preschoolers; Posttraumatic stress; Dissociation

Children of parents with posttraumatic stress (PTS) face heightened risk for developing emotional and behavioral problems, regardless of whether they experience a traumatic event themselves (for review see Lambert et al., 2014). Elucidating mechanisms that drive this intergenerational transmission of trauma-related risk is essential for early prevention and intervention. A substantial literature implicates the interaction of genes and the caregiving environment in risk transmission, with emphasis on genes involved in the hypothalamic-pituitary-adrenal (HPA) axis, a neuroendocrine system that regulates the nervous system in response to stress.

One such gene, *FKBP5*, encodes the glucocorticoid receptor (GR) co-chaperone protein (FKBP51), which is involved in an intracellular negative feedback loop responsible for effective recovery from the stress response. Under stress, glucocorticoids (GCs) released in the blood-stream activate GRs, which translocate to the nucleus to enhance or repress gene transcription. GCs mediate the feedback loop by activating GRs within the hypothalamus and pituitary to reduce secretion of corticosteroid releasing hormone and adrenocorticotropic hormone. FKBP51 interrupts the feedback loop by binding to GRs, reducing affinity to cortisol and impeding GR translocation to the nucleus. Evidence suggests that this process is partially moderated by genetic variation at the *FKBP5* gene, with the SNP rs1360780 being functional (Klengel et al., 2013; Sheerin et al., 2020). The minor allele of this variant (T) is associated with greater relative induction of *FKBP5* following GR activation, which suggests a compromised feedback loop and sustained stress response.

As reviewed in recent meta-analyses, several studies have now reported greater PTS in TT/CT carriers with a history of childhood trauma exposure, relative to trauma-exposed adults homozygous for the major allele (CC) (Hawn et al., 2019; Wang et al., 2018; Xie et al., 2010). This effect appears to be specific to childhood trauma, suggesting the presence of a sensitive period when trauma exposure may be more potent. However, few studies have examined these effects in children. One study reported increased cortisol reactivity during the Strange Situation in 14-month-old infant CT/TT carriers, relative to CC carriers (Mulder et al., 2017). In addition, few studies have investigated intergenerational effects associated with these processes, with a series of studies demonstrating allele-specific epigenetic effects associated with adult offspring of Holocaust survivors (Bierer et al., 2020; Yehuda et al.,

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2016), and another by our group showing allele-specific epigenetic differences in newborn offspring of mothers with and without prenatal PTS (Grasso et al., 2020).

To date, no studies have reported the potential interaction of maternal PTS and child *FKBP5* genotype on symptoms in young children. Given the robust association between maternal PTS and child symptoms, the question of whether *FKBP5* moderates risk, perhaps by modulating how children regulate increased stress in the caregiving environment, is a critical next step. To this end, the current study tested hypotheses that both child trauma exposure and maternal PTS would interact with *FKBP5* genotype to predict increased trauma-related symptoms in CT/TT carriers (rs1360780) relative to CC carriers.

1. Method

1.1. Procedures and sample

The current study leverages a longitudinal lab-based study of preschool-age children that has been previously described in detail (Nichols et al., 2015). This lab-based cohort was oversampled for exposure to intimate partner violence (IPV) and elevated child disruptive behavior as per the original study aims. Participating in laboratory visits were 425 dyads at the preschool wave, with 381 consenting to child DNA collection. Eligibility for the current analysis required that mothers have a history of trauma exposure (N = 242), child *FKBP5* genotype data were obtained (N = 212), and measures of maternal and child symptoms and child trauma history were obtained (N = 211). All criteria were met for 205 dyads. Maternal trauma exposure was positive if a mother reported childhood maltreatment on the Childhood Trauma Questionnaire (Bernstein et al., 1994) or physical IPV on the Family Socialization Interview-Revised (see Measures, for further details see Greene et al., 2020). The 205 eligible mother-child dyads were comparable to the remainder of the sample (N = 220) in terms of mother's age and child's age, gender, and ethnicity, and child trauma symptoms, ps > .10. As expected given their trauma histories, mothers were higher in PTS symptoms (p <.01). The sample was evenly distributed by child sex (49% boys) and diverse in child's ethnicity (49% African American, 33% Hispanic/Latinx, 18% European American, 1% other) and poverty (54% living in poverty). Mean child age at the time of the symptom assessment was 4.64 years (SD = 0.84; range = 3.1–7.2).

Participants attended laboratory visits that included maternal interviews and questionnaires concerning children's mental health, family environment, and childrearing practices, and collection of buccal swabs and/or saliva samples for genetic analysis. Mothers again completed questionnaires 6–9 months later. Mothers were compensated for participation and transportation, and provided informed consent. Study protocols were approved by University institutional review boards. Mandated child abuse and neglect reporting procedures were followed.

SNP rs1360780 was genotyped in using Taqman Assay on Demand (C___8852038_10) (Applied Biosystems, Foster City, USA), according to the manufacturer's protocol and genotype determined at end-point using an ABI 7900HT Sequence Detection System and SDS 2.3 software. Genotypes were in Hardy-Weinberg equilibrium (p=.83) [n (%): CC = 82 (40), CT = 100 (49), TT = 23 (11)].

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1.2. Measures

1.2.1. Demographics—Mothers provided basic demographic information about maternal and child age, poverty status, racial/ethnic backgrounds, and sex assigned at birth.

1.2.2. Child trauma exposure—Baseline trauma exposure was assessed by maternal interview. Children were considered positive for lifetime trauma exposure if they were the victim or witness of a posttraumatic stress disorder (PTSD) qualifying violent event reported on the Preschool-Age Psychiatric Assessment (PAPA) (Egger & Angold, 2004) or if had experienced "probable" or "definite" physical abuse by a parent figure or witnessed physical IPV according to the Family Socialization Interview-Revised (FSI-R), semi-structured, independently-coded interview (Dodge et al., 1994; O'Dor et al., 2017) (*Inter-rater reliability weighted Kappas*: 0.80 to 1.0).

1.2.3. Child trauma-related symptoms—Mothers completed the Trauma Symptom Checklist for Young Children (TSCYC) (Briere et al., 2001), a norm-referenced measure, appropriate for normative and clinical populations, that does not require a criterion traumatic event. Items assess symptoms of arousal, intrusion/re-experiencing, avoidance/numbing, and dissociation and are rated on a 5-point scale ranging from *Not true at all* to *Very often true*. Analyses used second wave data and utilized norm-referenced *T*-scores for the Total Trauma Symptom score, comprised of the arousal, intrusion/re-experiencing, and avoidance subscales, and Dissociation subscale (α = 0.83, 0.89, 0.89, and 0.90, respectively).

1.2.4. Maternal PTS—At baseline, mothers rated how much they were bothered by symptoms related to their traumatic experiences in the past month on the 17-item PTSD Checklist–Civilian version (PCL-C) (Weathers et al., 1993). The PCL-C is based closely on PTSD criteria from the DSM-IV (American Psychiatric Association, 2000). Items are rated on a 5-point scale from *Not at all* to *Extremely*. Total PTSD symptoms was calculated as the sum of all items (α= 0.93).

1.3. Statistical analyses

Data were analyzed using IBM SPSS (Version 25) statistical software. In line with previous research, children were classified as *FKBP5* CT/TT carriers (N = 123) or CC carriers (N = 82). Pearson's chi-square and independent-samples t-tests compared outcomes on demographic and clinical characteristics. Spearman correlations were used to examine bivariate associations among study variables. Hierarchical multiple linear regression analyses were applied to test main effects of predictor variables on child trauma symptoms, as well as genotype interactions on child trauma symptoms. Each model was built in the following blocks: 1 – demographic variables, 2 - *FKBP5*, 3 – child trauma exposure, 4 – maternal PTS. The final block tested one interaction (e.g., *FKBP5* X child trauma exposure or *FKBP5* X maternal PTS). Interaction variables were mean centered. Study analyses utilized bootstrapping (5000 samples) and results were considered significant at the $p < .05$ threshold.

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2. Results

Thirty-six percent of children had a positive trauma history. CT/TT and CC carriers were similar on demographic characteristics and trauma exposure (see Supplemental Table A1). Child trauma exposure, trauma symptoms, and dissociative symptoms correlated positively with maternal PTS (see Supplemental Table A2). Table 1 presents regression analyses. Controlling for demographic characteristics, maternal PTS was significantly predictive of child trauma and dissociative symptoms. *FKBP5* genotype did not interact with child trauma exposure to significantly predict child symptoms; however, controlling for child trauma history, genotype significantly interacted with maternal PTS to predict child trauma and dissociative symptoms. Specifically, maternal PTS was significantly predictive of child trauma and dissociative symptoms (see Fig. 1) for CT/TT carriers, but not CC carriers (Trauma: CT/TT, $b = 0.37$, $SE = 0.07$, $p < .001$; CC, $b = 0.11$, $SE = 0.10$, $p = .28$; Dissociation: CT/TT, $b = 0.38$, $SE = 0.06$, $p < .001$; CC, $b = 0.07$, $SE = 0.08$, $p = .37$).

3. Discussion

The current study presents novel findings that maternal PTS and child *FKBP5* genotype interact to predict child trauma and dissociative symptoms in preschool-age children, over and above child trauma exposure. This builds from a large literature demonstrating linkages between parental PTS and adverse mental health outcomes in children, regardless of child trauma exposure (Lambert et al., 2014), to implicate stress-related genetic vulnerability. *FKBP5* is a well-studied gene that is functionally tied to the intracellular negative feedback loop, which serves to regulate the stress response system in the context of stress. Numerous studies and several meta-analyses to date have reported allele-specific risk for PTS in adults exposed to childhood trauma. Our study may be the first to extend this effect to *maternal* PTS. Our data suggest that young children who are genetically predisposed to a sustained stress response (i.e., carrying the *FKBP5* risk allele) are more vulnerable to the adverse effects of maternal PTS. This vulnerability may be pronounced in early childhood because acquiring emotion regulation is a key developmental task of this period and parents play an important role in modeling and shaping children's emotion regulation. An early pairing of genetic and caregiving risks may place children on a lifelong path of enhanced vulnerability to stress-related psychopathology.

The interaction of maternal PTS and child *FKBP5* may serve as one mechanism by which trauma-related risk is transmitted across generations. Our findings highlight the importance of efforts to address trauma-related psychopathology (e.g. PTSD) in caregivers, which in turn may disrupt intergenerational risk processes and improve trauma-related symptom outcomes for children.

Contrary to studies of adult samples (Hawn et al., 2019; Klengel et al., 2013; Wang et al., 2018; Xie et al., 2010), we did not find a significant interaction between *FKBP5* genotype and child trauma exposure. One possible explanation is that while about a third of the preschool sample had a history of exposure to a PTSD-qualifying traumatic event, the overall sample was high risk, as all mothers had a history of trauma. Studies of *FKBP5* in adults have, in some cases, included adverse childhood experiences that do not qualify

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as PTSD-qualifying events (e.g., Wang et al., 2018). Thus, we may not have had sufficient variability to detect an *FKBP5* X trauma exposure effect in this sample.

Study findings should be interpreted in the context of limitations. Due to this being a clinically-enriched sample and requiring maternal trauma exposure, results may not generalize to lower-risk or non-clinical populations. Additionally, although a majority of participants were from racially marginalized populations, our study was not able to assess between-group differences, which have previously been reported in *FKBP5* research (Xie et al., 2010). Finally, our study does not take into consideration other genes that may interact with *FKBP5* or contribute to genetic risk for trauma-related impairment. This is a direction for future research. Despite these limitations, the current study elucidates allele-related risk as a potential mechanistic pathway between *FKBP5* and maternal PTS and child traumatic symptoms, laying the foundation for targeted prevention for CT/TT carrying children at highest biologic risk for adverse intergenerational transmission of trauma.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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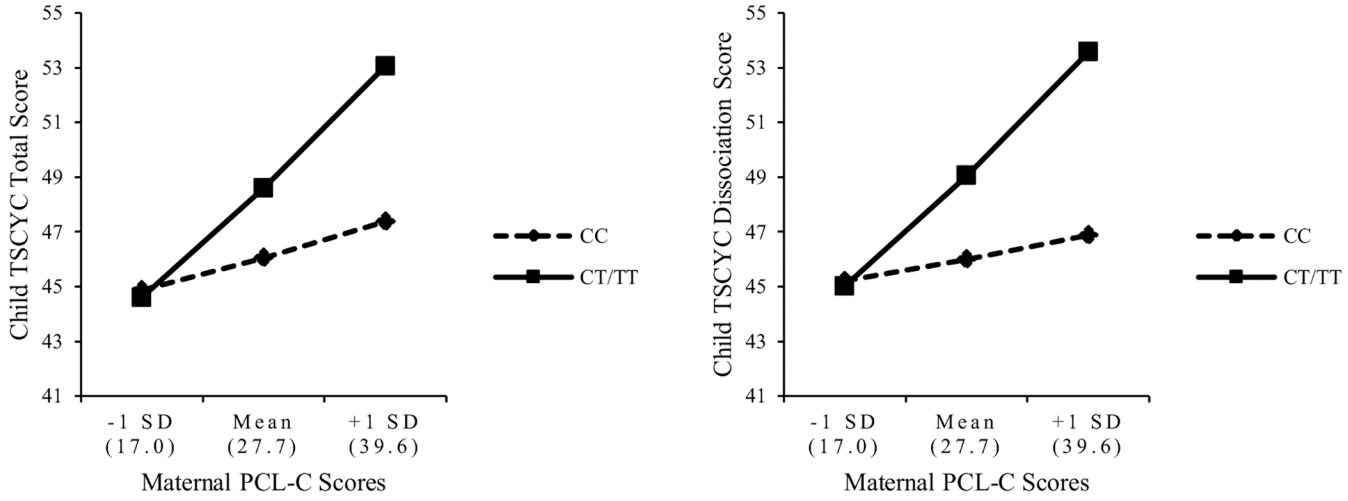

Fig. 1.

Interactions between FKBP5 and maternal PTSS on child trauma symptoms. *Note*. The interaction predicting child total trauma symptoms is displayed on the left while child dissociation symptoms is displayed on the right. PCL-C scores are displayed one standard deviation below and above the total sample mean. PTSS = Posttraumatic stress symptoms; TSCYC = Trauma Symptom Checklist for Young Children; PCL-C = Posttraumatic Stress Disorder Checklist–Civilian version.

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Table 1

Hierarchical regressions and interactions predicting child total trauma and dissociation symptoms.

Variable	Total trauma symptoms					Dissociation symptoms				
	F	r^2	Δr^2	β	SE	F	r^2	Δr^2	β	SE
Block 1	3.13	.03*	.03*			3.19	.03*	.03*		
Age				− 0.13*	0.70				− 0.04	0.60
Sex[a]				0.02	1.20				0.11*	1.03
Race/ethnicity[b]				− 0.10	0.31				− 0.12*	0.26
Block 2	2.82	.03*	.01			3.35	.04*	.01		
Age				− 0.13*	0.70				− 0.04	0.60
Sex[a]				0.02	1.20				0.11	1.03
Race/ethnicity[b]				− 0.09	0.31				− 0.11*	0.26
FKBP5[c]				0.08	1.22				0.11	1.05
Block 3	2.85	.04*	.01			2.98	.04*	.00		
Age				− 0.14*	0.70				− 0.04	0.60
Sex[a]				0.01	1.20				0.10	1.03
Race/ethnicity[b]				− 0.07	0.31				− 0.10	0.27
FKBP5[c]				0.07	1.22				0.10	1.05
Trauma exposure[d]				0.09	1.32				0.07	1.13
Block 4	9.82	.15***	.11***			10.78	.16***	.12***		
Age				− 0.13*	0.66				− 0.03	0.56
Sex[a]				0.00	1.13				0.09	0.96
Race/ethnicity[b]				− 0.08	0.29				− 0.10	0.25
FKBP5[c]				0.06	1.15				0.10	0.98
Trauma exposure[d]				0.00	1.28				− 0.03	1.10
Maternal PTS				0.34***	0.05				0.36***	0.05

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Variable	Total trauma symptoms					Dissociation symptoms				
	F	r^2	Δr^2	β	SE	F	r^2	Δr^2	β	SE
Block 5a	8.57	.15***	.00			9.32	.17***	.00		
Age				− 0.13*	0.66				− 0.03	0.56
Sex[a]				0.00	1.13				0.09	0.96
Race/ethnicity[b]				− 0.07	0.29				− 0.10	0.25
FKBP5[c]				0.03	1.36				0.07	1.17
Trauma exposure[d]				− 0.06	2.03				− 0.08	1.72
Maternal PTS				0.34***	0.05				0.36***	0.05
FKBP5 x trauma exposure				0.09	2.52				0.07	2.15
Block 5b	9.87	17***	.02**			10.81	.19***	.02**		
Age				− 0.13*	0.65				− 0.04	0.55
Sex[a]				0.01	1.11				0.09	0.96
Race/ethnicity[b]				− 0.07	0.29				− 0.10	0.25
FKBP5[c]				0.09	1.15				0.12*	0.99
Trauma exposure[d]				0.00	1.27				− 0.03	1.08
Maternal PTSS				0.15	0.15				0.16	0.08
FKBP5 x maternal PTSS				0.25**	0.11				0.26**	0.09

Note. n = 205. PTSS = Posttraumatic stress symptoms.

[a]0 = Male, 1 = Female.

[b]0 = African American, 1 = Hispanic/Latine, 2 = European American, 3 = Other.

[c]0 = CC group, 1 = CT/TT group.

[d]0 = no exposure, 1 = probable/definite exposure.

*$p < .05$.

**$p < .01$.

***$p < .001$.

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