Do Adolescent Offspring of Women with PTSD Experience Higher Levels of Chronic and Episodic Stress?

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Abstract

Offspring of mothers with posttraumatic stress disorder (PTSD) are at higher risk for a range of negative developmental outcomes, including differing forms of psychopathology. This paper suggests that the multigenerational impact of trauma may be partially attributed to increased levels of stress experienced by these offspring during childhood and adolescence. Diagnostic interviews were conducted with over 800 women and their offspring. Experiences of stress were assessed using multiple measures. Results indicate that offspring of mothers with PTSD or high levels of PTSD symptoms experienced higher levels of lifetime exposure to major stress $F(1, 814) = 18.04$, $p < .001$, current chronic stress due to family relations $F(1, 813) = 7.22$, $p = .007$, and a higher level of objectively rated recent episodic life stress $F(1, 758) = 5.17$, $p = .02$, compared to offspring of women without PTSD. These findings remained significant after controlling for maternal history of depression.

Empirical research during the last 40 years has demonstrated that traumatic events have clinical and biological repercussions not only for individuals immediately exposed, but their offspring as well. This phenomenon first appeared in the literature in the mid to late 1960s with case studies of children of Holocaust survivors, which anecdotally reported signs of "mental disturbances" (e.g. Rakoff, Sigal, & Epstein, 1966 as cited in Kellermann, 2001). Following the addition of Posttraumatic Stress Disorder (PTSD) to the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*; American Psychiatric Association, 1980), research studies began comparing children of trauma-exposed parents with and without PTSD. A recent review of this literature suggests that paternal PTSD is associated with a range of negative outcomes including higher rates of aggression, anxiety, behavior problems, and psychiatric symptoms (Dekel & Goldblatt, 2008). Further, studies which have conducted three-way comparisons between children of parents with trauma and PTSD, children of parents with trauma exposure but no PTSD, and children of parents with no trauma exposure and no PTSD have shown that parental PTSD is associated with

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psychopathology in offspring above and beyond exposure to trauma (Rosenheck, 1986; Yehuda, Schmeidler, Giller, Siever, & Binder-Brynes, 1998).

Up to this point, the majority of the research on intergenerational transmission of trauma has focused on inherited biological vulnerabilities, which could be considered a diathesis (For review please see Yehuda, 2009). Very little published literature has examined the potential next step—whether children of parents with PTSD are exposed to increased levels of stress, which may also help explain their increased risk for negative outcomes. Stressful life events have been shown to not be entirely 'environmental' in that they can be non-random, as individuals are active participants in the design of their own environment (Kendler & Eaves, 1986). Therefore, it is possible that the environment that mothers with PTSD are creating for their children are more stressful than that of children whose mothers do not have PTSD. Additionally, studies in the field of behavioral genetics that have examined the influence of shared environmental factors on dependent life events have demonstrated the impact of family environment on individuals' choices and voluntary experiences (Bemmels, Burt, Legrand, Iacono, & McGue, 2008). A better understanding of the level of stress as well as the types of stress (e.g., chronic versus episodic) that children of parents with PTSD are exposed to will potentially allow for the development of a more comprehensive model of the multigenerational impact of trauma.

Importantly, few studies have delineated between subjective ratings and objective ratings of stressful events. This is essential to consider, especially given that within the trauma literature the research findings are relatively inconsistent regarding the association between objective measures of trauma severity and the later development of psychopathology. The associations between subjective measures of trauma exposure, such as perceived life risk, and the later development of psychopathology are often stronger, suggesting that it may be a person's perception of the trauma that matters, rather than an objective rating of severity (e.g., Fujita & Nishida, 2008). Thus, the current study adds to the existing literature by exploring whether offspring are exposed to a greater number of stressors in their environments, or whether these youth perceive the events they encounter as more stressful.

The present study takes advantage of a subsample of a large prospective birth cohort in which participants were selected based on varying levels of maternal depression during the prenatal and postpartum period. Participants in the cohort consisted of women and their adolescent offspring born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Australia (Keeping et al., 1989). At offspring age 15 years, we oversampled youth at high risk for depression on the basis of mothers' depression scores on the Delusions-Symptoms States Inventory (DSSI) of Bedford and Foulds (1977).

In this study, we hypothesized that offspring of mothers with PTSD symptomatology would encounter higher rates of stressful life events, compared to offspring of controls, and that these associations would remain significant when maternal depression was controlled. Again, given the sample selection characteristics, it was essential to control for maternal depression in this sample to assess the unique contribution of PTSD on stress exposure in the offspring. In addition, we hypothesized that children of mothers with PTSD would be exposed to higher levels of episodic and/or chronic stress during childhood and adolescence, and that these offspring would perceive episodic stressors as more severe than controls. Overall, these hypotheses and analyses offered a different approach to the concept of the transmission of intergenerational stress in the hopes of providing a better understanding of the maternal PTSD—child stress relationship.

Methods

Participants and Procedure

Participants in the current analyses consisted of women and their adolescent offspring selected from a prospective birth cohort study of 7,223 children born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Australia (Keeping et al., 1989). The original cohort was predominantly Caucasian and of lower-middle and working-class socioeconomic status (SES). The Mater-University of Queensland Study of Pregnancy (MUSP) originally investigated the children's physical, cognitive, and psychological health as a function of pregnancy and obstetric conditions, birth weight, and psychosocial conditions, and to predict age 5 health, development, and behavior. Mothers in the sample were interviewed at their first prenatal clinic visit (mean gestation 18 weeks) and provided additional details about their infants 3–4 days after delivery, when the child was ages 6 months, 5 years, and 13 years old.

In preparation for a youth age 15 follow up we examined maternal depression scores on previously collected (i.e., pregnancy, birth, six months and five years) administrations of the Delusions-Symptoms States Inventory (DSSI) of Bedford and Foulds (1977). These scores were used to identify women varying in level and frequency of elevated depression histories (or never depressed), using specific algorithms (e.g., elevated scores on two or more occasions, elevated scores on only one occasion). We oversampled for women with higher DSSI depression scores such that in the final selected group, 32% of the mothers reported no or few depressive symptoms over the 4 assessments, and 11% reported severe depression two or more times; all the others had scores in between these extremes, reflecting less severe or less chronic symptoms. Although it was selected to represent varying levels of maternal depression, this subsample was considered high-risk in that, relative to the original birth cohort, it represented an oversampling of mothers with high levels of self-reported depressive symptoms. After sample selection for the age 15 follow-up, maternal diagnostic information was collected as described below.

A total of 991 families were targeted for inclusion in the high-risk subsample. Of the 991 identified, 815 consented and were included in the age 15 follow-up (82%); 68 families could not be located; 103 declined to participate; 3 could not participate due to the child's hearing or visual impairment; 1 child had died; and 1 child withdrew his data from the study.

The current study was focused on the youth from the age 15 follow-up. This sample included 815 youths (49% male) and their mothers. Child ethnicity was 91% white, and 9% minority (Asian, Pacific Islander, and Aboriginal). Median family income at the 15-year follow-up indicated lower/middle SES, and median mothers' education was grade 10 (equivalent to U.S. high school graduation).

The sample for the current study did not differ from the overall birth cohort in terms of youth gender $\chi^2(2, N = 7,223) = 0.6$, $p = .42$, ethnic minority representation $\chi^2(3, N = 7,018) = 6.10$, $p = .11$, maternal education $t(N = 7,164) = 1.33$, $p = .18$, or family income $t(N = 6,747) = 0.10$, $p = .92$.

Interviews with the mother and 15 year- old child were conducted in the family homes. Interviewers were blind to the mother's psychiatric history, and a team of two interviewers conducted the parent and youth interviews separately and privately. The team of interviewers was trained by Dr. Hammen to conduct the diagnostic evaluations and life stress interviews and all interviewers were advanced graduate students in clinical psychology. This study was reviewed and approved by Institutional Review Boards at Mater

Hospital, the University of Queensland, UCLA and Emory University. The parents gave written informed consent and families were compensated for their time.

Measures

Maternal Axis I diagnoses in the current study were based on the Structured Clinical Interview for *DSM-IV* (First, Spitzer, Gibbon, & Williams, 1995) administered in its entirety, when the child was 15 years of age. Ratings by independent judges of 52 cases yielded weighted Kappa values of .80 and above for current and past diagnoses.

Maternal PTSD was operationalized as meeting lifetime *DSM-IV* (American Psychiatric Association, 1994) diagnostic criteria for PTSD or having a lifetime history of subclinical PTSD. Subclinical PTSD was defined as significant symptoms of PTSD with some impairment, but either the duration or the number of symptoms failed to meet full criteria. A total of 118 women (14%) were classified as having PTSD ($N = 46$) or subclinical PTSD ($N = 72$) according to these criteria. For ease of exposition, we will refer to these women as those with full and partial PTSD. Maternal depression was operationalized as meeting lifetime *DSM-IV* diagnostic criteria for Major Depressive Disorder (MDD). A total of 275 women (34%) were classified has having MDD according to the criteria. Fifty-eight women (7% of the sample) met criteria for full or partial PTSD and MDD.

Youth lifetime stress was measure at the age 15 follow-up. Youth completed a self-report questionnaire adapted from the lifetime stress interview of Kessler & Magee, 1993 which focused on their exposure to ten childhood adversities including parental death, parental marital conflict, and serious illnesses and accidents. Each adversity was scored as 1 (*yes*) and 0 (*no*), and the total number of adversities was summed as a measure of youth lifetime stress.

Youth chronic stress was measured via a semistructured interview for adolescents developed from earlier versions of the UCLA Life Stress Interview. This interview measures chronic strain/functioning in adults and children (e.g., Hammen, 1991) and was administered to children at the age 15 follow- up. The adolescent version used in the current study consisted of ongoing (past 6 months) conditions in six domains: social life (peer functioning), close friendship, romantic relationships or dating interest, relations with family members, academic performance, and school behavior. Each domain was scored on a 5-point scale with behaviorally specific anchors such that 1 represented superior functioning and 5 represented severe difficulties. Interrater reliabilities were based on two independent judge's ratings of audio taped interviews, with sample sizes between 88–96 for individual items (depending on completeness of tape recordings). Single-measure intraclass correlations were: social life .63, close friendship .76, relationship with family members .84, romantic relationship .55, academic performance .94, and school behavior .88. A sum of the chronic stress ratings was used as a summary measure of chronic stress ranging from 6 to 30, with higher scores indicating greater total chronic stress.

Youth episodic stress was assessed with the UCLA Life Stress Interview at the age 15 follow-up (Hammen, 1991; Hammen et al., 1987). Assessment of episodic events covered the last 12 months, and probes were based on the contextual threat model of Brown and Harris (1978). Interviewers prompted youth to describe significant stressful life events in several domains using standard queries. They probed for information related to the events' impact, past familiarity with similar events, expectedness, and available support.

Total number of episodic stressful life events occurring during the 12-month period prior to the age 15 follow-up was calculated for use in the analyses. In addition, subjective and objective levels of episodic stress were calculated as follows:

For each event, the interviewer asked the youth to make a subjective rating of the impact of the stress on a scale of 1 (*no impact*) to 5 (*severe impact*). An average of the subjective ratings of negative life event impact was used as the measure of subjective episodic stress.

Narratives of each event were presented to a team of four to five raters who rated the likely impact of each life event on an average person in a similar situation. Raters rated each event on a scale of 1 (*no impact*) to 5 (*severe impact*) and were kept blind to psychiatric diagnosis, symptomatology, and the individual's subjective report of stress. An average of the team ratings of negative life event impact was used as the measure of objective episodic stress.

Interrater reliabilities between the Australian rating team and a second rating team at UCLA yielded intraclass correlations of .92 for severity ratings across 89 cases.

Results

Preliminary analyses revealed that there were no significant group differences in the youth's gender, socioeconomic status, or ethnicity based on maternal PTSD status. Youth who had a mother with PTSD were slightly older ($M = 15.24$, $SD = .39$ versus $M = 15.17$, $SD = .25$; $t(813) = 2.79$, $p = .04$).

Maternal PTSD and MDD were significantly associated in this sample $\chi^2(1, N = 815) = 14.66$, $p < .001$. Whereas 31.1% of women without PTSD qualified for a lifetime diagnosis of MDD, this percentage rose to 49.2% in the women with a history of PTSD.

Bivariate correlations between measures of stress and maternal diagnosis are presented in Table 1. Many of the measures of youth chronic stress were significantly positively correlated, although effect sizes were small. The subjective and objective ratings of youth episodic stress were moderately correlated $r(757) = .42$, $p < .001$.

Using factorial analysis of variance, group (maternal full or partial PTSD by maternal MDD) differences in youth lifetime stress, youth chronic stress (social life, family relations, close friend, romantic relationships, academic, school-behavior, and total), and youth episodic stress (number as well as subjective and objective ratings) were examined. Controlling for comorbid MDD, maternal PTSD was significantly and positively associated with offspring reports of lifetime stress $F(1, 814) = 18.04$, $p < .001$, $\eta^2 = .02$, chronic stress in family relations $F(1, 813) = 7.22$, $p = .007$, $\eta^2 = .01$, and objective levels of recent episodic stress $F(1, 758) = 5.17$, $p = .02$, $\eta^2 = .01$. Similar trends were noted for total reports of chronic stress $F(1, 813) = 3.53$, $p = .06$, $\eta^2 = .01$, as well as chronic stress in close friendships $F(1, 813) = 3.08$, $p = .08$, $\eta^2 = .01$. Controlling for comorbid PTSD, maternal MDD was significantly and positively related to offspring reports of lifetime stress $F(1, 813) = 16.41$, $p < .001$, $\eta^2 = .02$, total reports of chronic stress $F(1, 813) = 8.87$, $p = .003$, $\eta^2 = .01$, and chronic stress in close friendships $F(1, 813) = 6.24$, $p = .01$, $\eta^2 = .01$. A similar trend was noted for chronic stress in social life $F(1, 813) = 3.65$, $p = .06$, $\eta^2 = .01$.

To ensure that the trauma that caused the mother's PTSD was not overlapping with the youth reported lifetime stress, we ran an additional post-hoc analysis predicting the youth reported lifetime stress measure. For this analysis, we restricted the maternal PTSD groups to only those women whose onset of PTSD occurred prior to the birth of the youth in the study (approximately half of the mothers with PTSD fell into this category). The ANOVA once again revealed a main effect of maternal PTSD on youth reports of lifetime stress, even when comorbid maternal depression was controlled $F(1, 764) = 7.13$, $p = .008$. Youth whose mothers had PTSD had a higher level of lifetime stress than youth whose mothers did not ($M = 1.97$, $SD = 1.74$ and $M = 1.47$, $SD = 1.43$, respectively).

A significant interaction was also noted between maternal PTSD and maternal MDD in the prediction of objective ratings of episodic stress levels $F(3, 759) = 6.11$, $p = .01$, $\eta^2 = .00$. As can be seen from the pattern of means presented in Table 2, children of mothers with PTSD that was not comorbid with MDD evidenced the highest levels of objectively rated recent episodic stressors.

Discussion

This study found that adolescent offspring of mothers with a lifetime history of PTSD reported higher levels of lifetime stress and chronic stress related to family relationships than offspring of mothers without PTSD. While these at-risk youth did not experience significantly more recent episodic stressors, the events that they experienced were objectively rated to be more stressful than those of youth of mothers without PTSD. Contrary to our hypothesis, although there was a high correlation between objective and subjective stress ratings of recent episodic events experienced by these youth, there was no maternal PTSD group difference in the subjective ratings of the stressors. Importantly, the noted associations between maternal PTSD and offspring exposure to stress remained significant in this sample, even when maternal depression was controlled.

Our results suggest that offspring of mothers with PTSD experienced increased lifetime exposure to major stressors as well as chronic stress in their family relationships. These findings are in line with the research demonstrating that negative life events are not randomly distributed across individuals in a population, but tend to occur more frequently in the lives of those who are at risk due to poverty or parental psychopathology.

It should be noted that the effect sizes in this study were quite small, suggesting that many other factors besides maternal PTSD may be important to consider in terms of risk of childhood stress exposure. For example, our results suggest that offspring of mothers with major depression are also at increased risk for chronic and episodic stress experiences in childhood and adolescence. Other maternal diagnoses or social or biological contexts may make similar or more significant contributions to the prediction of stress exposure.

The current study has other limitations that should be noted as well. The measure investigating lifetime stress was retrospective and did not ask participants to specify when the stressor occurred as it simply asked if any of the events had occurred prior to age 15. Therefore, it may be difficult to draw specific conclusions regarding the temporal effect of the stressor on later outcomes. This sample was drawn from a prospective birth cohort, and although a high-risk subset was used, it remains a community study. Because of low prevalence of women with PTSD who met full diagnostic criteria in the sample, women with subclinical PTSD were also included in the PTSD group. However, this suggests that our results may be an underestimate of the effects of maternal PTSD on offspring stress exposure. Finally, because of the nature of the study and original population, there was much less racial diversity than optimal. Additional studies are needed to replicate these findings in a more racially diverse sample.

It is notable that children of mothers with PTSD reported significantly higher levels of chronic stress within the interpersonal domain (e.g., relations with family members and close friendships) but not in other domains such as academic performance or school behavior. More research is needed to better understand why parental PTSD may affect certain domains of functioning but not others. Interestingly, objective levels of stress rather than subjective levels were higher in the offspring, suggesting that having a mother with PTSD potentially affects the objectively perceived environment in which the child is raised; it does not simply increase the stress sensitivity of the offspring. In fact, the pattern of

results (a significant difference on the objective but not the subjective ratings of the same episodic events, in particular for children of mothers with PTSD that is not comorbid with depression) may suggest that offspring of parents with PTSD have a decreased or a muted sensitivity to stress. The discrepancy between the subjective and objective ratings of episodic stressors is smallest for this group. Potential bias in the subjective ratings of stress is an area that has not been explored in the literature to date, and is an important component to our understanding how trauma can have a multigenerational impact.

Although the current study focused on the relationship between PTSD symptoms and child stress, an additional finding that bears attention is the higher levels of lifetime stress and certain types of current chronic stress (e.g., family relations) in the offspring of mothers suffering from comorbid MDD and PTSD symptoms. These findings are in-line with theoretical models suggesting increased risk for offspring of parents suffering from compound stressors (Goodman & Gotlib, 1999), such as comorbid depression and interpersonal stress (Hammen et al., 1987; Hammen, Shih, & Brennan, 2004). Results from the current paper add to these models suggesting that mothers' PTSD symptoms may lead to specific patterns of risk for offspring.

When examined in conjunction with research on inherited biological vulnerabilities, our findings offer a more comprehensive picture of how trauma can have a multigenerational impact. It appears that the children of parents with PTSD may be exposed to higher levels of stress during early childhood, and more severe stressors during adolescence. It is likely that this exposure to more intense stressors plays a role in the increased likelihood of negative outcomes for offspring of parents with PTSD.

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Table 1

Means, Standard Deviations, and Correlations of Stress Variables and Maternal Diagnoses

Variable	M	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Mother PTSD	0.14	0.35	--											
2. Mother MDD	0.34	0.47	.05											
3. Social life	2.28	0.47	.05	.09*	--									
4. Family	2.34	0.58	.12*	.16*	.20*	--								
5. Close friend	2.21	0.50	.07*	.09*	.48*	.21*	--							
6. Romantic	2.15	0.37	.01	.05	.15*	.20*	.08*	--						
7. Academic	2.67	0.94	.03	.05	.15*	.25*	.11*	.07	--					
8. School	2.19	0.78	.04	.10*	.12*	.32*	.07	.08*	.59*	--				
9. Total chronic	13.85	2.23	.08*	.14*	.50*	.60*	.47*	.33*	.76*	.74*	--			
10. Number episodic	3.21	2.08	.01	.08*	−.03	.15*	−.12*	.10*	.10*	.23*	.15*	--		
11. Subjective episodic	2.41	0.79	.00	.02	.05	.12*	.02	.11*	−.01	.04	.08*	.08*	--	
12. Objective episodic	1.88	0.39	.09*	.01	.06	.13*	.04	.05	.00	.07	.09*	−.04	.42*	--
13. Early life stress			.17*	.19*	−.03	.15*	−.12*	.10*	.10*	.23*	.26*	.18*	.08*	−.04

Note. $N = 814$. PTSD = partial or full posttraumatic stress disorder; MDD = meeting DSM-IV diagnostic criteria for Major Depressive Disorder.

*
$p < .05$.

Table 2

Means and Standard Deviations of Offspring Measures of Stress by Maternal Diagnosis

	PTSD and MDD (*n* = 58)		PTSD without MDD (*n* = 60)		MDD without PTSD (*n* = 217)		Neither MDD nor PTSD (*n* = 480)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Lifetime Stressors	2.45	1.97	1.77	1.73	1.74	1.53	1.21	1.36
Current Chronic Stress								
Social Life	2.39	0.51	2.28	0.53	2.33	0.54	2.25	0.43
Family Relations	2.61	0.69	2.40	0.64	2.44	0.60	2.26	0.54
Close Friends	2.39	0.57	2.21	0.61	2.25	0.55	2.18	0.44
Romantic Relationships	2.16	0.42	2.14	0.39	2.18	0.39	2.13	0.36
Academic	2.80	1.08	2.68	0.92	2.72	1.00	2.64	0.89
School Behavior	2.31	0.89	2.23	1.01	2.29	0.84	2.12	0.70
Total	14.26	2.76	13.95	2.50	14.19	2.53	13.58	1.58
Recent Episodic Stressors								
Number	3.41	2.06	3.13	1.81	3.46	2.24	3.08	2.03
Subjective Stress Level	2.44	0.75	2.37	0.66	2.44	0.85	2.40	0.78
Objective Stress Level	1.88	0.33	2.05	0.59	1.89	0.39	1.85	0.37

Note. PTSD = partial and full PTSD