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Peer victimization, depressive symptoms, and high salivary cortisol predict poorer memory in children

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ABSTRACT

The predictive relations of peer victimization, depressive symptoms, and salivary cortisol on memory in 168 children aged 12 at Time 1 (T1) were examined using a longitudinal design in which data were collected on four occasions over a 2-year period. Results indicated that: (1) peer victimization, depressive symptoms, and evening cortisol were stable over time, (2) peer victimization and elevated symptoms of depression were concurrently linked at each time, (3) T1 peer victimization predicted elevated symptoms of depression at T2 which in turn predicted lower cortisol levels at T3, and (4) controlling for earlier associations, T3 peer victimization, depressive symptoms, and higher morning and evening cortisol levels uniquely predicted memory deficits at T4. The links between elevated cortisol, symptoms of depression, and poor memory are consistent with published research on depressed adults and extend the findings to children exposed to peer victimization. These findings highlight that peer abuse is harmful and may impact children's long-term mental health and memory functioning.

1. Introduction

Peer victimization is more than an occasional fight or disagreement between peers; it entails the *repeated*, *intentional* humiliation and oppression of a person who has *less power* than his or her aggressor(s) (Olweus, 1999). Peer victimization is pervasive among students in elementary and secondary schools around the world. Canadian prevalence data of children aged 11, 13, and 15 indicate that over one third of youth are victimized by their peers (UNICEF, 2007), the fourth highest peer victimization rate out of 21 economically advanced countries surveyed (UNICEF, 2007). Two more recent population-based studies of 16,799 and 11,152 Canadian children aged 8–18 show similar rates (Vaillancourt, Brittain et al., 2010; Vaillancourt, Trinh et al., 2010).

The high prevalence of bullying among Canadian children and youth constitutes a major health problem, given what is known about the impairment associated with peer victimization (see Vaillancourt, Clinton, McDougall, Schmidt, and Hymel (2010), for a review). Children and youth who are bullied by peers, relative to non-victimized youth, report lower self-esteem, are more lonely and socially withdrawn, and are more anxious and depressed. Bullied children and youth report being unhappy at school, liking school less, feeling unsafe, and perform less well academically. They also report more headaches, stomachaches and other somatic complaints, which may reflect stress-related illness. Given the adversity that these children and youth endure at school, the results of longitudinal studies demonstrating negative outcomes across a range of symptoms and disorders is not surprising (e.g., Arseneault et al., 2006).

It is clear that the experience of being bullied by peers is stressful. Accordingly, researchers have begun to examine the associations between the stress of being bullied and activation of the hypothalamic–pituitary–adrenal (HPA) axis. The HPA axis is triggered when the organism is physically or psychologically challenged (i.e., stressed). Exposure to stress results in a cascade of hormones released, beginning with a first, quick response of the autonomic nervous system leading to enhanced catecholamine activity with epinephrine and norepinephrine being released from the adrenal medulla. A second, slower response ensues with corticotropin releasing factor (CRF) being released from the hypothalamus, followed by adrenocorticotropin hormone (ACTH) from the

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pituitary, culminating in the release of glucocorticoids (GCs; cortisol in humans) from the adrenal cortex (see Lupien et al., 2005; Wolf (2003), for review).

Cross-sectional studies of bullied children have shown an association between peer abuse and HPA axis dysregulation as measured via salivary cortisol. Specifically, researchers have found that peer victimization is associated with *lower* levels of basal cortisol (e.g., Kliewer, 2006; Knack, Jensen-Campbell, & Baum, 2010; Vaillancourt, Duku et al., 2008). Although this pattern of hyposecretion is interesting insofar as higher cortisol would be expected, given the stressful nature of this interpersonal issue, it is consistent with published research in the area of child maltreatment and post-traumatic stress disorder (e.g., Bremner & Vermetten, 2001; Shea, Walsh, MacMillan, & Steiner, 2005). In their recent meta-analytic review of chronic stress and the HPA axis, Miller, Chen, and Zhou (2007) stated that "current theories view cortisol deviations in both directions as potentially detrimental; whether elevations or declines are pathogenic depends on the condition" (p. 26). Results of this meta-analysis revealed that chronic and severe stress tended to be associated with lower morning concentration of cortisol ($d = -0.08$) and higher afternoon/evening levels ($d = 0.18$), and that physical threats to self tended to be associated with lower morning ($d = -0.16$) and higher afternoon/evening ($d = 0.22$) levels of cortisol.

Bullying is a special case of aggression that is characterized as extreme and chronic and entails physical and verbal threats to self as well as social ostracism (Olweus, 1999; Vaillancourt et al., 2010). Given the differential links between morning and afternoon/evening cortisol to chronic stress, we examined repeated measures of morning and evening cortisol in relation to bullying. We hypothesized that exposure to bullying would be associated with the dysregulation of cortisol levels but did not specify a direction (high versus low).

Considering the established link between HPA axis dysregulation and exposure to bullying (e.g., Knack et al., 2010; Vaillancourt, Duku et al., 2008), we sought to extend these findings by examining the longitudinal effects of peer victimization and HPA dysregulation on memory. It is clear from the extant nonhuman animal and human literature that memory is influenced by the secretion of GCs during acute, and to a greater extent, chronic stress (Lupien et al., 2005; Wolf, 2003). Indeed, nonhuman animal and human studies have shown that excessive exposure to GCs is associated with memory impairment and hippocampal atrophy (e.g., Lupien et al., 1998). Most studies to date have focused on declarative memory, and hence the hippocampus, when assessing the effects of GCs on cognition. Focusing on the hippocampus makes sense in that GCs bind to both the mineralocorticoid (MR) and glucocorticoid (GR) receptors which are both located in the hippocampus. However, MR receptors seem to be present exclusively in the limbic system, while GR receptors are also distributed in the subcortical and cortical structures with a preferential distribution in the prefrontal cortex (Lupien et al., 2005, p.226). According to Lupien and colleagues (2005), GCs should affect the frontal lobe as well as the hippocampus, something that is often overlooked by researchers examining the effects of stress on memory function. In the present study, we assessed the longitudinal effects of an extreme and chronic stressor, being bullied in childhood, and HPA dysregulation on cognitive functioning using well-validated measures that assess both hippocampal- and prefrontal cortex-dependent memory functions.

We also extended our inquiry to examine the association between depressive symptoms and memory. The relation between stressful life events and the onset of depression is well established (e.g., Maciejewski, Prigerson, & Mazure, 2001) as is the relation between higher levels of cortisol (basal and reactive) and depression (e.g., Gold, Goodwin, & Chrousos, 1988; Goodyer, Herbert, & Altham, 1998), and the strong association between depression and memory deficits (e.g., Burt, Zembar, & Niederehe, 1995). Prolonged stress is hypothesized to cause dysregulation of the HPA axis and increased GCs which in turn results in hippocampal neurotoxicity and associated memory deficits (e.g. Sapolsky, Romero, & Munck, 2000). Studies of peer-victimized children show robust concurrent and longitudinal associations with depression (see Hawker & Boulton, 2000). Furthermore, a recent study by Rudolph, Troop-Gordon, and Granger (2011) found that, after adjusting for earlier levels of depressive symptoms, peer victimization among 9-year-olds predicted elevated depressive symptoms 1 year later in children who showed high levels of cortisol while waiting for a peer-oriented social challenge to take place in the laboratory. For children with low levels of anticipatory cortisol, peer victimization did not predict depressive symptoms. We hypothesized that it is this factor of cortisol dysregulation that is critical in the process.

Using a longitudinal design in which data were collected on four separate occasions over a 2-year period, we examined the predictive relations of peer victimization, depressive symptoms, and salivary cortisol on memory in children aged 12 at Time 1 (T1). This age group was chosen based on prevalence studies showing that peer victimization peaks at this age (middle-school years; Pepler et al., 2006; Teicher, Samson, Sheu, Polcari, & McGreenery, 2010; Vaillancourt, Brittain et al., 2010; Vaillancourt, Trinh et al., 2010). Moreover, a recent study by Teicher et al. (2010) suggests that exposure to peer abuse during middle school was the "most consequential" with respect to elevated mental health symptoms such as anxiety, depression, dissociation, and drug use.

Based on the literature reviewed herein, we expected that (1) childhood peer victimization, cortisol dysregulation (morning and evening), and depressive symptoms would all be interrelated at each time point, and that each of these predictors would be stable over time. Although most studies on depressed children have reported higher cortisol levels (e.g., Lopez-Duran, Kovacs, & George, 2009), we nevertheless did not predict a direction because these studies have not taken into account the role of peer victimization, which as mentioned previously, has been linked to lower cortisol levels (e.g., Vaillancourt, Duku et al., 2008). We further hypothesized that (2) peer victimization would predict elevated depressive symptoms and that both peer victimization and elevated depressive symptoms would predict cortisol dysregulation (morning and evening). Finally, we expected that (3) taking into account the anticipated longitudinal association just mentioned (additive effects), T3 peer victimization, elevated depressive symptoms, and cortisol dysfunction (morning and evening) would each uniquely predict poorer hippocampal and prefrontal cortex functioning assessed at T4 using neuropsychological tests sensitive to memory impairment in these brain regions. These memory outcome measures were expected to be correlated with one another at T4. Our hypothesized model is presented in Fig. 1.

2. Method

2.1. Participants

A total of 168 (91 boys and 77 girls) predominantly Caucasian (78%) middle-income children aged 12 at T1 ($M_{\text{age in months}} = 147$, $SD = 9$) recruited from newspaper advertisements participated in at least one of the four phases of the study. T1, T2 and T3 data were collected 6 months apart and T4 data were collected 2–4 months after T3. The participation rate was as follow: T1 $n = 167$, T2 $n = 156$, T3 $n = 139$ and T4 $n = 134$. The attrition rate from T1 to T2 was 6.59% 9 ($n = 11$), from T2 to T3 was 10.90% ($n = 17$), and from T3 to T4 was 3.73% ($n = 5$). Note one participant had missing

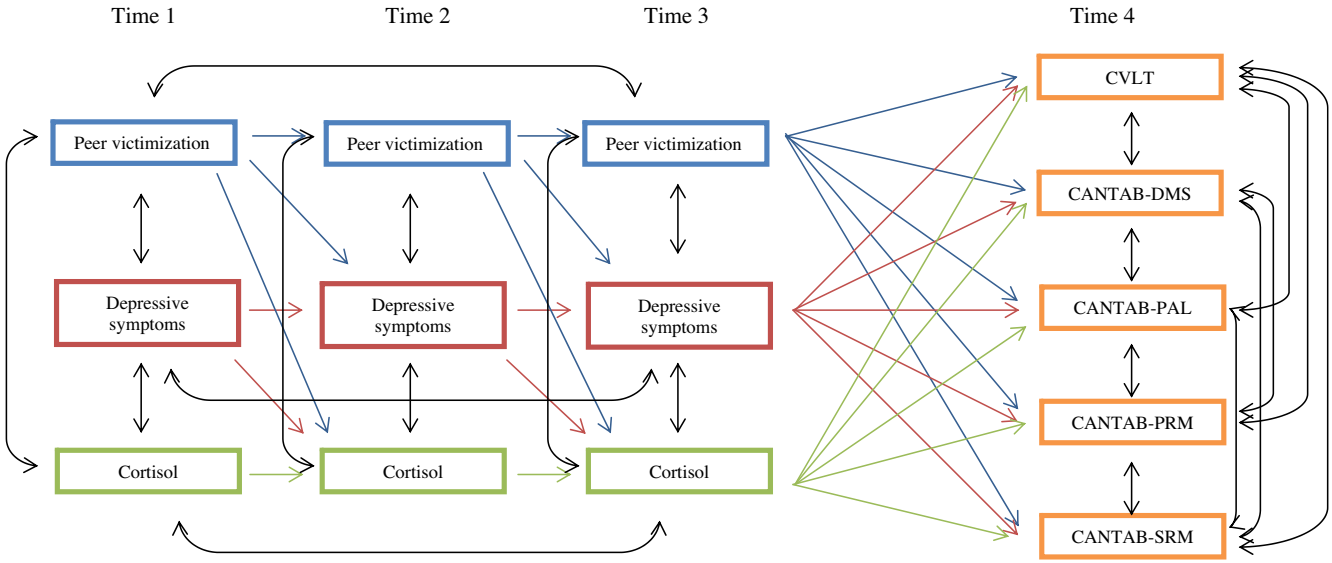

Fig. 1. Hypothesized path model.

data at T1 and T2 but participated at T3 and T4. Dropout status was not related to any of the measures used in this study.

Exclusion criteria for the study included parent and child reports of: (1) a history of childhood maltreatment, (2) a diagnosed psychiatric condition or significant psychological issue, (3) foster care placement, (4) a history of aggression directed toward peers and/or family members, and (5) current psychotropic medication or oral contraceptives use. Based on these criteria, 7 participants were excluded from the study at T1. At T3, one participant disclosed being abused by a caregiver and his/her data were excluded from further analyses and the proper authorities were notified (as described in the consent and assent forms).

2.2. Procedures

T1, T2 and T3 data were collected from participants in their homes and T4 data were collected in the laboratory of the first author. All participants were paid $20.00 for each data collection phase.

2.3. Measures

2.3.1. Peer victimization

Information about participants' experiences with bullying was collected at T1, T2, and T3 using an empirically validated self-report questionnaire (Vaillancourt, Brittain et al., 2010; Vaillancourt, Trinh et al., 2010). Participants were first asked to read a standard definition of bullying which clearly differentiated peer victimization from teasing. They were then asked to indicate the extent to which they had been bullied at school during the past 2 months along a 5-point scale: 0 "not at all", 1 "once or a few times", 2 "2 or 3 times a month (every month)", 3 "every week", and 4 "many times a week". Using this severity scale, participants rated their experiences with three types of peer victimization (physical, verbal and social) which were followed by detailed behavioral examples for each. A peer victimization composite score was computed by combining the frequency data on the three different forms of peer victimization into one score for each time point. Cronbach's $\alpha = 0.74$ T1, $\alpha = 0.70$ T2; $\alpha = 0.71$ T3.

2.3.2. Depressive symptoms

Information on participants' depressive symptoms was assessed at T1-T3 using the *Children's Depression Inventory* (Kovacs, 2001). The CDI (26 items) is a well-validated, psychometrically sound clinical scale of children's self-reported depressive symptoms. The suicide item was omitted from the CDI at the request of the Research Ethics Board. Cronbach's $\alpha = 0.86$ T1, $\alpha = 0.86$ T2; $\alpha = 0.89$ T3.

2.3.3. Salivary cortisol

Cortisol was measured using passive drool collected from participants in their homes 20 min after waking and at 21:00 h on Monday and Thursday at T1, T2, and T3. Several steps were taken to ensure proper data collection: (1) trained research assistants went to the homes of participants to teach them and their parents how to gather their saliva; (2) telephone reminders were made on the Sunday before the Monday collection; (3) participants were instructed not to eat during the 2-h period prior to spitting (passive drool) and to chew on a piece of Wrigley's Extra™ Peppermint sugar free gum before providing their sample; (4) multiple samples were obtained to ensure a valid representation of each child's adrenocortical activity; and (5) participants completed a food and time log, indicating the exact time at which they produced their sample, the food they had ingested 2 h prior to their saliva collection, as well as whether anything stressful had occurred on the day of saliva collection. An examination of the food and time log indicated good compliance with these instructions (i.e., no eating 2 h prior to providing samples and samples produced 20 min after waking and at around 21:00 h).

Enzyme immunoassay procedures were developed by modifying those methods reported by Munro and Stabenfeldt (1985; see also Carré, Muir, Belanger, & Putnam, 2006). All saliva samples were stored at −20 °C. Saliva was centrifuged at 3000 × *g* for 15 min and only the supernatant was assayed. The assay had a minimum sensitivity of 19.4 pg/mL and a range of 19.4 pg/mL to 40 ng/mL. Interplate variation (CV) was 6.45%, whereas intraplate variation was 6.51%.

Cortisol follows a clear circadian rhythm with higher values found in the morning than in the evening. Accordingly, separate morning and evening cortisol composites were created (Monday + Thursday/2 then log transformed). Log-transformations were used because the distributions of the cortisol measurements were skewed and too peaked. Multilevel modeling supported aggregating the cortisol data into these two categories (see Vaillancourt, Duku et al., 2008).

2.3.4. Memory

Hippocampal and prefrontal cortex functioning was assessed at T4 using the *California Verbal Learning Test Children's Version* (CVLT-C; Delis, Kramer, Kaplan, & Ober, 1994) and sub-tests from the *CANTABeclipse*® (Cambridge Neuropsychological Test Automated Battery, licensed from Cambridge Cognition, Ltd.).

The CVLT-C is a well-validated assessment of verbal learning and memory in children ages 5–16 (Delis et al., 1994). Imaging studies of adults have shown that the performance on the CVLT is related to activation of the left medial temporal lobe, the right hippocampus, and the right frontal lobe (Johnson, Saykin, Flashman, McAllister, & Sparling, 2001).

The CVLT-C presents two "shopping" lists (List A and List B) with 15 words each to children over five successive learning trials (List A), followed by a novel list of words (List B). After List B is presented, participants are asked to recall as many words as possible from List A with and without cueing. Finally, participants are asked to recall List A after a 20 min delay in which unrelated nonverbal tasks are performed, again with and without being cued (Delis et al., 1994; O'Jile et al., 2005). Although many measures can be obtained from the CVLT-C, in this study a composite score was created based on factor analytic results. Specifically, the following four sub-scales (standard scores) were combined into one summary measure of verbal memory with higher scores reflecting better memory ($\alpha = .93$): (1) List A Short Delay Free Recall (measure of short-delay recall), (2) List A Long Delay Free Recall (measure of long-delayed recall), (3) List A Short Delay Cued Recall (measure of confabulation and rate of forgetting), and (4) List A Long Delay Cued Recall (measure of confabulation and rate of forgetting).

The *CANTABeclipse*® is a well-validated touch screen computerized test used on people age 4 and up that does not rely on expressive language to assess visual memory, executive function, attention, semantic/verbal memory, and decision making (Cambridge Cognition, 2010). Given our interest in the effects of peer victimization, depressive symptoms, and cortisol on GC rich receptor sites we concentrated on assessing visual memory (medial temporal lobe area and hippocampus) using the following tests: (1) Delayed Match to Sample (DMS) difference score (% correct on 12,000 ms delay −% correct 0 ms delay); (2) mean total errors on Paired Associates Learning (PAL) (6 shapes + 8 shapes); (3)% correct on Pattern Recognition Memory (PRM); (4)% correct on Spatial Recognition Memory (SRM). Prior to completing these tests, participants were warmed-up with the Motor Screening test to familiarize them to the touch screen and the simple requirements of the tests. For a detailed description of these specific tests refer to e.g. Becker, MacQueen, and Wojtowicz (2009).

2.4. Analytic plan

For descriptive purposes, mean levels and bivariate correlations among peer victimization, depressive symptoms, and cortisol were examined along with possible sex differences given reported difference in depressive symptoms and peer victimization rates among boys and girls (e.g., Vaillancourt, Duku et al., 2008).

To examine the hypothesized longitudinal relations among our predictor variables (peer victimization, depressive symptoms, and cortisol) in relation to our outcome memory measures we conducted a path analysis in Mplus Version 5.1 (see Fig. 1). Two separate analyses were conducted, one for morning cortisol and the other for evening cortisol, in which the following were estimated: (1) concurrent correlations among peer victimization, depressive symptoms, and cortisol at T1, T2, and T3, and memory at T4, (2) predictive links between peer victimization, depressive symptoms, and cortisol across T1, T2, and T3, (3) cross-lagged effects between peer victimization at T1 and depressive symptoms and cortisol at T2 and peer victimization at T2 and depressive symptoms and cortisol at T3, and (4) predictive links between peer victimization, depressive symptoms, and cortisol at T3 on T4 memory (accounting for earlier longitudinal relations between predictors variables). All correlations and paths were freely estimated across time and no cross-group equality constraints were imposed in these models.

3. Results

3.1. Prevalence of peer victimization

Although in subsequent analysis peer victimization is treated as a continuous variable, prevalence rates are reported to place the findings in context with other research. Using the cut-off recommended by Solberg and Olweus (2003) to identify bullied children, 35.4% of children at T1, 27.8% at T2 and 21.4% at T3 were frequently bullied in the past 2 months.

3.2. Bivariate correlations: Peer victimization, depressive symptoms, and cortisol

As seen in Table 1, peer victimization was stable over time ($r = .46$ for T1–T2, $r = .45$ for T1–T3 and $r = .56$ for T2–T3) and not related to sex. Symptoms of depression were also very stable across the three time points ($r = .59$ for T1 to T2, $r = .65$ for T1–T3 and $r = .77$ for T2–T3) and not correlated with sex. Within time, log (cortisol) was positively correlated ($r = .23$ T1 AM and T1 PM; $r = .21$ T2 AM and T2 PM; $r = .37$ T3 AM and T3 PM). Stability was found for evening log(cortisol) ($r = .27$ for T1 to T2, $r = .19$ for T1 to T3 and $r = .24$ for T2 to T3) and not morning log(cortisol). Only T1 evening cortisol was related to sex ($r = -.16$) with girls being slightly higher than boys. Finally, depressive symptoms and peer victimization were related at each time point (r range = .33 to .42, $p < .001$) and only T3 depressive symptoms were associated with T1 evening cortisol ($r = .20$).

3.3. Bivariate correlations: Memory

Examining the correlations between the five memory outcome measures revealed that the CVLT (verbal memory) composite was positively correlated with pattern recognition memory (PRM; $r = .29$, $p < .001$) and spatial recognition memory (SRM; $r = .23$, $p < .008$). Paired associated learning (PAL) was negatively correlated with PRM ($r = -.21$, $p<.02$), and SRM ($r = -.27$, $p<.002$). Delayed match to sample (DMS) was not correlated with any of the other memory measures nor was the participants' sex. Note that PAL is a measure of the number of errors whereas PRM, SRM, and DMS are measures of correct responses and therefore negative correlations with the PAL were expected.

3.4. Longitudinal relations: Peer victimization, depressive symptoms and cortisol predicting memory

The two longitudinal models (morning and evening cortisol) showed good fit to the data, χ^2 (48) = 50.57, CFI = 0.995, RMSEA = 0.018 (95 percent CI = 0.000–0.054), TLI = 0.989 for morning cortisol and χ^2 (48) = 66.84, CFI = 0.964, RMSEA = 0.048 (95% CI = 0.012–0.074), TLI = 0.924 for evening cortisol. The moderating role of sex was not examined because of the lack of associations found at the bivariate level and because we did not want to reduce power for the path analyses. Results from these path models can be seen in Fig. 2 (morning cortisol) and Fig. 3 (evening cortisol).

Stability coefficients (standardized betas) were high for peer victimization ($b = .22$–$.46$) and depressive symptoms ($b = .29$ to .61). Only evening cortisol, and not morning cortisol, was stable

Table 1
Descriptive statistics and bivariate correlations.

	M	*SD*	Correlations											
			1	2	3	4	5	6	7	8	9	10	11	12
1. Sex	–	–	–											
2. T1 peer victimization	1.34	1.36	.08											
3. T2 peer victimization	1.28	1.14	.02	.46***										
4. T3 peer victimization	1.19	1.06	−.03	.45***	.56***									
5. T1 depression	0.25	0.23	.08	.43***	.38***	.33***								
6. T2 depression	0.23	0.23	−.06	.34***	.51***	.40***	.59***							
7. T3 depression	0.22	0.24	−.04	.37***	.37***	.35***	.65***	.77***						
8. T1 AM log(cortisol)	4.17	1.12	−.12	−.19	−.14	.014	−.06	−.03	.06					
9. T1 PM log(cortisol)	0.38	1.68	−.16*	−.12	−.11	.00	−.08	.14	.20**	.28**				
10. T2 AM log(cortisol)	3.93	1.54	−.12	−.06	.13	.04	.07	.13	.15	−.04	.02			
11. T2 PM log(cortisol)	0.81	1.82	−.16	−.11	.06	−.09	.01	.08	.15	.13	.27**	.21**		
12. T3 AM log(cortisol)	4.14	1.20	−.02	.03	−.03	−.03	−.00	−.17	−.11	.06	.08	.13	.14	
13. T3 PM log(cortisol)	1.16	1.97	.00	−.07	−.03	−.07	.13	.03	.10	.12	.19*	.13	.24**	.37***

Girls coded as 0 and boys as 1; cortisol unit = ng/mL.
* $p < .05$.
** $p < .001$.
*** $p < .0001$.

across time (b=.26 from T1–T2 and b = .19 from T2–T3). As predicted, a cross-lag effect was found between T1 victimization and T2 depressive symptoms (b = .17) when morning and evening cortisol were modeled. Moreover, as expected, T2 depressive symptoms predicted T3 cortisol dysregulation with higher depressive symptoms predicting lower evening cortisol levels (b = −.20). Consistent with our initial hypothesis, taking into account earlier associations between peer victimization, depressive symptoms, and morning cortisol, negative direct effects were found for T3 depressive symptoms on T4 CVLT (b = −.29) and spatial recognition memory (b = −.33), as well as for morning cortisol on delayed match to sample (b = −.19) and spatial recognition memory (b = −.21) (see Fig. 2). Contrary to initial predictions, a positive direct effect was found between T3 peer victimization on T4 spatial recognition memory (b = .22).

For evening cortisol, the negative direct effects were again found for T3 depressive symptoms on T4 CVLT (b = −.29) and spatial recognition memory (b = −.27), as well as for evening cortisol on delayed match to sample (b = −.22) and spatial recognition memory (b = −.26) (see Fig. 3). Moreover, evening cortisol was also negatively related to the CVLT composite (b = −.20). Once again, contrary to initial predictions, a positive direct effect was found between T3 peer victimization and T4 spatial recognition memory (b = .19).

4. Discussion

We examined the predictive links of peer victimization, depressive symptoms, and cortisol dysregulation on memory in children at age 12 at T1. The memory tests used in this study were ones that have been shown to be especially sensitive to the effects of elevated GCs—the hippocampus and the prefrontal cortex (Lupien et al., 2005). Results indicated that consistent with previous research, peer victimization and depressive symptoms were stable over one year (Sweeting, Young, West, & Der, 2006). We also found evidence for the stability of evening cortisol over time but not morning cortisol. A longitudinal study of 3- to 6- year-old children living in foster care and a community comparison group reported stable morning and evening cortisol pattern over a 12-month

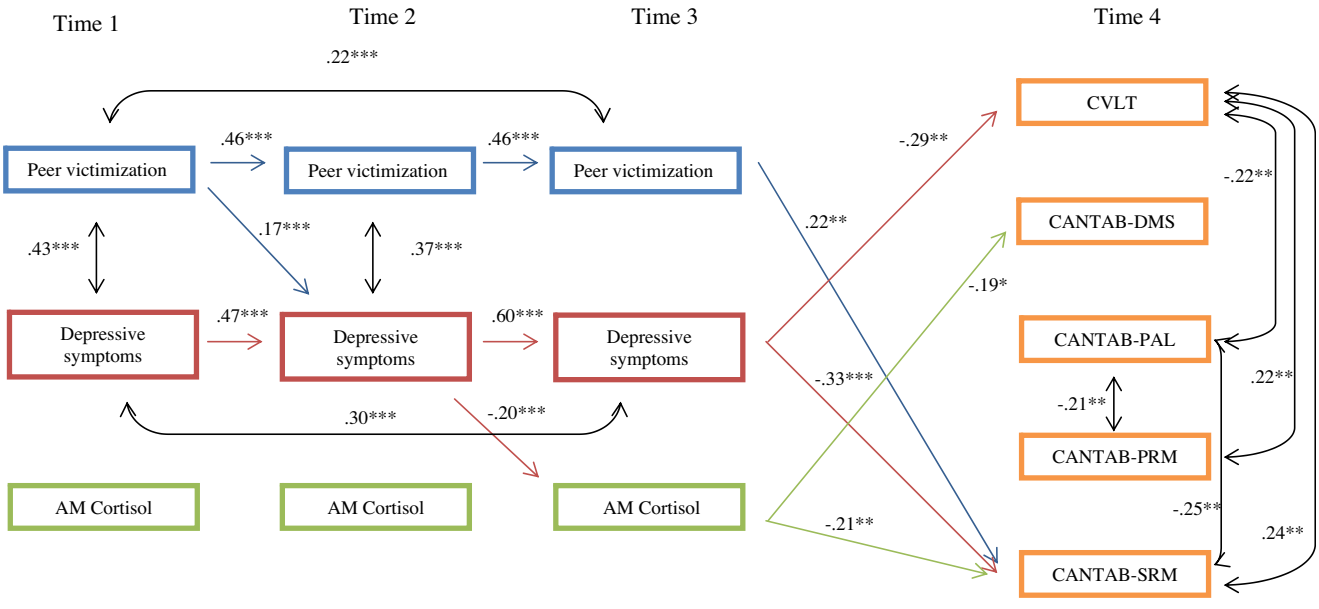

Fig. 2. Final Path Model (Morning Cortisol). *Note*. Only statistically significant standardized path coefficients are depicted. ***$p < .001$, **$p < .01$, *$p < .05$.

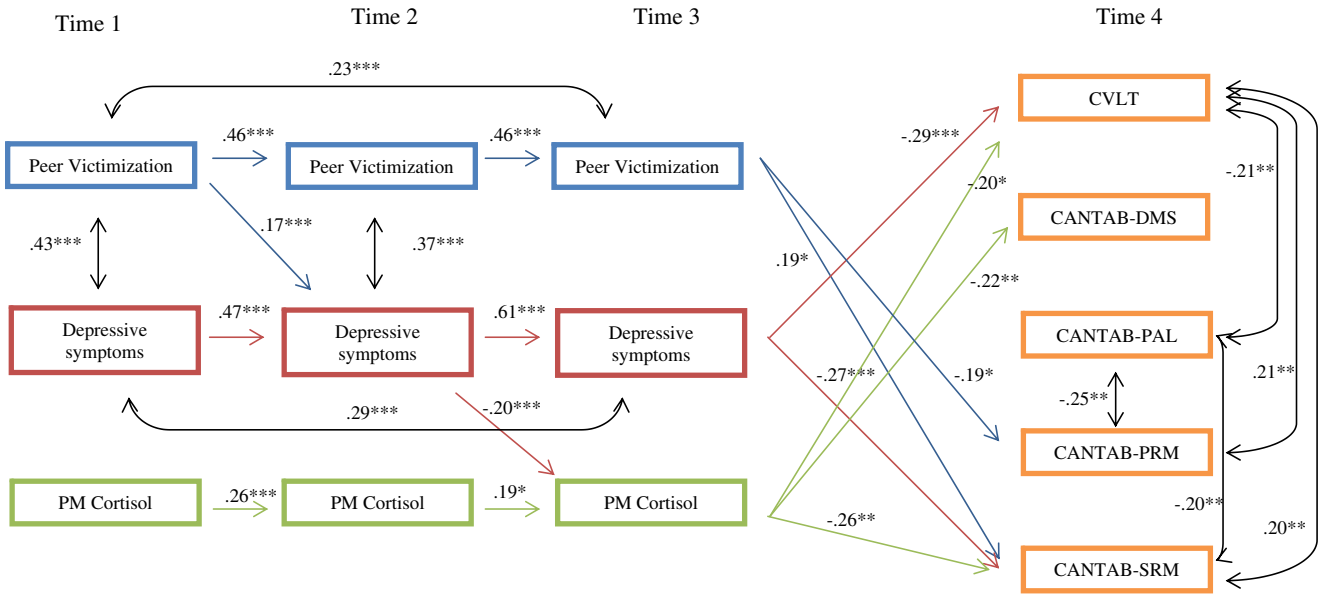

Fig. 3. Final Path Model (Evening Cortisol). *Note*. Only statistically significant standardized path coefficients are depicted. $^{***}p < .001$, $^{**}p < .01$, $^{*}p < .05$.

period, especially among foster care children (Fisher, Stoolmiller, Gunnar, & Burraston, 2007). The fact that we found stability only for evening cortisol may reflect a morning compliance issue. Kudielka, Broderick, and Kirschbaum (2003) reported that non-compliance with study instructions had an effect on measuring the cortisol awakening response (CAR). A proper evaluation of CAR requires that participants produce their sample directly upon waking and then again 30–40 min after and that they do not eat, brush their teeth, take certain medications, or smoke (Kudielka et al., 2003; Saxbe, 2008). We were concerned about instruction compliance given the age of our participants and instead chose to have participants produce their sample 20 min after waking. It is quite possible that some participants did not produce their sample after 20 min of waking and the lack of stability reflects this potential issue. It is worth highlighting however that all non-laboratory based studies examining morning cortisol are hampered by the same methodological challenge of not being able to verify wake time (Saxbe, 2008).

Consistent with previous research and our initial hypothesis, we found that peer victimization and elevated symptoms of depression were concurrently linked (e.g., Hawker & Boulton, 2000). Contrary to our initial hypothesis, however, cortisol was not concurrently linked to peer victimization or symptoms of depression. Nevertheless, peer victimization at T1 did predict elevated symptoms of depression at T2 consistent with the findings of Rudolph et al. (2011) and Sweeting et al. (2006). Depression in turn predicted lower morning and evening cortisol levels at T3. The link between elevated depressive symptoms and lower cortisol levels is inconsistent with most published studies on depression (see meta-analysis by Lopez-Duran et al. (2009)). This discrepant finding may be explained by methodological differences between the present study and other published studies. First, the link between high cortisol and depression has been typically reported in clinical studies of patients with a diagnosis of depression (Lopez-Duran et al., 2009). In this study, we examined symptoms of depression and not the disorder *per se*. Second, although some have reported that elevated cortisol predicts major depression in children and youth (e.g., Goodyer et al., 1998), none of the published studies are truly longitudinal in nature and only one study, to our knowledge, has taken into account the role of peer victimization. Rudolph et al. (2011) recently reported that peer victimization predicted depressive symptoms 1 year later in children who were high on anticipatory cortisol. No main effect was found between anticipatory cortisol and elevated depressive symptoms. In the current study, we examined basal cortisol (i.e., morning or evening levels) and not reactive cortisol (i.e., hormonal challenge) in children's natural environment (their home). We also took into account the relation peer victimization held to elevated depressive symptoms. Considering that stressors that are extreme, chronic, and involve a threat to the physical self have been shown to be associated with higher morning and lower evening cortisol (Miller et al., 2007), and that bullying is by definition repeated, extreme, and physical in nature, it seems plausible that higher depressive symptoms could predict lower cortisol (in particular evening cortisol) when peer victimization is taken into account. Studies linking high cortisol to high symptoms of depression have not considered history of adversity, and how this may be linked with depressive symptoms. Perhaps accounting for the role of peer victimization as one example, may change the direction of these findings. More longitudinal work in this area is needed to understand these results.

Some of the memory deficits found in association with elevated peer victimization, depressive symptoms, and cortisol levels were broadly consistent with the pattern of memory deficits observed in adults with major depression in a wide range of tasks, including the CVLT (MacQueen et al., 2003) and all of the CANTAB subtests employed here, reflecting deficits in prefrontal executive functions and medial temporal lobe memory functions in addition to fronto-striatal motivational deficits (Elliott et al., 1996). Moreover, depression status and cortisol levels, while highly correlated, appear to make independent contributions to memory deficits (Gomez et al., 2009; Hinkelmann et al., 2009). Although the hypothesized causal link between dysregulated cortisol, hippocampal memory deficits, and depression remains somewhat controversial, it is strongly supported by meta-analyses showing that hippocampal volume loss correlates with total lifetime duration of illness in depression (Campbell, Marriott, Nahmias, & MacQueen, 2004) and that such volume loss is only observed after more than one disease episode or more than two years of total illness (MacQueen, Yucel, Taylor, Macdonald, & Joffe, 2008). Thus, peer-victimized children who go onto suffer a dysregulated HPA axis may be more vulnerable to hippocampal neurotoxicity, resulting in persistent, irreversible memory deficits. This idea is an

important avenue for future study, as hippocampal volume loss predicts both treatment response rates and relapse in depression (Kronmuller et al., 2008; MacQueen et al., 2008).

In younger adults, reports of memory and other cognitive deficits in relation to stress, depression, and cortisol levels are mixed. For example, Castaneda et al. (2008) found only mildly compromised verbal learning in a depressed population of 21–35-year-olds. Conversely, in a large sample of unmedicated depressed individuals ranging in age from young adulthood to midlife, Grant, Thase, and Sweeney (2001) found no major memory deficits on a range of memory scales including several CANTAB subtests, whereas persistence of depressive symptoms did relate to performance on the CANTAB DMS task. A deficit on the CANTAB DMS has also been observed in a population of young adults in a self-reported "preclinical" first episode of depression (Becker et al., 2009); however, in contrast to the findings reported here, that study found no deficits on the CANTAB SRM, PRM or PAL. These mixed findings point to the necessity of employing a wide battery of memory tests and assessing the independent contributions of stress, depression, and cortisol on memory functions.

We speculate that some of the memory deficits we observed among bullied children and those with elevated depressive symptoms may be at least partially attributable to a reduction in hippocampal neurogenesis. It is well established in non-human animals that both stress and glucocorticoids suppress adult hippocampal neurogenesis (e.g. Tanapat, Galea, & Gould, 1998). Moreover, manipulations that suppress neurogenesis impair some but not all forms of hippocampal-dependent memory (Clelland et al., 2009; Dupret et al., 2008; Garthe & Kempermann, 2009; Shors et al., 2001; Snyder, Hong, McDonald, & Wojtowicz, 2005; Winocur, Wojtowicz, Sekeres, Snyder, & Wang, 2006). Based on these empirical findings and predictions from computational models, Becker and colleagues (Becker, 2005; Becker & Wojtowicz, 2007) proposed that the main function of hippocampal neurogenesis is to provide a contextually distinct signature to each memory trace, thereby mitigating against interference (see also Aimone, Deng, & Gage, 2010; Appleby & WIskott, 2009; Chambers and Conroy (2007); for related proposals). It is, thus, noteworthy that in the present study, higher morning and evening cortisol levels uniquely predicted poorer performance on several memory tasks including Delayed Match to Sample (DMS), Spatial Recognition Memory and Verbal Memory (CVLT), taking into account prior associations between peer victimization, depressive symptoms, and cortisol. Attending to the DMS finding first, Becker et al. (2009) reported a similar DMS deficit in young individuals likely to be in a preclinical stage of first episode of depression, as predicted by their theory of hippocampal neurogenesis. The CANTAB DMS task involves studying a complex unfamiliar visual pattern, and then after a delay of 0–12 s, making a 4-alternative forced choice as to which pattern was just seen. The four lures are all highly similar, making this a challenging recognition memory task. Such tasks are often referred to as "pattern separation" tasks as they require the formation of a highly accurate memory trace that can separate the original target stimulus from the overlapping sample patterns. Interestingly, rats with reduced neurogenesis also perform poorly on visual pattern separation tasks (Clelland et al., 2009). In sum, this pattern of results supports the view that memory deficits associated with elevated cortisol may relate, at least in part, to a decrease in neurogenesis, resulting in deficits on neurogenesis-dependent forms of memory. Fortunately, pathological brain changes including reduced neurogenesis may be partially reversed by factors such as physical exercise and exposure to enriched environments (Van Praag, Kempermann, & Gage, 2000; Van Praag, Kempermann, & Gage, 2005). This notion an important avenue for future research, as there may be a causal link between the restoration of neurogenesis and recovery from depression (Becker & Wojtowicz, 2007).

The CVLT taps into both hippocampal memory and prefrontal strategic control processes. Deficits in executive functions are well established in major depression, likely reflecting dysregulation in fronto-striatal and/or fronto-hippocampal circuits (Michopoulos et al., 2008). If the deficits on the CVLT in our peer victimized group were due to hippocampal dysfunction alone, then they should be more severe in individuals who show substantial hippocampal volume loss compared to those who do not. Interestingly, MacQueen et al. (2003) compared never-treated patients in a first episode of depression to those who had experienced multiple-episodes, and found that while both were equally impaired on words recalled in the CVLT, only the multiple episode group exhibited significant hippocampal volume loss. This result suggests that the deficits we observed on the CVLT in association with cortisol and depressive symptoms may reflect dysregulated prefrontal executive functions more so than a compromised hippocampal/medial temporal lobe memory system. A replication of our study with a larger battery of tests could tease apart these issues by independently probing executive and long-term memory functions.

Beyond cortisol and depressive symptoms, we also found that, as hypothesized, peer victimization uniquely predicted poorer memory but only on visual pattern recognition (PRM) measured using CANTAB. Unexpectedly, however, peer victimization also predicted better spatial recognition memory (SRM). In the depression literature, evidence of CANTAB SRM deficits is mixed, with some reports of SRM deficits in depressed adults (e.g. Elliott et al., 1996), and contradictory findings of normal SRM performance in adolescent unmedicated depressed girls, in spite of a range of other visual memory and executive deficits (Matthews, Coghill, & Rhodes, 2008). Interestingly, the CANTAB SRM, PRM and PAL are differentially sensitive to focal brain lesions, suggesting that the SRM may be more dependent upon the frontal lobe, while PRM and PAL are more dependent upon the medial temporal lobe (Owen, Sahakian, Semple, Polkey, & Robbins, 1995). Stress also differentially affects various types of memory. For example, in healthy young adult males, Luethi, Meier, and Sandi (2010) found that social stress resulted in impaired working memory but enhanced spatial recognition memory. We speculate that being subjected to a stressor such as bullying could lead to increased vigilance and enhanced spatial attention, explaining the positive association between peer victimization and SRM in our study. Such positive, adaptive responses to constant threat could enhance performance on spatial tasks, in opposition to the deleterious effects of dysregulated cortisol and depression.

4.1. Limitations

Our sample involved middle-income children, which limits the generalizability of these findings, as does the fact that almost 80% of our sample was Caucasian. We did not witness participants producing their saliva samples and therefore there could be issues with compliance. Although there were no sex differences at the bivariate level, Vaillancourt, Duku et al., 2008 showed that sex was an important moderator in the relation between bullying status and HPA activity. Specifically, when they modeled sex in their multi-level analysis they found that bullied boys produced more cortisol than non-bullied boys whereas bullied girls produced less cortisol than non-bullied girls. There are also notable sex differences in depression and bullying rates with girls being more depressed than boys post-puberty (e.g., Angold & Worthman, 1993) and boys being bullied more than girls (e.g., Vaillancourt, Duku et al., 2008). Larger studies are needed to examine the links presented in this study so that the possible moderating role of sex can be examined. As this is the first prospective study to examine bullying, depressive symptoms, and cortisol in relation to memory, it requires replication.

4.2. Conclusion and implications

Children who are bullied by their peers are subjected to adversity that is often chronic and involves humiliation. Such exposure is stressful and therefore not surprisingly, linked concurrently and prospectively to depressive symptoms in this study (Hawker & Boulton, 2000; Rudolph et al., 2011). Peer-victimized children also evince dysregulation of their HPA axis (e.g., Vaillancourt, Duku et al., 2008) in a way that is consistent with studies of individuals exposed to extreme and chronic stress that threaten a person's sense of safety (e.g., Miller et al., 2007). Depressed children typically have higher cortisol levels than non-depressed children (e.g., Lopez-Duran et al., 2009) and depressive symptoms have been linked to poorer memory in adults (Burt et al., 1995). Finally, nonhuman animal studies suggest a causal link between stress and high cortisol and their deleterious effects on memory (Wolf, 2003). Studies on humans, particularly adults, show similar results (Lupien et al., 2005; Wolf, 2003). Although these findings share similar links, to date, these variables have not been considered together in one explanatory model, nor have they been examined prospectively.

The present study examined peer victimization, symptoms of depression, and cortisol in relation to memory in one explanatory model and found results that were consistent with nonhuman animal studies and studies of depressed adults. These results underscore the aversive nature of bullying by demonstrating predictive links to elevated symptoms of depression which in turn predicted HPA dysregulation and ultimately poor memory functioning.

In a recent review of the bullying literature, Vaillancourt et al. (2010) called for researchers to reconsider the prevailing wisdom concerning the link between poor academic performance and peer victimization, arguing for research that went beyond the idea that psychological difficulties mediate the relation between negative peer experiences and poor academic performance (e.g., Buhs, Ladd, & Herald, 2006). Specifically, Vaillancourt and colleagues argued that "perhaps bullied children [did] poorly in school because of a structural change to their brain that [was] associated with functional differences (i.e., poor memory) that [were] mediated through the repeated activation of the HPA axis" (p. 297). Results of the present study provide initial evidence for this type of model and highlight the need to consider the biological effects of stress on the brain and on cognitive functioning developmentally.

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