

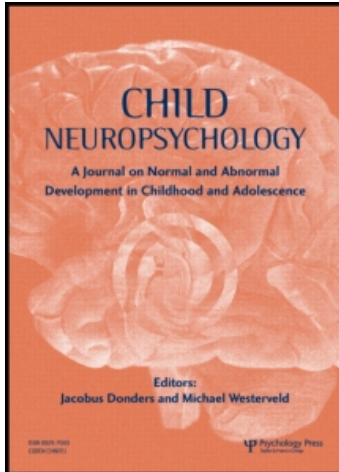
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### Relationship between the number of life events and memory capacity in children

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## Relationship between the number of life events and memory capacity in children

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Stressful life events can result into declined memory performance at later age. One hypothesis suggests that stress affects the hippocampus, a brain area important for memory functioning. This study explored a potential relationship between the number of negative stressful life events and hippocampus-dependent declarative but not hippocampus-independent procedural memory performance in a community sample of 255 children, aged 6–12 years. The findings revealed that negative stressful life events were negatively related to verbal declarative memory, but not to nonverbal declarative and procedural memory. The memory impairments could not be accounted for by attention and sleep disturbances, and parenting characteristics as perceived by the child did not influence the vulnerability for the stress-related memory impairments. These findings provide further insight into the deleterious effects of negative stressful life events on learning in school-aged children.

**Keywords:** Life events; Memory; Children; Stress.

### INTRODUCTION

A growing body of evidence suggests that stressful life events, such as the separation or divorce of parents or a change in school or residence, can have profound negative effects on memory function, and that the impairments are not confined to the information associated with the life events specifically (Frankola et al., 2010; Jouriles, Brown, McDonald, Rosenfield, & Leahy, 2008). One of the proposed explanations is that life events result into high levels of distress and that this can provoke a chronic state of hyperarousal (Jouriles et al., 2008). According to the inverted U-shape theory (Yerkes & Dodson, 1908), arousal beyond a moderate level impedes general cognitive performance by impairments in attention and executive function. In line with this, children and adults suffering from Posttraumatic Stress Disorder (PTSD), but also children with traumatic experiences

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but without PTSD, demonstrated performance decrements in a wide range of cognitive domains (Luksys Gerstner, & Sandi, 2009; Samuelson, Krueger, Burnett, & Wilson, 2010).

A more specific hypothesis pertaining particularly to the memory domain suggests that elevated levels of stress hormones due to one or more traumatic event affect hippocampus function. In response to stress, glucocorticoids (corticosterone in animals and cortisol in humans) are secreted from the adrenal glands through activation of the hypothalamus-pituitary-adrenal (HPA) axis (Sapolsky, 2003). Although glucocorticoids are central to a successful coping with short-term stressors, prolonged or recurrent exposure to glucocorticoids can have adverse effects on synaptic plasticity of the hippocampus (Sapolsky, Uno, Rebert, & Finch, 1990). Studies have demonstrated a smaller hippocampal volume and other morphometric differences in adults after exposure to severe stress during childhood (Bremner, 1999; Wheeler & Buckner, 2004). Stress-related decreases in hippocampal volume have not been found in children but might become manifest later during development (Woon & Hedges, 2008). The hippocampus plays a critical role in the formation of declarative memory — that is, memory for facts or specific events (Squire, Haist, & Shimamura, 1989) — but plays, however, a negligible role in procedural memory — that is, memory for skills that is thought to be related to other brain regions such as the striatum and cerebellum (Van Mier, 2000). In line with the model, hippocampus-dependent memory impairments were found in both rodents and humans, while hippocampal-independent cognition remained intact (Kirschbaum, Wolf, May, Wippich, & Helhammer, 1996; Young, Sahakian, Robbins, & Cowen, 1999). Human studies even demonstrated hippocampus-dependent memory impairment after moderate levels of chronic stress in daily life (Schwabe, Dalm, Schachinger, & Oitzl, 2008).

One other brain area possibly involved in the decreased memory function after stress is the prefrontal cortex (PFC). The PFC forms, together with the hippocampal region, a crucial part of the declarative memory network (Buckner, Kelley, & Petersen, 1999). The PFC organizes information, directs effective organizational strategies during encoding and is strongly implicated in memory retrieval, which promotes memory for associations among items in particular (Blumenfeld & Ranganath, 2007). Furthermore, the PFC controls “selection” processes that direct attention towards relevant information (Blumenfeld & Ranganath, 2007). It has been argued that memory impairments in PTSD may be secondary to attention problems; although recent evidence suggests that memory problems in PTSD in adults consists after removing the effect of attention problems (Johnsen, Kanagaratnam, & Asbjørnsen, 2008). Research has shown that the PFC seems to be particularly sensitive to architectural changes induced by chronic stress compared with other brain regions (Arnsten, 2009). Accordingly, patients with PTSD showed diminished prefrontal cortex activity (Carrion, Garrett, Menon, Weems, & Reiss, 2008), and particular memory impairments associated with disturbed executive control by the prefrontal cortex (Johnsen & Asbjørnsen, 2009).

The present study is the first to investigate the potential relation between stressful life events and declarative, but not procedural, memory in a community sample of children. Although declarative memory impairments have been demonstrated in clinical groups of children with PTSD, it is unknown whether children who experienced negative stressful life events, and who do not suffer (anymore) from PTSD, also show declarative memory impairments. This study therefore may provide important new insight into the relation between the number of negative stressful life events and declarative memory function in the general child population. Based on studies regarding the cumulative effects of stressful life events and risks leading to clinical symptoms (Appleyard, Egeland, Van Dulmen, &

Sroufe, 2005; Gustafsson, Nilsson, & Svedin, 2009), we expect to find a negative relationship between the number of stressful life events and declarative memory performance. We hypothesize that the association of life events and memory impairments are confined to the declarative memory domain, and that procedural memory function is spared.

Elevated arousal levels and PFC impairment associated with stress can both affect attention that is needed to select relevant information as well as for the encoding of information. Since we expect that hippocampal dysfunction is the major mechanism underlying potential memory impairments associated with life events, and that attention problems are secondary to this, we hypothesize that potential effects of life events on declarative memory are not fully accounted for by stress-related deterioration of attention function (Yehuda, Golier, Halligan, & Harvey, 2004).

Furthermore, life events during childhood are strongly associated with sleep disturbances (Koskenvuo, Hublin, Partinen, Paunio, & Koskenvuo, 2010). There is ample evidence that processes of sleep are important for memory function (Diekelmann & Born, 2010), also in children (Backhaus, Hoeckesfeld, Born, Hohagen, & Junghanns, 2008). Sleep disturbances in children affect memory (Carskadon, Harvey, & Dement, 1981) and academic performance (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010); although not all studies were in congruence (Randazzo, Muehlbach, Schweitzer, & Walsh, 1998). The current study controls for possible mediation of sleep problems in the association between life events and memory function.

Moreover, positive parent-child interaction can serve as a buffer or protective factor for the negative impact of life events on the child's stress system (Gunnar & Quevedo, 2007; Skopp, McDonald, Jouriles, & Rosenfield, 2007). Positive parenting allows children to elicit help by expressing their negative emotions; a mechanism through which parents can prevent elevations in stress responses activated during stressful life events (Gunnar & Quevedo, 2007). The stress-buffering model (S. Cohen & Wills, 1985) proposes that, when faced with negative experiences, individuals with greater support from families and friends are less likely to experience negative outcomes, while poor family circumstances may increase the impact of stressful life events. A recent study showed that positive parent-child interaction was found to be associated with a weaker relation between intimate partner physical violence and explicit memory function of preschoolers (Jouriles et al., 2008). Therefore, we evaluate whether certain types of positive mother-child interactions that provide cognitive stimulation for children, or that calm children who have been exposed to stressful events, may moderate the potential negative influences of life events on declarative memory.

## METHOD

### Participants

Children aged 6 to 12 years were recruited from 16 different regular primary schools in different urban and rural areas in the Netherlands. After permission of the schools' headmaster, parents were contacted through a letter. To avoid response bias, only the global aims of the study, without specific hypotheses, were mentioned. A total of 275 children and their parents were willing to participate. To minimize confounds, exclusion criteria included previously diagnosed mental retardation ( $IQ < 70$ ), learning disabilities, developmental disorders such as attention deficit/hyperactivity disorder (ADHD), anxiety disorder, and autism spectrum disorder (ASD), and a history of diagnosed sleep problems.

A total of 16 children were excluded from participation for the following reasons: ADHD ( $n = 6$ ), ASD ( $n = 5$ ), hearing problems ( $n = 2$ ), dyscalculia and working memory problems ( $n = 1$ ), dyslexia, anxiety and sleep problems ( $n = 1$ ), and giftedness ( $n = 1$ ). All children were administered two subtests (Vocabulary and Block design) of the Wechsler Intelligence Scale for Children-III (WISC-III; Kort et al., 2005), to estimate general level of intelligence, since subtest scores correlate in the .90 range with full-scale IQ (Sattler & Saklofske, 2001). Four children who had estimated full-scale IQ scores in the deficient range ( $< 70$ ; Resing & Blok, 2002) were excluded from the sample, as intelligence is found to be highly associated with short-term memory and working memory (Tillman, Nyberg, & Bohlin, 2008). All parents gave written informed consent. Ethical approval was given by the institutional review board of Leiden University.

The final sample consisted of 255 children (44% boys) (mean age = 9.58 years;  $SD = 1.81$ ). Most children lived with their biological parents (98%). An average score of educational level for the parents was based upon the scores of both the father and the mother, using the International Standard Classification for Occupations (Ganzeboom & Treiman, 1996), using seven response categories: (a) no education, (b) primary, (c) lower secondary, (d) higher secondary, (e) lower tertiary, (f) middle tertiary, and (g) higher tertiary. An average score of both parents was calculated. Most parents (98%) had at least 12 years of education (i.e., vocational education or lower secondary school), 74% of the parents had at least completed higher secondary school. The majority of children were Caucasian (85%); other children most commonly had a Turkish, Moroccan, Surinam, or Antillean background (10%), and for 5%, the ethnic group was unknown. The Child Behavior Checklist (CBCL; Achenbach, 1991) was completed by each child's primary caretaker and consists of 113 problem-behavior items that are rated as not true (0), somewhat true (1), or very true (2). Mean test-retest reliability in the original standardization sample was  $r = .89$ , interparent agreement was  $r = .70$ , and stability over two years was  $r = .71$  (Achenbach, 1991). The standardized  $T$ -scores of the internalizing, externalizing, and total behavioral problems were within the normal range (respectively,  $M = 47.85$ ,  $SD = 9.39$ ;  $M = 47.16$ ,  $SD = 9.13$ ;  $M = 46.54$ ,  $SD = 9.54$ ).

PTSD symptoms were determined with the Child PTSD Symptom Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001), which was completed by each child under supervision of the test leader. The instruction to the test leader was to ask for any event that was distressing to the child. In case the child could not report an event (17%), the questionnaire was not further administered. The CPSS consists of 26 items and assesses PTSD-symptom severity according to the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*; American Psychiatric Association [APA], 1994) PTSD-criteria, and functional impairment. The CPSS yields a total symptom severity score (ranging from 0 to 51) and a total severity-of-impairment score (ranging from 0 to 7), with higher scores indicating more functional impairment. Internal consistency ranged from  $\alpha = .70$  to  $.89$ . Of the children that reported a distressing event, 11 (4.3% of total sample) reported a potential *DSM-IV* Criterion-A event (APA, 1994) (e.g., death of close family member, intensive surgery, near accident). Three out of these 11 children showed a symptom severity score above the clinical cutoff level of 15 (1.2% of total sample).

## Measures and Procedures

**Stressful Life Events (SLE).** Stressful life events were assessed with the Questionnaire of Life Events (Veerman, Janssen, Ten Brink, Van der Horst, & Koedoot,

2003), a 24-item questionnaire measuring intense, stressful events that can occur within families, such as parental death or divorce, hospitalization or serious illness, or traffic accidents. The questionnaire contains 15 items that describe an a priori negative event, three items that describe an a priori positive event, and six items that describe an a priori ambiguous event. The questionnaire was completed by the primary caretaker of the child. The items addressed occurrence (yes/no), frequency, date, and whether the event was experienced as positive or negative by the child. A previous version of the questionnaire (Veerman, Ten Brink, Van der Horst, & Koedoot, 1997) yielded a test-retest reliability over a 9-month period of  $r = .80$ . For the purpose of the present study, only the events endorsed as having been a negative experience to the child were used for the analyses. In the present sample, 78% experienced at least one event; the mean number of events was 1.64 ( $SD = 1.33$ , range 0–5). The total (cumulative) number of negative life events was the main variable for the statistical analyses. In order to enable more detailed analysis between the different levels of experienced stress, a second variable was created. The main variable was recoded for this purpose into three different categories, based on the experienced number of stressful life events, and applying a classification system that yielded the most balanced distribution: (a) no-events group (22.2% of the children), (b) one or two events group (52.4%), and (c) three or more events group (25.4%).

**Verbal Declarative Memory (15WT).** Verbal declarative memory was assessed with a Dutch version of the Auditory Verbal Learning Test (AVLT): the 15-Words Test (15-WT; Van den Burg & Kingma, 1999), which assesses immediate memory span, new learning, and recognition for verbal material. Research in children with Closed Head Injury (CHI) and children with Fetal Alcohol Spectrum Disorders (FADS) indicated that a smaller hippocampal volume was related to worse verbal learning and memory performance on the California Verbal Learning Test, a test that is very similar to the 15-WT (Di Stefano et al., 2000; Willoughby et al., 2008). The 15-WT consists of five learning trials, in which the child was presented an identical list of 15 common, but unrelated, words through an audio file. After each trial, the child was asked to recall as many words as could be remembered. After 30 minutes, an unannounced delayed recall trial took place. Immediately after the delayed recall, a yes/no recognition test was administered, consisting of the 15 target words and 15 nontarget words. The parallel test-retest reliability was  $r = .70$  (Van den Burg & Kingma, 1999). The dependent variables were (a) Total Learning; the sum of correctly recalled words on Trials 1–5 (max = 75), (b) Delayed Recall; the number of words recalled correctly on the free recall trial (max = 15), and (c) Recognition; the number of words correctly recognized in the recognition trial (hits and correct rejections) (max = 30).

**Nonverbal Declarative Memory (RVDLT).** Nonverbal declarative memory was assessed with a revised, computerized, version of the Rey Visual Design Learning Test (RVDLT; Wilhelm, 2004; original version by Rey, 1964, as cited in Spreen & Strauss, 1991), which assesses immediate memory span, new learning, and recognition for nonverbal material. Previous studies reported deficits in visuospatial learning and recall on the Rey Osterrieth Figure Task (ROCF) in patients with right hippocampal damage (Bohbot et al., 1998). Similarly, in children with FADS, smaller right hippocampal volumes were associated with deficits in visuospatial learning and recall on the ROCF (Willoughby et al., 2008). The ROFC is comparable to the RVDLT in that it assesses visual and nonverbal memory; however the RVDLT is favored over the ROFC due to its superior design and psychometric properties (Wilhelm, 2004). Fifteen simple geometric figures were presented,

1 per 2 seconds, on a computer screen. After all test items were presented, the child was asked to draw as many items as possible, each item on a separate sheet of paper. This procedure was repeated another four times. After a delay of 20 minutes, the child was unexpectedly asked to draw as many items as could be remembered (delayed recall). Then, a recognition task followed with 15 targets and 30 nontargets. Test-retest reliability over a 3-month period is  $r = .87$  (Wilhelm, 2004). The dependent variables were (a) Total Learning; the sum of memorized items over Trials 1 to 5 (max = 75), (b) Delayed Recall; the total number of words recalled correctly in delayed recall trial (max = 15), and (c) Recognition; the sum of the correctly identified items in the recognition trial (hits and correct rejections) (max = 45).

**Procedural Memory (MT).** A mirror-tracing task was used to measure procedural memory, which was a modification of the Tracking Task, which is part of the Amsterdam Neuropsychological Tasks Program (ANT; De Sonneville, 1999). Mirror tracing is a motor skill that requires learning new associations between vision and hand movement and improves with repetition of the task, even when the individual has impaired recall and recognition for the task (i.e., explicit memory) (Gabrieli, Corking, Mickel, & Crowdon, 1993). The child was asked to trace a circle with a mouse cursor as quickly and accurately as possible between an inner and outer circle presented on a computer screen. For detailed task descriptions, see, for example, Huijbregts et al. (2003). For the purpose of this study, a mirror transformation was applied by placing the mouse upside down, so that each movement of the mouse cursor on the computer screen was the opposite of the child's hand movement. Thus, each child had to learn a new association between vision and hand movement. After three practice trials, five mirror-reversed trials followed, and after a 2-hour interval again five mirror-reversed trials. Variables are (a) mean of the absolute distance between the cursor trajectory and the midline per circle segment (60 radially equal segments in total), (b) standard deviation of the 60 distance values, and (c) trial duration (movement time), reflecting accuracy, stability, and speed, respectively. Procedural memory capacity is reflected in improvement of performance from Trial 1 to Trial 10 (i.e., the decrease of mean absolute distance, standard deviation of distance values, and trial duration). Therefore, the outcome variables were calculated as difference scores: Trial 1 minus Trial 10. With regard to the validity of the task, analyses were performed to evaluate whether procedural learning took place with this task; that is, whether there was an increase in speed with successive trials. The results showed that there was a significant increase in speed with successive trials,  $F(1, 143) = 4.85, p < .05, \eta_p^2 = .03$ , which provides support for the validity of the task. No significant effects were found for accuracy and stability.

**Attention (FA & CBCL Attention).** A subtask of the Focused Attention task of the ANT (De Sonneville, 1999) was used to measure selective attentional capabilities. The child was required to search a display on a computer screen, consisting of four consonants presented in the corners of a square, for one of three target letters on two relevant diagonal locations. Detailed task descriptions can be found in, for example, Mennes et al. (2004). Dependent variables were mean reaction time (RT) and error percentage. The Attention problems subscale (11 items) of the CBCL (Achenbach, 1991) was used as a measure of attention problems. Muris and Meesters (2003) found that the scale was highly correlated to other measures of inattention and impulsivity symptoms.

**Sleep (Sleep).** Sleep quality and quantity was measured with the Children's Sleep Habits Questionnaire (CSHQ; Owens, Nobile, McGuinn, & Spirito, 2000), including 33 items, covering eight domains of sleep problems, with a 3-point answering scale (usually, sometimes, rarely), to be completed by the primary caretaker. Test-retest reliability was found to be within the acceptable range (0.62 to 0.79), and internal consistency was adequate ( $\alpha = .68$ ) (Owens, Spirito, & McGuinn, 2000). Sleep quality (sleep disturbance) was operationalized as the total score (higher scores indicating more disturbed sleep). Due to a bimodal distribution, this variable was recoded into an ordinal variable consisting of five groups with equal numbers of participants (quintiles). Quantity of sleep was reported as the usual total sleep time (TST) on schooldays by the primary caretaker.

**Perceived Parenting Behavior (EMBU-C).** The child version of the Egena Minnen Beträffande Uppfostran (EMBU; Perris, Jacobsson, Lindström, Von Knorring, & Perris, 1980), the EMBU-C (Markus, Lindhout, Boer, Hoogendijk & Arrindell, 2003), was used to measure perceived parenting behavior. The original EMBU-C contained 81 items. For the present study, a shorter version (52 items) was used, developed by Markus et al. (2003). The questionnaire consists of four scales: Emotional warmth (giving special attention, praising for approved behavior, unconditional love, and being supportive and affectionately demonstrative), Rejection (hostility, punishment, derogation, and blaming of the child), Overprotection (fearful and anxious for the child's safety, guilt engendering, intrusiveness [e.g., being expressed in meddling in the child's affairs, in high standards in the choice of friends, accomplishment in school and behavior in general]), and Favoring subject (parental favoritism directed toward the child). For the purpose of the present study, only the scales Emotional Warmth (EW) en Rejection (Rej) were included in the analyses. The EMBU-C uses a 4-point Likert-scale (1 = *No*, 2 = *Yes, Sometimes*, 3 = *Yes, often*, 4 = *Yes, almost always*). The internal consistency for the scales is  $> .83$  (Markus et al., 2003) and test-retest stability over 2 months is  $r > .78$  (Muris, Meesters, & Van Brakel, 2003). The items for both parents were highly associated, for Rejection ( $r = .68$ ) and for Emotional warmth ( $r = .79$ ). Therefore, they were combined into a single measure. When information for one of the parents was missing, the composite score was based on the information of only one of the parents.

All children were tested individually by trained undergraduate students during school hours (8.30am–3.30pm) in a quiet room (duration approximately 2.5 hours, including breaks). The order of the tests was the same for all children.

## Statistics and Data Analysis

Pairwise-deletion for missing data was employed. The study variables were examined for specific assumptions applying to statistical tests that were used. Outliers and normality of the variables were examined by means of descriptive statistics, histograms, scatter plots, and quantile-quantile plots (QQ plots). Variables that did not fulfill the statistical assumptions for normality were transformed with a logarithmic transformation. The results obtained with the logarithmically transformed variables did not differ from the results with the original variables. Therefore, the results with original variables are presented. Demographic variables (age, gender, and parental educational level) were included as covariates in all analyses. Total intelligence score (TIQ) was not included as a covariate in the analyses, for reasons discussed elsewhere (Dennis et al., 2009). Effect sizes were estimated by means of partial eta squared ( $\eta_p^2$ ). Large effects correspond with  $\eta_p^2 \geq .14$ ,



moderate effects with  $\eta_p^2 \geq .06$ , and  $\eta_p^2 \leq .14$ , and weak effects with  $\eta_p^2 \leq .06$  (Cohen, 1988).

Hierarchical regression analyses were performed to investigate the contribution of the number of negative stressful life events as predictor for (verbal and nonverbal) declarative memory performance and procedural memory performance, while controlling for demographic variables. These analyses were repeated with inclusion of attention measures, to examine whether negative stressful life events relate to memory performance above and beyond attention performance.

In order to enable analysis of the effect of life events on learning curves by means of analysis of variance repeated measures (ANOVA-RM), groups with different cumulative numbers of life events were used (i.e., no events, 1–2 events,  $\geq 3$  events). A two-way ANOVA-RM was performed with trials as within-subjects factor, group as between-subjects factor, and demographic variables as covariates. The ANOVA-RM was followed by separate analyses of covariance (ANCOVA) to analyze group differences in Total Learning, Delayed Recall and Recognition, covarying for demographic variables.

Path analyses were conducted to test the hypotheses that attention or sleep mediates the relation of stressful life events with declarative memory. We used Sobel's test (1982) to test the mediated pathway, which directly tests the significance of the indirect (mediated) effect. Potential moderation of the relation of stressful life events with declarative memory by perceived parenting was analyzed by entering interaction terms in the regression model. All analyses were conducted using the Statistical Package for Social Sciences (SPSS for Windows, version 16.0, SPSS Inc., Chicago).

## RESULTS

### Relation between Negative Stressful Life Events and Memory Performance

A higher number of negative stressful life events was related to lower scores on 15-WT Total Learning,  $\beta = -.19$ ,  $t(230) = -3.57$ ,  $p < .001$ , and Delayed Recall,  $\beta = -.21$ ,  $t(210) = -3.47$ ,  $p < .001$ , after controlling for the demographic variables (Table 1). In Figure 1a and b, the 15-WT Total Learning and Delayed Recall scores are plotted against the number of negative stressful life events (all groups with more than five life events consisted of less than three cases and were therefore omitted in Figure 1a and b). There was no relationship between life events and 15-WT Recognition. Furthermore, there was no relationship between life events and any of the nonverbal declarative, or procedural memory measures over and above the effects of the demographic variables.

### Analysis of Group Differences in Verbal Declarative Memory Performance

There were no significant differences in demographic and behavioral characteristics between the different event-rate groups (Table 2). A significant effect was found of group for total learning,  $F(2, 229) = 9.71$ ,  $p < .001$ ,  $\eta_p^2 = .08$ , and delayed recall,  $F(2, 209) = 5.52$ ,  $p < .01$ ,  $\eta_p^2 = .05$  (Table 3), with significantly better Total learning and delayed recall scores in the no-events group than in the 1–2 event-, and  $\geq 3$  events group. The ANOVA-RM Trial x Group interaction was not significant, indicating that

**Table 1** Hierarchical Linear Regression Analysis of Life Events Predicting Memory Performance.

	Step 1 <sup>a</sup>		Step 2 <sup>b</sup>			
	R <sup>2</sup>	F(df)	R <sup>2</sup>	ΔR <sup>2</sup>	ΔF (df)	B
15-WT Total Learning	.37	45.06(2, 231)**	.40	.03	12.71(1, 230)**	-.19
15-WT Delayed Recall	.24	21.56(3, 211)**	.28	.04	12.02(1, 210)**	-.21
15-WT Recognition	.13	11.07(3, 230)**	.14	.01	3.36(1, 229)	-.11
RVDLT Total Learning	.33	38.63(3, 231)**	.34	.00	1.21(1, 230)	-.06
RVDLT Delayed Recall	.28	29.97(3, 231)**	.28	.00	0.27(1, 230)	-.03
RVDLT Recognition	.08	6.55(3, 221)**	.08	.00	0.15(1, 220)	-.03
MT movement time (Trial 1–Trial 10)	.06	3.21(3, 167)*	.06	.00	0.78(1, 166)	.07
MT standard deviation (Trial 1–Trial 10)	.13	8.49(3, 167)**	.13	.00	0.10(1, 166)	-.02
MT absolute deviation (Trial 1–Trial 10)	.08	4.96(3, 167)*	.09	.00	0.53(1, 166)	.06

<sup>a</sup>Variables entered: age, gender, parental educational level.

<sup>b</sup>Variable entered: negative stressful life events.

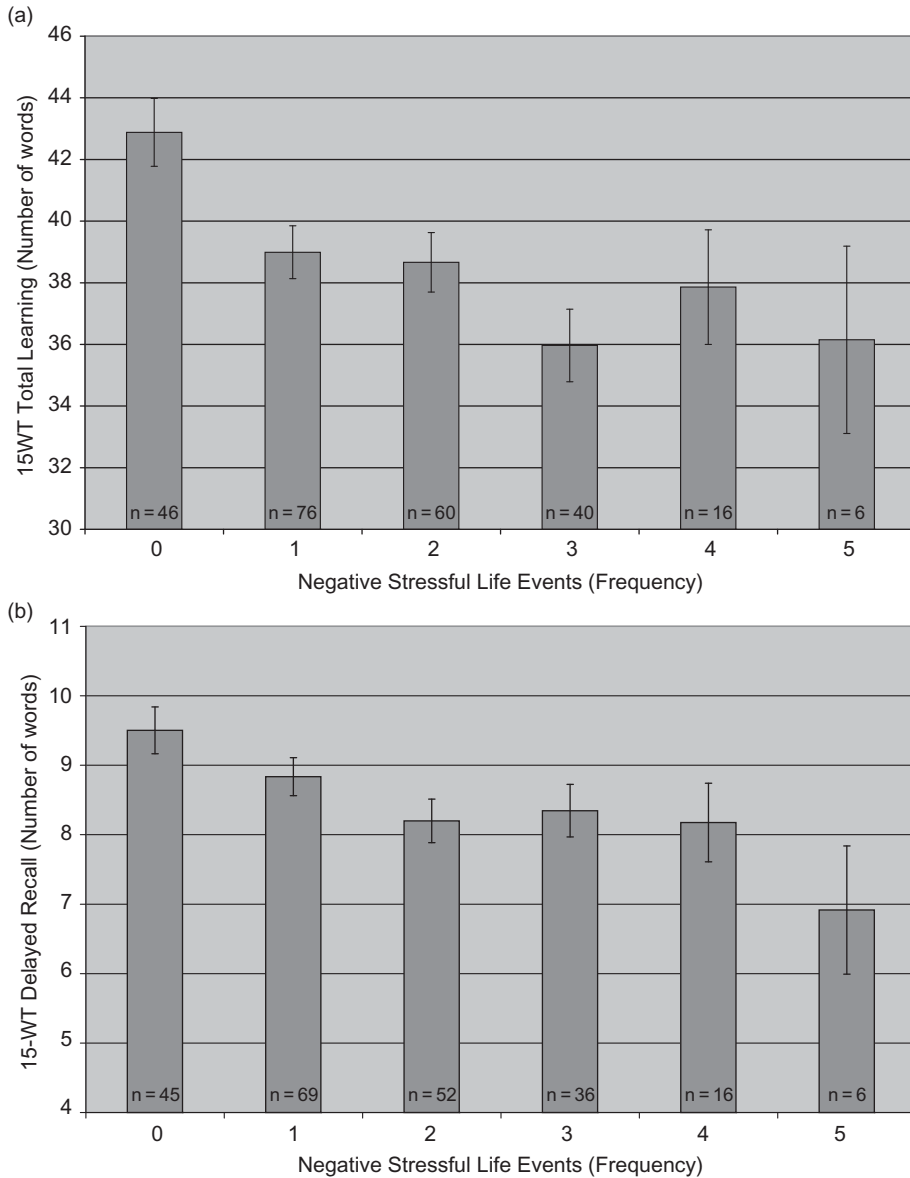
\* $p < .05$ . \*\* $p < .001$ .

group differences did not change across the successive trials (Figure 2). A further analysis was done to investigate whether the group differences in delayed recall could be explained by the differences in previous learning: An ANCOVA for delayed recall with total learning as additional covariate yielded a significant covariate effect for total learning,  $F(1, 208) = 163.30$ ,  $p < .001$ ,  $\eta_p^2 = .44$ , but the overall group effect for delayed recall disappeared.

### Potential Mediators and Moderators of the Relation between Negative Stressful Life Events and Memory

The CBCL attention score significantly predicted 15-WT Total Learning over and above the effects of demographic variables and negative stressful life events,  $\beta = -.11$ ,  $t(220) = -2.01$ ,  $p < .05$ , while none of the focused attention variables significantly predicted verbal declarative memory over and above the effects of demographic variables and negative stressful life events. However, further regression analyses revealed that the contribution of the number of negative stressful life events to 15-WT Total Learning scores was significant,  $\beta = -.17$ ,  $t(232) = -3.23$ ,  $p = .001$ , above and beyond CBCL attention scores and demographic variables. Likewise, the contribution of negative stressful life events to 15-WT Total Learning remained significant above and beyond the focused attention task variables and demographic variables.

For 15-WT Delayed Recall, only focused attention Part 2 significantly predicted verbal declarative memory over and above the effects of demographic variables and negative stressful life events,  $\beta = -.19$ ,  $t(201) = -2.85$ ,  $p < .01$ ,  $\eta_p^2 = .04$ . Negative stressful life events contributed significantly to 15-WT Delayed Recall,  $\beta = -.16$ ,  $t(204) = -2.54$ ,  $p = .012$ , above and beyond CBCL attention scores and demographic variables. Also, 15-WT Delayed Recall was significantly predicted by negative stressful life events above and beyond the focused attention task variables and demographic variables. The results of the path analyses revealed that none of the attention measures showed significant mediation in the relation between life events and memory variables.



**Figure 1** Performance on 15-WT Total Learning (a) and 15WT Delayed Recall (b) plotted against the number of negative stressful life events (errors bars represent standard errors).

Series of hierarchical linear regression analyses were conducted to examine the ability of sleep quality (total sleep disturbance) and quantity (total sleep time) to predict verbal declarative memory (15-WT Total Learning and Delayed Recall) over and above the effects of demographic variables and negative stressful life events. The analyses revealed that sleep quality and quantity did not significantly predict verbal declarative memory over the effects of the demographic variables and negative stressful life events. The path analyses revealed neither sleep quality nor quantity mediated the relation between life events and memory variables.

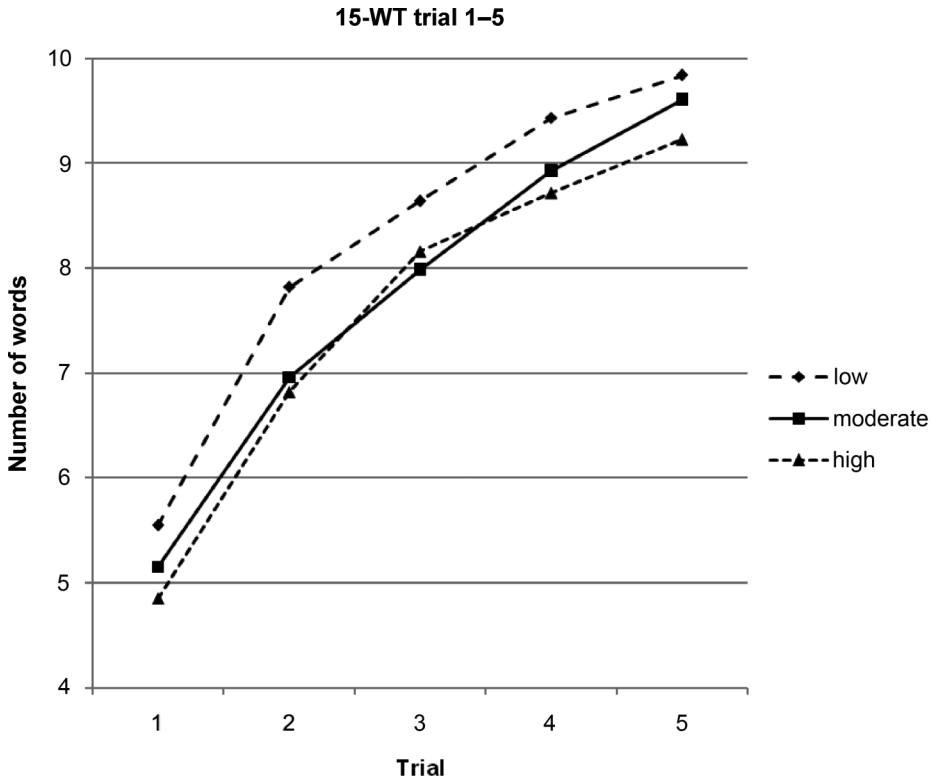
**Table 2** Demographics and Behavioral Characteristics.

	Total ( <i>N</i> = 252)		No events ( <i>n</i> = 56)		1–2 events ( <i>n</i> = 132)		≥ 3 events ( <i>n</i> = 64)		Group comparisons
	Mean ( <i>SD</i> )		Mean ( <i>SD</i> )		Mean ( <i>SD</i> )		Mean ( <i>SD</i> )		
Age	9.58(1.81)		9.54(2.07)		9.39(1.73)		9.95(1.70)		$F(2, 249) = 2.14, p = .120$
Gender (% male)	43.5%		48.2%		40.2%		46.9%		$\chi^2(2) = 1.40, p = .496$
Parental educational level	4.42(1.04)		4.35(.95)		4.51(1.12)		4.30(1.04)		$F(2, 232) = 1.00, p = .370$
WISC-III <sup>NL</sup> TIQ	103.31(13.31)		101.58(14.07)		104.59(13.19)		103.31(14.05)		$F(2, 249) = 1.25, p = .288$
WISC-III <sup>NL</sup> Vocabulary <sup>a</sup>	11.05(2.93)		10.84(2.84)		11.43(2.82)		10.11(2.87)		$F(2, 249) = 1.83, p = .162$
WISC-III <sup>NL</sup> Block Design <sup>a</sup>	10.17(3.25)		9.78(3.50)		10.33(3.08)		10.17(3.25)		$F(2, 249) = 0.59, p = .553$
CBCL total problems <sup>b</sup>	46.44(9.42)		46.82(9.95)		45.58(9.62)		47.95(9.42)		$F(2, 233) = 1.34, p = .264$
CBCL externalizing problems <sup>b</sup>	47.08(9.04)		48.06(10.66)		46.21(8.30)		48.06(9.06)		$F(2, 235) = 1.45, p = .290$
CBCL internalizing problems <sup>b</sup>	47.75(9.26)		45.58(8.92)		47.67(9.40)		49.61(9.00)		$F(2, 235) = 2.63, p = .075$
PTSD symptom severity <sup>c</sup>	8.17(6.99)		7.67(7.28)		8.28(7.17)		8.21(7.69)		$F(2, 133) = 0.06, p = .946$

<sup>a</sup>Scaled scores.<sup>b</sup>Standardized *T*-scores.<sup>c</sup>Means and standard deviations presented for cases reporting about an event > 1 month ago (*n* = 136), clinical cutoff level is a symptom severity > 15.**Table 3** Comparison of the Number of Correctly Recalled or Recognized Words on a Verbal Memory Task, Across Three Event Rate Groups.

15-WT	No events ( <i>n</i> = 56)		1–2 events ( <i>n</i> = 132)		≥ 3 events ( <i>n</i> = 64)		Group comparisons	$\eta^2$	Pairwise Comparisons <sup>a</sup>
	<i>M</i> ( <i>SD</i> )		<i>M</i> ( <i>SD</i> )		<i>M</i> ( <i>SD</i> )				
Total Learning	41.36(9.63)		38.82(9.67)		37.75(8.93)		$F(2, 229) = 9.71, p < .001$	.08	1 > 2*, 3***
Delayed Recall	9.30(2.61)		8.63(2.67)		8.42(2.24)		$F(2, 209) = 5.52, p < .01$	.05	1 > 2*, 3***
Recognition	29.07(1.28)		29.07(1.17)		28.93(1.36)		$F(2, 228) = 0.94, p = .391$	.008	NA

<sup>a</sup> Pairwise comparisons with Bonferroni correction.<sup>c</sup> Means and standard deviations presented for cases reporting about an event > 1 month ago (*n* = 136), clinical cutoff level is a symptom severity > 15.\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .



**Figure 2** Verbal memory learning curve for three stressful life event rate groups (low: no life events; moderate: 1–2 life events; high: three or more life events).

As for moderation by perceived parenting, no significant interaction effect was found. Additional analyses were performed to evaluate possible moderation by demographic variables. However, no significant moderation was found.

## DISCUSSION

This study aimed to evaluate the association between negative stressful life events and memory performance in children. The results confirm the hypothesis that the cumulative experience of negative stressful life events is associated with a decline in declarative memory performance, while an association with procedural memory performance was absent. The finding that performance decrements were specific to declarative memory and did not pertain to procedural memory suggests that the dominant underlying mechanism is a dysfunction of the hippocampus, presumably being the result of (a history of) elevated stress hormone levels. Physiological studies are needed to confirm this mechanism but are hampered because of the following reasons: (1) Elevated cortisol levels resulting from stressful life events are detected only around the time of the life event, because, in the vast majority, levels normalize with the passage of time (Shalev et al., 2008), and (b) the hippocampus may be volumetrically normal at the time of the initial stressful life event(s) but becomes smaller over the subsequent years compared with healthy controls

(Carrion, Weems, & Reiss, 2007). Although we do suggest that the mechanisms underlying the memory impairments are similar to those in PTSD, we emphasize that long-lasting dysregulations of the stress system, such as in PTSD, were probably rare in the current sample, considering the estimated 1.2% PTSD prevalence rate in the current sample. This rate is in line with previously reported PTSD prevalence of 1%–3% found in the general adult population (Breslau, 2009) and suggests that the current sample is a good representation of the general population.

Regardless of the mechanisms, the current findings show that the potential cumulative impact of negative stressful life events on declarative memory performance is of vital importance to daily life. Children who experienced three or more negative stressful life events recalled 9% less both in the short-term and the long-term phase of the verbal declarative memory task compared to children who did not experience any negative stressful life events. The impact of one or two life events on memory was less distinct but still significant. Declarative memory is of key importance to academic success (Catroppa & Anderson, 2007; Riding, Grimley, Dahraei, & Banner, 2003) and plays a central role in many cognitive functions that are important in daily life functioning, such as problem solving, reasoning, comprehending instructions, and decision making (Cantor & Engle, 1993).

Interestingly, the number of negative stressful life events was not related to non-verbal declarative memory. A similar dissociation between verbal and performatory memory performance (Yasik, Saigh, Oberfield, & Halamandaris, 2007) and verbal and performatory IQ (Saigh, Yasik, Oberfield, Halamandaris, & Bremner, 2006) has been shown previously in children with PTSD. This accords with suggestions that memory impairment in PTSD is modality specific with more pronounced impairments for verbal declarative memory than for visual declarative memory (Horner & Hamner, 2002). Previous suggestions that this disparity in effects on verbal and visual memory performance are due to differences in task characteristics (Jelinek et al., 2006) are untenable with regard to the current findings, since the design of the visual and verbal memory task is almost identical. In light of the extensive evidence that the hippocampus is involved in visuospatial processing and memory (Tsanov & Manahan-Vaughan, 2008), the current findings are hard to explain. The effects of stress on the hippocampus are more pronounced in the right hippocampus — which is related to visual memory — than in the left — which is related to verbal memory (Mitra, Sundlass, Parker, Schatzberg, & Lyons, 2006), which does not help either to explain the current and previous findings that impairments are more distinct in the verbal memory domain. As yet, no clear explanation exists why stressful life events seem to affect verbal memory performance more than visual memory. From an evolutionary perspective, it is certainly advantageous when visual memory in threatening situations is conserved after the stress response, because it facilitates the avoidance of similar future threatening situations. On the contrary, the evolutionary importance of memory for verbal information in threatening situations seems less clear.

The question rises that aspects of memory function are affected by life events. The current findings show that recognition of target words was unaffected, whereas learning and long-term memory function were deteriorated. As for the long-term memory impairments, the data revealed that, when controlling for the total number of words learned during the first five trials, the differences in delayed recall between the no-event and 1–2 event groups and no-event and  $\geq 3$  event groups disappeared. In sum, these findings indicate that maintenance and retrieval phases of memory function are spared, but that deficiencies are related to encoding and acquisition of new information. This is in line with recent

results that demonstrated that the memory problems in adult PTSD patients were confined to the encoding phase of the memory system (Johnsen & Asbjørnsen, 2009). Both the hippocampus and PFC are involved in the encoding (and retrieval) stage of memory processing (Long, Oztekin, & Badre, 2010). Thus, the fact that children with stressful life events demonstrated encoding problems does not reveal whether the memory decrements are due to hippocampus and/or PFC dysfunction. However, the findings of the procedural memory task suggest that PFC impairments were absent. The procedural learning task resembles the mirror-tracing task, which requires subjects to inhibit and reverse a highly overlearned association between vision and the hand and arm movements used in tracing. Therefore, performance on this task is particularly sensitive to deficits in higher order cognitive processes mediated by the frontal lobe (Kennedy & Raz, 2005). The conservation of procedural memory function in the current study implies that PFC impairments were absent; although imaging studies are needed to confirm that.

To what extent was memory dysfunction in the current study due to attention problems? The encoding problems, without retrieval problems, suggest that attention problems are involved in the stress-related memory impairments. The results showed that attention task performance and behavioral attention problems were indeed significantly associated with the memory performances. However, in spite of the attention influences on memory performance, the effect of life events on memory remained significant after correction for attention measures. This implies that if the PFC were partly involved in the memory decline after stressful events, it is primarily problems in the executive and organizational function of the PFC in the memory system, and not in the attentional contribution, that would play a role. That is to say, PFC dysfunction in children with stressful life events could affect organizational strategies during encoding, by diminished facilitation of the temporal order of information and by decreased reduction of proactive interference (Blumenfeld & Ranganath, 2007). Nonetheless, it is of note that different top-down and bottom-up attention mechanisms are part of the encoding stage in the memory system, in which different areas in the prefrontal cortex and parietal cortex are involved (Crespo-Garcia, Cantero, Pomyalov, Boccaletti, & Atienza, 2010). It is possible that the focused attention task and behavior checklist, which were used in the present study, were not sensitive to some attention mechanisms involved in the memory system. Furthermore, since memory problems were found in the verbal memory domain, use of an auditory attention task, instead of a visual attention task, could possibly have led to different results as for the involvement of attention mediation. Notwithstanding, behavioral attention problems and attention task performance were both significantly related to verbal and visual memory performance, which corroborates previous findings that attention is important for memory processes (Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001).

Sleep quality and quantity were not related to short- and long-term memory function, and life events did not correlate to sleep quality and quantity. Hence, sleep problems did not mediate the negative association between stressful events and memory performance. There is ample evidence that sleep plays an important role in memory consolidation (Diekelmann & Born, 2010). Possibly, the sleep measures used in this study are too global and therefore insensitive to the intricate aspects of sleep that are essential to memory consolidation processes.

Perceived parenting did not play a role as moderator in the relation between negative stressful life events and verbal declarative memory. Several studies have found a stress-buffering role for positive parenting in the behavioral adjustment in children and adolescents, protecting them against potential negative effects of stressful life events

(Skopp et al., 2007). For instance, a study conducted by Jouriles et al. (2008) found that higher levels of mothers' positive parenting were associated with a weaker relation between intimate partner physical violence and explicit memory functioning in preschoolers. However, this study was conducted with younger children and mother's positive parenting may have a larger effect on cognitive functioning when children are younger.

Individual differences in the vulnerability to the effects of stressful life events on memory form a challenging topic for further research. The interindividual variation in acute physiological and psychological response to stressful situations across children is large (Gunnar, Talge, & Herrera, 2009). Similarly, the subjective experience of negative stress with particular life events may differ considerably across children. In our view, the frequency of the life events is an important predictor of the cognitive outcome; however, it is the intensity of the child's emotional response that eventually determines whether the stress system exerts its effects on the parts of the brain that are important to memory function. Further research is needed to explore potential dispositions, such as the effectiveness of arousal regulation, that could contribute to resistance or vulnerability for effects of stressful life events on memory.

Strengths of this study are the relatively large sample size, the use of well-validated, standardized performance tasks and questionnaires, and an extensive evaluation of potential mediating and moderating factors such as attention, sleep, and perceived parenting. We found a weak, although positive significant, correlation between age and life events ( $r = .13$ ,  $p < .05$ ), which indicates that older children experienced more events than younger children, as may be expected of a questionnaire that asked for lifetime prevalence of life events. However, the lack of information about the actual neurophysiological levels of stress caused by these life events is a limitation of the study. Although our findings strongly suggest that the negative impact of stressful life events on memory is mediated by elevated levels of cortisol on a neurophysiological level, we can only speculate that these stressful life events were indeed accompanied with increased cortisol levels.

To conclude, the results of the present study provide support for a dimensional relation between negative stressful life events and verbal declarative memory in a nonclinical sample of school-aged children. Controlling for measures of attention and sleep minimized the possibility that the difference in verbal learning and memory was due to attentional dysfunction or sleep disturbances. Furthermore, perceived parenting did not influence the vulnerability for the effects of life events on memory impairment. The present results showed that children who did not experience any negative stressful life events recalled 9% more of the learned information than children who experienced three or more negative stressful life events. Such deficits in learning and memory can have broad developmental consequences for children, affecting both daily functioning and academic achievement (Catroppa & Anderson, 2007; Riding et al., 2003). The results of the present study may provide important information for clinical practice and future research concerning memory problems in children.

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