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Posttraumatic stress disorder in young children three years post-trauma: prevalence and longitudinal predictors

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Abstract

Objective—Age-appropriate criteria for post-traumatic stress disorder (PTSD) in young children have been established. The present study investigated the long-term course of such PTSD and its predictors in young children.

Methods—Young children (aged 2-10 years) and parents/caregivers who had attended Emergency Departments following motor vehicle collisions (MVCs) between May 2004 and November 2005 were assessed at two-to-four weeks and six months post-MVC; 71 families were re-interviewed three years post-MVC. Participants were assessed according to standard DSM-IV criteria for PTSD and a well-validated alternative algorithm for diagnosing PTSD in young children (PTSD-AA). Demographic, trauma-related and parental mental health variables and intellectual ability were also assessed at baseline.

Results—Using an 'optimal report' procedure (a positive diagnosis according to parent or child for older children, or just parent for younger children) 7.0% met criteria for DSM-IV PTSD and 16.9% for PTSD-AA at 3 years; using parent-report alone, these rates were 1.4% and 2.8%. Parent-child agreement for PTSD and PTSD-AA was no better than chance (Cohen's kappa = −.03 and −.04, respectively). Baseline parent posttraumatic stress relating to the child's trauma, and not trauma severity, was correlated with optimal report child PTSD-AA at each assessment (*r*s=.29-.31), and accounted for unique variance in logistic regression models of this outcome at each assessment.

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Conclusions—PTSD-AA in young children can persist for years but is under-recognized by parents, despite being shaped to a large extent by parents' own acute traumatic stress in response to the child's trauma.

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Introduction

Posttraumatic stress disorder (PTSD) is a common debilitating disorder in youth[1]. Until recently the reactions of pre-school and young children to trauma have received little attention[2]. The past decade has witnessed far greater coverage of the reactions of young children to traumatic stressors[3], a step change facilitated by the proposal of age-appropriate criteria for diagnosing PTSD. These make allowances for both the developmental limitations when assessing symptoms in this age group and the need to rely on parental/caregiver reporting[4]. Amending the number of avoidance symptoms required (from three to one) and dropping the requirement that a peri-traumatic affective response be observed yields a far greater proportion who meet the threshold for 'caseness' in young children[5,6]. Multiple studies have attested to the construct and predictive validity[7,8] of this age-appropriate alternative algorithm for diagnosing PTSD in young children (hereafter PTSD-AA), leading to the introduction of a preschool PTSD diagnosis within the DSM-5[9], based with minor amendments on the original PTSD-AA criteria. Both PTSD-AA and DSM-5 preschool PTSD do not require a peritraumatic affective response, but require one re-experiencing symptom, one avoidance symptom, and two hyperarousal symptoms, where clinically significant distress or impairment in relationships are present.

With the establishment of a reliable and valid diagnostic algorithm in younger children, it has been possible to turn to researching the prevalence, course, etiology and treatment of PTSD in this age group. Previously we examined the reactions of 2-10 year old children exposed to MVCs, and explored the course of DSM-IV-defined PTSD (hereafter just PTSD) and PTSD-AA from the acute post-trauma phase (i.e. the first four weeks) to a six-month follow up assessment[7]. Although the subsequent preschool PTSD criteria within DSM-5 specify an age range up to six years, at the time of this initial study it was important to examine the boundary conditions of the original PTSD-AA diagnosis up to the age of 10[4]. The present study presents data from a three-year follow up of this cohort, allowing consideration of several pertinent questions relating to younger children's responses to trauma.

First, the longer-term course of post-traumatic stress reactions in early childhood needs to be established. Adults report significant psychopathology related to traumatic experiences in early childhood[10]; however, many of these studies are based on retrospective accounts of multiple and repeated trauma exposure (e.g. abuse) in childhood typically accompanied by other adversities (e.g. neglect, parental mental illness). Long-term follow up studies of *single* traumas have addressed older children and adolescents and have found that PTSD may persist for years in a substantial minority[11].

Second, it is important to consider the validity of parental reports of their child's dysfunction. Reliance on parent reporting introduces tremendous scope for bias, in terms of under- and over-reporting of symptomatology.

Third, early diagnostic markers for predicting a long-term suboptimal response to traumatic stressors can be identified. Unique to this study is the ability to consider acute stress disorder (ASD), an early diagnostic marker found to have utility in adults and older youth[12,13].

Finally, it is essential to identify early demographic, trauma severity, and familial predictors of PTSD/PTSD-AA. These data would inform the early clinical management of PTSD and theoretical models of dysfunction in this age group[3]. A recent review[2] suggested that there have been inconsistent results observed for the relationship between demographic variables (i.e. age, gender) and PTSD in preschool and young children, but more substantive evidence for trauma severity and parent mental health as risk factors for PTSD. Apart from a study[14] that tracked preschoolers' PTSD symptoms in response to a missile attack, most research has been cross-sectional and has commonly addressed children exposed to interpersonal or domestic violence[2].

The study therefore addressed the following research questions:

1) What is the prevalence of PTSD/PTSD-AA in children three years after a MVC, and to what extent is this affected by age at the time of the trauma?

2) To what extent do children and their parents/caregivers agree in their assessment of the child's PTSD/PTSD-AA?

3) How well does ASD function as a predictor of PTSD/PTSD-AA at this three year follow up?

4) Which (if any) demographic, trauma severity, parental mental health and intellectual variables predict PTSD/PTSD-AA at each time point?

Participants

Participants were from a previously reported prospective study of young children aged 2-10 years at the time of study entry who attended an Emergency Department (ED) in South London between May 2004 and November 2005 following an MVC[7]. Exclusion criteria were the presence of moderate-to-severe intellectual disabilities, or moderate-to-severe traumatic brain injury. Of 114 child-parent dyads recruited for the initial two-to-four week post-trauma assessment (T1), 109 (95.6%) completed a six month follow up assessment (T2). At T2 permission was sought to re-contact families for a potential three year post-trauma assessment (T3); all families gave consent to being re-contacted.

Families who completed T1 and T2 (N = 109) assessments were re-approached. Thirty-three (30.3%) could not be contacted again and five (4.6%) were no longer willing to participate. Parents or caregivers (hereafter just "parents") of 71 children (62.2% of T1 sample, 65.1% of T2 sample) consented to the T3 interview. Interviews were conducted with the child's mother (n=64), father (n=2), grandmother (n=2), uncle (n=2) or aunt (n=1). In 53 cases the children themselves (if seven years or older) completed a structured interview for PTSD. The 18 children who did not complete the T3 interview assessment themselves were either still aged under seven (n=12), not allowed to do the interview by their parents (n=3), unwilling to participate (n=2) or withdrew during the interview (n=1).

The final T3 sample (age at MVC, M=6.5 years, SD=2.8; age at T3, M=10.3 years, SD=2.9), were assessed on average 3.8 years (SD=.4) post-MVC. Thirty-four (47.9%) were female, and 42 (59.2%) belonged to a minority ethnic group. Twenty-seven (38.0%) were pedestrians, 36 (50.7%) car passengers, four (5.6%) cyclists (who collided with a motor vehicle), and four (5.6%) bus passengers. Twenty (28.2%) participants had no injuries, 46 (64.8%) had only soft tissue injuries, and five (7.0%) had sustained a fracture. Four (5.6%) participants had lost consciousness and 11 (15.5%) were admitted to hospital. There were no significant differences between participants at T3 and those who only participated at the T1 and T2 assessments with respect to age, sex, ethnicity, MVC type, triage category, degree of injury, loss of consciousness, or hospital admission (all *p*s>.18). Furthermore, there were no differences in T2 parent- or child-reported PTSD symptom counts between those families that completed the T3 assessment and those that did not (t[106]=.36, p=.7 and t[42]=.87, p=.4, respectively).

Measures

Demographic and trauma-related variables—These data were gathered from the hospital ED records or parent interviews at T1. The presence of pre-MVC emotional or behavioral disorders was established in an interview with parents (endorsed by parents as present or absent).

Child PTSD—The study outcome measures were structured interviews of child PTSD/PTSD-AA, completed by parents, and children aged seven years or older. In addition to reporting parent- and child-report data, “combined report” diagnoses (i.e. from both parent and child responses, where *both* are available) and “optimal report” diagnoses (based on the maximal information available, i.e. parent-only responses for children under seven, or combined-report for children seven years or older) are presented. This is consistent with practice parameter recommendations that data from multiple informants be routinely obtained and its utility maximised[15]. While the use of the optimal report diagnosis increases the available sample size, the use of different assessment strategies in each age group may be viewed as methodologically problematic. In order to be transparent, the data for combined-report PTSD (i.e. for children aged seven years or older) are therefore also reported.

The PTSD Semi-Structured Interview and Observational Record for Infants and Young Children (IORYC)[4,6] was used to assess parent-report PTSD/PTSD-AA at each assessment point. The IORYC can yield a DSM-IV PTSD as well as PTSD-AA diagnosis. The IORYC possesses good inter-rater reliability[4,5] and construct validity[7].

At T1 and T2, child-report PTSD/PTSD-AA were assessed (for those aged seven years or older) using the Clinician Administered PTSD Scale - Child and Adolescent Version (CAPS-CA)[16]. The CAPS-CA is a child-report structured interview for assessing PTSD that possesses good psychometric properties[17]. For children aged seven years or older at T3, child-report PTSD/PTSD-AA were assessed using the PTSD schedule of the Anxiety Disorders Interview Schedule, child version (ADIS-C/P)[18]. The ADIS-C/P can be used to derive a PTSD-AA diagnosis (as PTSD-AA criteria are a sub-set of DSM-IV criteria). The ADIS-C/P was selected for use at T3 given its suitability for telephone administration.

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Continuous measures of PTSD severity were derived by counting endorsed symptoms; these data are reported in supplementary analyses (see Supplementary Tables 1-6).

Parental mental health—Parental post-traumatic stress symptomatology (PTSS) at T1 relating to the child's MVC was indexed using the Posttraumatic Diagnostic Scale (PDS)[19]. Parental depressive symptomatology at T1 was assessed using the Depression sub-scale of the Hospital Anxiety and Depression Scale (HADS)[20].

Child intellectual performance—To investigate whether posttraumatic stress was related to intellectual functioning, the British Picture Vocabulary Scale II (BPVS-II), a standardized test of receptive vocabulary[21], was administered at T1.

Procedure

The T3 assessment was approved by Bexley and Greenwich Research Ethics Committee (08/H0809/18). To facilitate participation in the T3 assessment, telephone interviewing was used.

Results

Prevalence of PTSD at three year follow-up (T3)

The prevalence of PTSD-AA and PTSD diagnoses at T3, differentiated by age group at MVC and informant, are displayed in Table 1. Parent-report diagnoses were only reported in one or two cases. Child- and combined-report or optimal report diagnoses were much more frequently endorsed (ranging from 7.0-20.8%).

In the case of child- and combined-report diagnoses, these rates were reduced relative to T1 and T2 (child-report ASD at T1, 22.9%; child-report PTSD at T2, 13.3%; child-report PTSD-AA at T1 and T2, 35.4% and 17.8%, respectively; combined-report ASD at T1, 29.2%; combined-report PTSD at T2, 18.8%; combined-report PTSD-AA at T1 and T2, 50.0% and 40.0%)[7]. The proportion of cases at T3 meeting criteria for PTSD-AA (16.9%) or PTSD (7.0%) based on an optimal report procedure also show improvement relative to earlier assessments (not previously reported; PTSD-AA prevalence was 28/113 [24.6%] at T1 and 24/108 [21.1%] at T2; ASD prevalence was 14/113 [12.3%] cases at T1 and PTSD prevalence was 9/107 [7.9%] cases at T2). PTSD-AA, regardless of informant, was observed at a rate more than twice as great as that observed for standard PTSD criteria, as found previously[7].

To investigate whether being a pre-school-aged child at the MVC influenced the risk of developing PTSD/PTSD-AA at T3, age-related comparisons were made. No age group-related differences were observed, i.e. children not old enough to self-report PTSD symptoms at T1/T2 (i.e. 2-6 year olds) were as likely to meet criteria for PTSD/PTSD-AA (regardless of informant) at T3 as those children who had provided self-report at T1/T2 (i.e. 7-10 year olds; Fisher's exact test, ps >.3); moreover, there were no differences in mean child-reported symptoms at T3 between children aged 2-6 years and children aged 7-10 years at T1 ($t[51]=.17$, ns).

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Since PTSD-AA can be diagnosed on the basis of fewer symptoms than PTSD, the increased prevalence of PTSD-AA may not be related to its developmental appropriateness but to its reduced symptom requirement. We therefore examined the mean number of symptoms for positive cases of each diagnosis at T3. There were no significant differences in symptom counts (although PTSD symptom counts were numerically higher) for diagnoses based on child report, combined parent-child report or optimal report (all ts<1.92, ns); parent-report comparison was not possible as only one child met criteria for parent-report PTSD. Moreover, with respect to child-report impairment severity there were no differences between child-report PTSD and PTSD-AA cases (t=.3, p>.6), or between optimal or combined report PTSD and PTSD-AA (optimal and combined report, t=.3, p>.6).

Parent-child agreement at T3

Parent-child agreement (Cohen's kappa) for the 53 children where data from both informants were available for PTSD was −.03 and for PTSD-AA was −.04; i.e. no better than chance. These figures did not differ even when restricting the calculations to older children who had been able to provide self-report at earlier assessments (−.06 and −.05, respectively; n=27).

The utility of baseline (T1) PTSD diagnoses in predicting PTSD at three year follow up (T3)

The sensitivity, specificity, positive predictive value (proportion diagnosed at T1 who retained the diagnosis at T3), and negative predictive value (proportion with no diagnosis at T1 who remained diagnosis free at T3) for each diagnosis and informant are reported in Table 2. The strong relationship between parent-reported T1 ASD and T3 PTSD is most likely a statistical artefact caused by the low numbers of positive cases for parent-report. The ability of T1 diagnoses to predict PTSD or PTSD-AA at three years (T3) was weak. For diagnoses that involved child-report (child-alone, combined report or optimal report), prediction statistics for T3 PTSD-AA were generally superior to those for T3 PTSD, notably when considering sensitivity and positive predictive values. The addition of parent-report within combined-report diagnosis did not improve predictive ability above that of child-report alone; indeed, specificity statistics for T3 PTSD-AA appeared to be weakened. Parent-report diagnoses (T1 ASD or PTSD-AA) were very insensitive predictors of corresponding child-report diagnoses at T3.

Of 11 cases who met criteria for combined-report PTSD-AA diagnosis at T3, only four had PTSD-AA at T1 based on parent-report; likewise, for the 12 cases meeting criteria for PTSD-AA at T3 based on optimal report, only four had PTSD-AA at T1 based on parent-report.

Correlates and predictors of optimal report PTSD-AA at each assessment

As the optimal report PTSD-AA diagnosis is most closely aligned with best diagnostic practice of utilizing multiple informants[15], this diagnosis was the dependent variable for correlational analyses (see Table 3) and regression modelling addressing possible risk factors for PTSS. Logistic regression was used to explore which variables accounted for unique variance in optimal report PTSD-AA. Only significant zero-order correlates were included in these models; a forward conditional method of entry was used. For optimal report PTSD-

AA at T1, age, sex, persistent injury and concurrent parental PTSS scores accounted for unique variance (χ^2=41.25, df=4, p<.0001; Nagelkerke R^2=.53). Ethnicity, parental presence during the collision, and parental depression at T1 were not retained in the model. For optimal report diagnoses at the 6 month assessment (T2), T1 persistent injury and T1 parental PTSS were retained in the model (χ^2=21.91, df=2, p<.0001; Nagelkerke R^2=.41), but not age, ethnicity, intellectual performance, triage, fracture, admission or post-MVC separation greater than one hour. For optimal report diagnoses at the 3 year assessment (T3), T1 parental PTSS was retained in the model (χ^2=6.30, df=1, p<.012; Nagelkerke R^2 = .16), but not post-MVC separation greater than one hour or parental depression. Parental PTSS at baseline (T1) was therefore the only variable to account for unique variance at each assessment point.

When initial optimal report diagnosis at T1 was also entered in the first step of the model for T2, only persistent injury was retained in the model with optimal report diagnosis at T1 also accounting for unique variance (χ^2=26.11, df=3, p<.0001; Nagelkerke R^2=.47). When initial optimal report diagnosis at T1 was also entered in the first step of the model for T3, only T1 parental PTSS was retained in the model, with T1 optimal report diagnosis not accounting for significant unique variance (χ^2=12.29, df=3, p<.007; Nagelkerke R^2=.30).

To allow closer investigation of predictive data, parent-report, child-report, and optimal report correlational data for symptom counts and the corresponding regression models are presented in supplementary data (Supplementary Tables 1-6); these are broadly consistent with findings presented here.

Discussion

This study addressed the assessment, prevalence, course and risk factors for DSM-IV PTSD and PTSD-AA in young children exposed to a MVC three years earlier. With respect to our first question (i.e. the prevalence of PTSD at 3 years), child- and combined-report data suggested that a significant minority continues to present with clinically significant PTSS, even at this long-term follow up assessment. Reliance on parent-report alone, however, yielded very few cases, regardless of diagnostic algorithm. The use of PTSD-AA at T3 doubled the prevalence of disorder (whether based on child-report, combined-report or optimal report) relative to PTSD at T3, consistent with data from T1 and T2 assessments[7]. The discrepancy between parent- and child-report was further borne out when addressing our second question, where parent-child agreement at T3 was no better than chance at the level of diagnosis.

With to respect to our third question (i.e. the ability of T1 diagnoses to predict T3 diagnoses), T1 PTSD-AA was more powerful at predicting the same diagnosis at T3 than T1 ASD at predicting PTSD at T3. Nevertheless, there seemed to be considerable natural recovery for PTSD-AA, as well as instances where children had PTSD at T3 (according to either diagnostic algorithm) but were not recognized as such during the T1 assessment. Parent report was either a weak predictor of diagnosis at T3 follow up or did not add to child-report alone.

In addressing our fourth question (i.e. risk factors for PTSD), parents' own PTSS at study entry (T1) accounted for unique variance in optimal report PTSD-AA at each assessment. Persistent injury at T1 accounted for unique variance in models of PTSD-AA at T1 and T2 (consistent with other studies showing a link between trauma severity and PTSD[2]) but not at T3, while female sex and age were only risk factors (i.e. accounted for unique variance) for PTSD-AA at T1. Intelligence was mostly not significantly related to PTSD.

These findings emphasize the importance of considering young children's long-term responses to traumatic stressors. As with earlier findings[6,14] young children may develop clinically significant PTSS that persists for years and that in many instances may not be recognized by their parents. The number of participants reporting PTSD-AA three years on that had not been recognized by parents at an earlier assessment suggests that, even with the accommodations made for diagnosing PTSD in this age group, the reliance on parent report may be inadequate for identifying PTSD in some young children. Pre-schoolers were as likely as elementary school-aged children to meet criteria for PTSD at T3. These findings also emphasize the importance of using a developmentally appropriate tool for diagnosing PTSD in children (e.g. DSM5 preschool child PTSD); the use of the PTSD-AA diagnosis more than doubled the number of cases identified by the DSM-IV PTSD diagnosis alone.

The present study adds to earlier work in this area by relating chronic reactions to acute responses, both in terms of children's own acute reactions and their parent's reactions. With respect to the early identification of children at risk of a chronic reaction to a traumatic stressor, diagnostic tools (particularly child-report PTSD-AA) have some predictive ability. However, the utility of such constructs should not be overstated; there is considerable natural recovery over time, as well as some cases who met diagnostic threshold at a three year follow up (T3) but who did not do so at previous assessments.

The current study strengthens the case for considering parental mental health in the aftermath of trauma, with parental acute PTSS at study entry consistently predicting their child's PTSS, even three years post-trauma; strikingly, this was despite parents being very unlikely to acknowledge clinically significant PTSS in their child at this assessment. The direction of this effect is unclear. Parents' own initial T1 PTSS could have been worsened by their children's T1 symptoms, children's responses across time points could have been shaped by their parents' initial reactions (e.g. modelling avoidance), or a bidirectional effect may have resulted in a mutual amplification of symptomatology[22].

The importance of familial factors in driving PTSD in this group was further underscored by the significant relationship between separation from parent during the trauma and PTSD in the first weeks following the trauma. The role of families in the development of chronic responses to traumatic stressors in young children still warrants further research, but these data speak to the need to consider trauma-exposed parent-child dyads from the outset, rather than young children in isolation. Providing support for parents in the aftermath of trauma (e.g. with psychoeducation about their *own*, as well as their child's, post-traumatic stress) is therefore indicated. With respect to other risk factors, the relationship between trauma severity indices and PTSD-AA was mainly limited to the six-month follow up, but is

supportive of a dose-response relationship for trauma exposure and PTSD risk in young children.

The study has several limitations. In particular, it is suffers from a relatively small sample size. Given the number of analyses undertaken (even if many were only exploratory), the chance of a type I error occurring was increased. Moreover, the power of the regression models was modest; larger samples may have revealed more predictor variables.

In conclusion, this study suggests that a significant minority of young children exposed to single event trauma may develop PTSD/PTSD-AA that persists for years. While parents' own PTSS in the acute phase may contribute to persistent PTSD in young children, the present study also suggests that parents may fail to observe persistent PTSD in their children when the trauma occurred in early childhood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Clinical Points

- There is limited data concerning the long-term course and etiology of PTSD in pre-school and young children.

- Using age-appropriate diagnostic criteria, PTSD persists in a minority of young children exposed to single-event traumatic stressors.

- Parental acute posttraumatic stress is a risk factor for acute and chronic child PTSD in young children.

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Table 1

Prevalence of PTSD and PTSD-AA by time point, informant and age group at time of MVC

		T1			T2			T3		
		2-6 at MVC	7-10 at MVC	Total sample	2-6 at MVC	7-10 at MVC	Total sample	2-6 at MVC	7-10 at MVC	Total sample
Informant	**Diagnosis** [a]	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**
Parent		(n=62)	(n=52)	(n=114)	(n=60)	(n=48)	(n=108)	(n=42)	(n=29)	(n=71)
	PTSD-AA	4 (6.5%)	9 (17.7%)	13 (11.5%)	6 (10.0%)	9 (18.8%)	15 (13.9%)	1 (2.4%)	1 (3.4%)	2 (2.8%)
	ASD/PTSD	1 (1.6%)	2 (3.9%)	3 (2.6%)	1 (1.7%)	1 (2.1%)	2 (1.9%)	0 (0.0%)	1 (3.4%)	1 (1.4%)
Child			(n=48)			(n=45)		(n=26)	(n=27)	(n=53)
	PTSD-AA	-	17 (35.4%)	-	-	8 (17.8%)	-	5 (19.2%)	4 (14.8%)	9 (17.0%)
	ASD/PTSD	-	11 (22.9%)	-	-	6 (13.3%)	-	2 (7.7%)	2 (7.4%)	4 (7.5%)
Combined-report			(n=48)			(n=45)		(n=26)	(n=27)	(n=53)
	PTSD-AA	-	24 (50.0%)	-	-	18 (40.0%)	-	6 (23.1%)	5 (18.5%)	11 (20.8%)
	ASD/PTSD	-	14 (29.2%)	-	-	9(18.8%)[b]	-	2 (7.7%)	3 (11.1%)	5 (9.4%)
Optimal report		(n=62)	(n=48)	(n=113)	(n=60)	(n=45)	(n=108)	(n=42)	(n=29)	(n=71)
	PTSD-AA	4 (6.5%)	24 (50.0%)	28 (24.6%)	6 (10.0%)	18 (40.0%)	24 (21.1%)	7 (16.7%)	5 (17.2%)	12 (16.9%)
	ASD/PTSD	1 (1.6%)	14 (29.2%)	14 (12.3%)	1 (1.7%)	9(18.8%)[b]	9 (7.9%)[c]	2 (4.8%)	3 (10.3%)	5 (7.0%)

[a]Note. ASD at T1, PTSD at T2 and T3;

[b]Total N = 48;

[c]Total N = 107.

MVC = motor vehicle collision; PTSD = posttraumatic stress disorder; PTSD-AA = PTSD, alternative algorithm; ASD = Acute stress disorder; Combined report = derived from both parent and child responses, where both are available; Optimal report = derived from parent-only responses for children under seven, or combined-report for children seven years or older.

Table 2

Predictive Value of T1 Diagnoses for T3 Diagnoses, by informant and diagnostic algorithm

T1 Diagnosis	T3 Diagnosis	Positive Predictive Value	Negative Predictive Value	Sensitivity	Specificity	Diagnoses Identified Correctly
Parent-report [a]						
PTSD-AA	PTSD-AA	12.5	98.4	50.0	89.9	88.7%
ASD	PTSD	50.0	100.0	100.0	98.6	98.6%
Child-report [b]						
PTSD-AA	PTSD-AA	33.3	94.4	75.0	73.9	74.1%
ASD	PTSD	0.0	89.5	0.0	68.0	63.0%
Combined-report [b]						
PTSD-AA	PTSD-AA	28.6	92.3	80.0	54.5	59.3%
ASD	PTSD	11.1	88.9	33.3	66.7	63.0%
Parent- to child-report [c]						
PTSD-AA	PTSD-AA	25.0	84.4	22.2	86.4	75.5%
ASD	PTSD	0.0	92.2	0.0	96.0	88.7%
Optimal report [d]						
PTSD-AA	PTSD-AA	35.3	88.9	50.0	81.4	76.1%
ASD	PTSD	10.0	93.4	20.0	86.4	81.7%

Note. T1 = 2-4 weeks post-trauma; T3 = three years post-trauma; PTSD = posttraumatic stress disorder; PTSD-AA = PTSD, alternative algorithm; ASD = Acute stress disorder; Combined report = derived from both parent and child responses, where both are available; Optimal report = derived from parent-only responses for children under seven, or combined-report for children seven years or older.

[a] n=71.

[b] n=27.

[c] i.e. Parent-report diagnoses at T1 as a predictor of child-report diagnoses at Time 3, n=53.

[d] n=71.

Table 3
Correlates and predictors of optimal report PTSD-AA diagnosis by assessment point

	T1 PTSD-AA		T2 PTSD-AA		T3 PTSD-AA	
	ρ/φ [a]	n	ρ/φ [a]	n	ρ/φ [a]	n
Prior symptomatology						
T1 PTSD-AA Diagnosis (optimal)			.51**	108	.28*	71
T2 PTSD-AA Diagnosis (optimal)					.10	71
T1 PTSD-AA Symptoms (optimal)			.35**	106	.15	69
T2 PTSD-AA Symptoms (optimal)					.22	71
Demographic & psychosocial variables						
Age	.43**	113	.23*	108	.01	71
Sex [b]	−.20*	113	−.09	108	−.02	71
Ethnicity [c]	−.19*	113	−.23*	108	−.07	71
No. life events in previous 6 mos.	−.04	112	.00	108	−.10	71
Prior trauma exposure	.03	112	.12	108	.03	71
Prior emotional problems	.11	111	.14	107	−.12	71
Prior behavioral problems	−.02	111	.00	107	.05	70
British Picture Vocabulary Scale (Second Edition)	−.05	81	−.23*	78	−.10	55
Trauma severity variables						
Triage category [d]	−.16	111	−.19*	106	.00	70
Fracture	.14	113	.26**	108	−.12	71
Admitted	.09	113	.30**	108	.01	71
Loss of consciousness	−.15	113	.13	108	−.11	71
Persistent injury at T1	.35**	113	.41**	108	.21	71
Parent variables						
With child during collision	−.21*	113	−.11	108	.00	71
Separation of one hour or more	.11	113	.24*	108	.25*	71
Posttraumatic Diagnostic Scale (T1)	.30**	88	.29**	87	.31*	59
Hospital Anxiety and Depression Scale, depression sub-scale (T1)	.30**	88	.15	87	.26*	59

[a] Note. Correlation coefficients represent Spearman's rho (where the predictor/correlate is continuous) or phi (where the predictor/correlate is dichotomous).

[b] Scored 0 for female, 1 for male.

[c] Scored 0 for minority ethnicity, 1 for white ethnicity.

[d] Triage scores ranged from 1-4, with lower scores indicating need for more urgent treatment.

* p<.05;

** p<.01; T1 = 2-4 weeks post-trauma; T2 = six months post-trauma; T3 = three years post-trauma; PTSD-AA = PTSD, alternative algorithm.