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Abstract. This study aimed to assess the symptomatic associations between attention-deficit/hyperactivity disorder (ADHD) symptomatology, trauma exposure, and posttraumatic stress disorder (PTSD) symptomatology. The sample consisted of 16,340 children from a population-based Swedish twin study. Trauma exposure, PTSD, and ADHD symptomatology was parent-rated in a structured telephone interview. Trauma exposure and subsequent PTSD was positively associated with ADHD prevalence. ADHD in trauma-exposed individuals was more severe than in non-trauma-exposed individuals. Individuals with PTSD and ADHD displayed an elevated ADHD symptomatology compared to trauma-exposed individuals without PTSD. In comorbid ADHD and PTSD, the order of trauma and onset of ADHD did not impact ADHD severity. The study confirms the association between ADHD, trauma exposure, and PTSD and suggests that trauma-exposed children are at risk for more severe ADHD.

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder that is characterized by deficiencies in executive control, emotional regulation, delay aversion, impaired working memory, motivational problems, and diminished persistence of effort (Roberts, Milich & Barkley, 2015; Willcutt, 2015; American Psychiatric Association, 2013). Willcutt (2012) has estimated the ADHD prevalence to be 11.4 % in 6-12 year olds, based on a meta-analytic review of 24 studies including 56,088 6-12 year-olds. A slightly lower prevalence of 5-6 % in the age group 6-11 was found in a worldwide metaregression analysis by Polanczyk et al. (2007) and a similar lower prevalence of 5.2 % was found in a study of Norwegian 7-9 year-olds (Ullebø, Posserud, Heiervang, Obel & Gillberg, 2012). ADHD is a highly heritable condition, with genetic factors explaining 60 – 95 % of the variation in the traits comprising the disorder (Barkley, 2015; Larsson et al., 2013).

A growing body of research indicates that individuals with ADHD, when compared to individuals without ADHD, display a higher prevalence of experiences of physical abuse (Fuller-Thomson, Metha & Valeo, 2014), sexual abuse (Sonnby, Åslund, Leppert & Nilsson, 2011; Briscoe-Smith & Hinshaw, 2006), and maltreatment (Capusan et al., 2016; Ouyang et al., 2008), and an elevated prevalence of posttraumatic stress disorder (PTSD) (e.g., Antshel et al. 2013; Husain, Allwood & Bell, 2008; Kessler, 2006). When examining seven studies that compared rates of PTSD in children with and without ADHD, Spencer et al. (2015) found that six of them showed a significantly increased rate of PTSD in children with ADHD. The meta-analysis also reviewed articles that examined the reversed risk (i.e., the rates of ADHD in children with and without PTSD). Out of ten studies, six showed significantly higher rates of ADHD in children with PTSD.

The relation between childhood adversity, PTSD, and ADHD

Several hypotheses have been put forth to explain the associations between adversity, PTSD and ADHD. Six of these hypotheses are described below.

The first hypothesis is that the association between childhood adversity and ADHD could be explained by the fact that trauma exposure might lead to symptoms that are similar to ADHD symptoms. Childhood maltreatment has been found to affect working memory, executive functions, and emotional control (Gould et al., 2012), as well as inhibitory network connectivity and response inhibition (Elton et al., 2014). Maltreatment-related PTSD has furthermore been associated with poorer performance on measures of attention, abstract reasoning, and executive function in children (Beers & De Bellis, 2002). These are all neurocognitive factors associated with ADHD (Capusan et al., 2016). Some studies (Elton et al., 2014; Danese & McEwen, 2012) thus suggest that childhood maltreatment may alter the functional neurodevelopment of inhibitory behavioral control.

However, the hypothesis that the association between adversity and ADHD could be explained by trauma exposure leading to ADHD-like symptoms has been criticized (Barkley, 2015). In the light of twin studies that show negligible, if any, contributions of the shared environment to the expression of symptoms of ADHD (Nikolas & Burt, 2010; Burt, Larsson, Lichtenstein & Klump, 2012), a second hypothesis has been put forth. This hypothesis claims that the associations between an abusive home environment and ADHD could in fact be derived from the genetic contribution to the family environment (in form of an increased risk for ADHD in parents to children with ADHD). The genetic contribution may thus account for the largest degree of risk for ADHD and for a broader spectrum of psychopathology in traumatized or abused children (Barkley, 2015). In an attempt to rule out the potential effect of genetic and environmental confounding, a large twin study of 18,168 adult twins in the Swedish Twin Registry (Capusan et al., 2016) was conducted. Capusan et al. (2016) reported that the association between childhood maltreatment and adult ADHD symptoms were partly due to familial confounding (meaning that environmental or genetic factors partly confounded the association), but was also consistent with a causal interpretation with a small effect. However, another twin study of two large population-representative birth cohorts by Danese et al. (2016) suggests that the association between childhood violence victimization and later cognition was largely non-causal. This study suggests that the difference in cognitive deficits between victims and non-victims could be largely explained by cognitive deficits that predated the childhood victimization and was confounded by genetic and environmental risks. Dinkler et al. (2017) examined the effect of familial confounding on the association between childhood maltreatment and neurodevelopmental disorders in a co-twin control analysis, showing that childhood maltreatment was not associated with an increased amount of neurodevelopmental disorders when genetic and shared environmental factors were taken into account. None the less, when comparing co-twins who were discordant regarding childhood maltreatment, a small increase in ADHD symptoms was found in twins with experiences of childhood maltreatment.

A third hypothesis describes an alternative causal pathway between ADHD and trauma exposure, suggesting that ADHD in itself might be a risk factor for trauma exposure. Children with ADHD may put themselves in more high-risk situations than comparable children without ADHD, and thus be more vulnerable for victimization,

conflict, and maltreatment (Husain et al., 2008; Ford et al., 2000). For example, several studies have found evidence for an increased risk for automobile accidents in individuals with ADHD compared to controls (Barkley, 2004; Barkley, Guevremont, Anastopoulos, Dupaul & Shelton, 1993). The empirical support for this causal pathway in children has been somewhat inconsistent and Wozniak et al. (1999) found little meaningful differences between ADHD and control children with respect to the rate of trauma exposure. However, Wozniak et al. (1999) did find that children with ADHD and comorbid bipolar disorder had an increased risk for trauma exposure. Ford et al. (2000) found that children with ADHD had a somewhat increased rate of previous exposure to maltreatment trauma, but less so than children diagnosed with oppositional defiant disorder (ODD). Similarly, a longitudinal study by Koenen et al. (2005) found that individuals who had had problems with hyperactivity, antisocial behavior, and difficult temperament during their childhood were 50 % more likely to experience a trauma compared to individuals without such history. Altogether, individuals with ADHD, and particularly individuals with ADHD and coexisting bipolar disorder, ODD, or antisocial behavior, seem to have an increased risk for trauma exposure, but whether the association between ADHD and trauma can be fully explained by this increased risk of trauma exposure remains unclear.

In addition to a potentially increased risk for trauma exposure in individuals with ADHD, some research indicates a fourth hypothesis for the association between ADHD and PTSD. This hypothesis claims that trauma-exposed individuals with ADHD might suffer an elevated risk to develop PTSD after trauma compared to trauma-exposed individuals without ADHD (Spencer et al., 2017; Adler, Kunz, Chua, Rotrosen & Resnick, 2004). Wozniak et al. (1999) examined the prevalence of PTSD in trauma-exposed children with and without ADHD and found no significant difference in regard to risk for subsequent PTSD. However, ADHD has been found to be highly comorbid with many psychiatric conditions such as mood disorders, anxiety disorders, and substance use disorders (Kessler et al., 2006), which could indicate that ADHD might act as a general vulnerability to psychopathology, including a vulnerability to PTSD. This hypothesis was supported by Spencer et al. (2017), who found signs of dysfunctional activation in brain structures that mediate fear extinction learning in non-traumatized adults with ADHD. This abnormal fear circuitry associated with ADHD may thus predispose individuals with ADHD to psychiatric disorders characterized by abnormalities in fear extinction and recall, such as PTSD (Spencer et al., 2017).

The fact that individuals with ADHD might have a vulnerability to develop PTSD after trauma exposure could indicate that there is a common susceptibility behind both disorders. This could be described as a fifth hypothesis for the strong association between ADHD and PTSD. Candidates for that kind of shared vulnerability for ADHD and PTSD are temperament or personality traits such as neuroticism, negative emotionality, or low constraint, which could increase the general risk for psychopathology (Harrington et al., 2012). These traits have been associated with a higher risk for ADHD (Martel & Nigg, 2006) as well as PTSD (Miller, 2003). Research on the two disorders further indicates that noradrenergic dysregulation could be another example of a shared vulnerability factor (Husain et al., 2008).

Since ADHD and PTSD share several salient symptoms (e.g., poor concentration, hyperarousal, irritability, and restlessness), a sixth hypothesis is that the diagnostic overlap between the disorders could reflect symptom similarity rather than conceptual or etiological relationships (Husain, et al., 2000; Ford et al., 2000;

Weinstein, Staffelbach & Biaggio, 2000). Symptom similarities might be particularly perceptible with more general symptom descriptions (e.g., difficulty concentrating). PTSD symptoms might thus mimic ADHD symptoms, which could increase the potential for misdiagnosis and, by extension, inappropriate or inadequate treatment (Weinstein et al., 2000). This hypothesis was supported by a study by Glod and Teicher (1996), who found that abused children had significantly higher activity levels than non-abused children. However, the idea that ADHD symptoms among PTSD patients can be fully explained by PTSD symptoms mimicking those of ADHD has been challenged by Biederman et al. (2013). In order to test this idea, Biederman et al. compared the rate of ADHD in relatives to children with ADHD and PTSD to the rate of ADHD in relatives to children with ADHD without PTSD. The results showed that the relatives of children with comorbid ADHD and PTSD had a similar rate of ADHD to relatives of children with ADHD without comorbid PTSD. This suggests that the ADHD symptoms in children with ADHD and PTSD cannot be fully accounted for by ADHD-like PTSD symptoms, since ADHD-like PTSD symptoms would not be associated with elevated rates of ADHD in the child's relatives.

The nature of ADHD with comorbid PTSD

Since a considerable part of individuals with ADHD have a history of trauma exposure and maltreatment, as well as comorbid PTSD, some studies have examined the nature of comorbid ADHD and PTSD compared to ADHD without PTSD. Ford et al. (2000) has hypothesised that PTSD symptoms of intrusive re-experiencing and hyperarousal may worsen ADHD symptoms of impulse control and attentional focusing and that PTSD symptoms of avoidance and emotional numbing may contribute to ADHD's motivational or social problem-solving deficits. Antshel, Biederman, Spencer, and Faraone (2016) examined the neuropsychological profile of comorbid PTSD in adult ADHD and found that the comorbidity with PTSD was associated with weaker cognitive performance on several tasks that appear related to spatial/perceptual abilities and fluency, compared to adults with ADHD without comorbid PTSD. Biederman et al. (2013) examined children with ADHD and PTSD compared to children with ADHD without PTSD across multiple domains of functioning. The findings indicated no differences in regard to the clinical features of ADHD (e.g., prevalence of individual ADHD symptoms). However, comorbid ADHD and PTSD was associated with greater clinical severity in regard to other psychiatric comorbidities, psychosocial dysfunction, poorer social functioning, risk of psychiatric hospitalization, and school impairment. Ansthel et al. (2013) found similar results in adults, indicating no differences in regard to mean number of ADHD symptoms but greater severity in terms of psychiatric comorbidity and psychosocial functioning in individuals with comorbid ADHD and PTSD compared to individuals with ADHD without PTSD.

Aim and research questions

In spite of a large body of research on trauma, PTSD, and ADHD, several questions remain unanswered. No studies have examined the covariance of trauma exposure, PTSD symptomatology, and ADHD symptomatology in Swedish children

using a large, population-based sample. In studies comparing individuals with comorbid ADHD and PTSD with individuals with ADHD without PTSD, the samples have often been small ($N = 271$ in Biederman et al., 2013; $N = 190$ in Antshel et al., 2013), which could reduce the generalizability of the results. One aspect of the comorbidity of ADHD and PTSD that has not been studied is the effect of temporality regarding trauma exposure and onset of ADHD symptoms. By comparing individuals with an onset of ADHD after the trauma exposure with individuals with an onset of ADHD that predates the trauma exposure, a new light might be shed on the complex and multifaceted association between trauma exposure and ADHD symptomatology.

The overall aim of this study was to assess the symptomatic associations between ADHD symptomatology, trauma exposure, and PTSD symptomatology. The specific research questions of this study were: 1) Is trauma exposure associated with the prevalence of ADHD?, 2) Is PTSD associated with the prevalence of ADHD?, 3) Does ADHD in non-trauma-exposed individuals differ on aspects of ADHD from ADHD in trauma-exposed individuals with and without subsequent PTSD?, and 4) Does ADHD with comorbid PTSD differ on aspects of ADHD based on whether the onset of ADHD predates or postdates the trauma exposure?

Methods

Subjects

The subjects in this study consisted of participants in the Child and Adolescent Twin Study in Sweden (CATSS). In the CATSS study, all Swedish 9-year-old twins are identified on-going through the Swedish Twin Registry. The parents are contacted and asked if they wish to participate in a structured telephone interview on somatic and mental health (Anckarsäter et al., 2011). Interviewers from a professional interview company, "Intervjubolaget", carry out the telephone interviews using a computerized version of the interview. The overall response rate in the CATSS study as of 2010 was 80 % (Anckarsäter et al., 2011). Anckarsäter et al. (2011) performed systematic analyses regarding differences between non-responders and responders, on the basis of an anonymized merge between the CATSS database and official files such as the National Board of Health and Welfare database on socio-economic circumstances, in- and outpatient diagnostics, and pharmacological treatment. Non-responders were slightly more likely to be diagnosed with ADHD and belong to a lower socio-economic stratum. In an analysis of the ratio of mothers, fathers, and other guardians of the children amongst the CATSS telephone interview responders as of 2010, Anckarsäter et al. (2011) found that mothers answered 87.5 % of the questionnaires, fathers 12.2 % %, and other guardians answered 0.3 %. Kerekes et al. (2013) found no significant differences between mothers', fathers', and guardians' ratings on The Autism-Tics, AD/HD, and other Comorbidities (A-TAC) inventory as of 2010.

Data for the present study were drawn from the database in March 2016, when telephone interview data was available for 22,332 9-year-olds born between the 1 of June 1995 and the 1 of February 2007. The interviews were conducted between 2004 and 2016. Due to a change of questionnaires in 2006, individuals interviewed before 2006 ($n = 5,992$) were excluded. The final study population thus consisted of $N = $

16,340 children with a close to equal sex distribution (50.5 % were boys, and 49.5 % were girls).

Measures

Assessment of ADHD symptomatology. ADHD symptomatology was assessed with the Autism-Tics, AD/HD, and other Comorbidities (A-TAC) inventory**.** The A-TAC is a parental telephone interview designed for large-scale epidemiological research and covers a broad range of neurodevelopmental disorders (Hansson et al., 2005). The inventory consists of 96 questions divided into domains (e.g., ADHD and autism spectrum disorder), worded to reflect DSM-IV criteria and clinical features. Questions are answered in a lifetime perspective with the response categories "No" = 0, "Yes, to some extent" = 0.5, and "Yes" = 1. The score of the single items are added together to form sum scores measuring the resemblance to clinical diagnoses. The ADHD domain of A-TAC consists of 19 items and can be divided into two modules, 1) Concentration and attention (nine items) and 2) Impulsivity and activity (ten items). Several clinical validation studies (Hansson et al., 2005; Larson et al., 2010, Larson et al., 2013) have reported that the ADHD domain of A-TAC has an excellent cross-sectional validity in identifying ADHD, with an area under the receiving operating characteristic curve (AUC) of 0.94. Cronbach's alpha for the ADHD score has previously been established to be 0.90 (Anckarsäter et al., 2011).

As a proxy for ADHD diagnosis, a cut off of ≥ 6.0 out of a total of 19.0 on the ADHD domain of A-TAC was utilized. This cut off has been recommended as a broad screening cut off with a sensitivity/specificity of 0.91/0.73 in clinical and community samples (Larson et al., 2010) and a sensitivity/specificity of 0.64/0.78 when compared to a comprehensive clinical follow-up three years later (Larson et al., 2013).

Assessment of trauma exposure. Trauma exposure was assessed with a questionnaire consisting of binary questions targeting whether the child had ever experienced any of 11 potentially traumatic events (see Table 1).

Table 1.

The 11 potentially traumatic events included in the assessment of trauma exposure.

1) A serious car accident
2) Any other serious accident
3) Emotional abuse/neglect
4) Physical neglect
5) Physical abuse
6) Sexual abuse in form of touching
7) Sexual abuse in form of rape
8) Witnessing violence between family members
9) Witnessing a threatening or violent criminal incident
10) Witnessing any other serious incident
11) Exposure to any other cruel or terrifying event

The five items on sexual abuse and emotional or physical abuse/neglect were rephrased versions of items in the Life Stressor Checklist-Revised (LSC-R), a 30-item adult self-report screening of traumatic life events (Wolfe & Kimerling, 1997). The remaining six items where included in order to target common traumatic events in

children. If a traumatic event was reported, the parent was asked follow-up questions regarding the child's age at the event and, when applicable, the frequency at which the event happened, the child's age when the event happened the last time and the relationship with the perpetrator.

Trauma exposure was defined as a parental report of at least one defined potentially traumatic event. The last two items (whether the child had witnessed or been exposed to any other serious incident or any other cruel or terrifying event) was not considered as trauma exposure. This was due to the large variety of severity in the reported events (when reviewing some of the open answers it was concluded that most could not be considered sufficiently traumatic, e.g., the death of a pet or watching a disturbing video online). Age at trauma was defined as the child's age at the event or the age when the event happened the first time. For individuals reporting multiple traumas, age at trauma was defined as the age at the first trauma.

Assessment of PTSD symptomatology. If a traumatic experience was reported, the parent was asked follow-up questions on PTSD symptoms in the child. These symptoms were assessed with a revised and shortened 10-item version of the PTSD Checklist for DSM-IV, civilian version (PCL-C), developed by Weathers, Litz, Herman, Huska, and Keane (1993). The original PCL-C is a 17-item adult self-report rating scale assessing the 17 DSM-IV symptoms of PTSD. The full version of the PLC-C has shown good psychometric properties (Bliese et al., 2008; Lang & Stein, 2005, Freedy et al., 2010). The revised and shortened version used in this study included 10 of the 17 items in the PCL-C, rephrased from self-reporting questions into parent-reporting questions and with response options ranging between "No" = 0, "Yes, to some extent", = 0.5, and "Yes" = 1. The checklist included 5 intrusion symptoms and 5 symptoms of increased arousal, representing the B (intrusion symptoms) and D (symptoms of increased arousal) criteria for PTSD in DSM-IV (American Psychiatric Association, 2013). The full 17-item PCL-C includes 7 additional items assessing the prevalence of avoidance symptoms (C criterion for PTSD in DSM-IV). Several studies have confirmed the use of other forms of abbreviated versions of the PCL-C. Bliese et al. (2008) has shown that that 4 of the 17 items display high information relative to the other items and that those 4 items alone shows good psychometric properties. In a similar way, Lang & Stein (2005) has shown good psychometric properties for a 6-item version of the PCL-C. However, neither all items in the 4-item version (Bliese et al., 2008), nor all items in the 6-item version (Lang & Stein, 2005) were included in the 10-item version used in this study.

As a proxy for PTSD diagnosis, a cut off of at least 1 of 5 intrusion symptoms and at least 1 of 5 symptoms of increased arousal was used. Since the questionnaire didn't fully mirror all PTSD criteria in DSM-IV, one symptom from each of the examined criteria was assumed to result in a fair balance between sensitivity and specificity.

Analytical procedures

Definitions of groups. In order to answer the four research questions, the subjects were compared with each other in different constellations and groups.

First, the association between trauma exposure and ADHD was examined through a comparison between trauma-exposed and non-trauma-exposed individuals regarding prevalence of ADHD.

Second, the association between subsequent PTSD and ADHD was examined by dividing the trauma-exposed individuals into individuals with and without subsequent PTSD. These two groups were thereafter compared regarding prevalence of ADHD.

Third, the individuals with ADHD were divided into three groups in order to assess whether ADHD in non-trauma-exposed individuals differs from ADHD in trauma-exposed individuals with and without subsequent PTSD. The three groups were; 1) Individuals displaying ADHD without trauma exposure, hereafter called *ADHD only*, 2) Individuals displaying ADHD who were trauma-exposed without subsequent PTSD, hereafter called *ADHD+trauma,* and 3) Individuals displaying ADHD who were trauma-exposed with subsequent PTSD, hereafter called *ADHD+PTSD* (see Figure 1). Individuals who had missing data on trauma exposure or PTSD symptoms were excluded in the analyses comparing *ADHD only, ADHD+trauma,* and *ADHD+PTSD*.
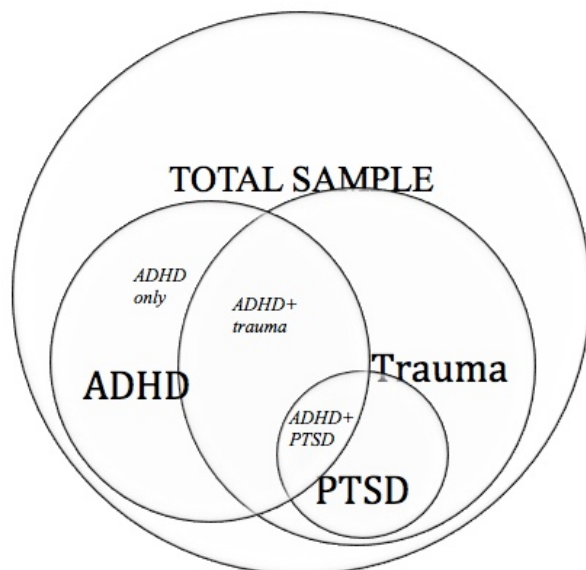
ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.
ADHD only = Individuals displaying ADHD without trauma exposure.
ADHD+trauma = Individuals displaying ADHD who were trauma-exposed without subsequent PTSD.
ADHD+PTSD = Individuals displaying ADHD who were trauma-exposed with subsequent PTSD.

Figure 1. An explanatory diagram for the three ADHD groups (*ADHD only, ADHD+trauma* and *ADHD+PTSD*). Group names in italics. The sizes of the circles do not correspond to the exact sizes of the groups in the sample.

Fourth, the individuals in the *ADHD+PTSD* group were further divided into two groups in order to assess whether ADHD with comorbid PTSD differs on aspects of ADHD based on whether the onset of ADHD predates or postdates the trauma

exposure. The two groups were 1) Individuals with ADHD and PTSD with an age at onset of ADHD before the age at trauma, hereafter called *ADHD before trauma,* and 2) individuals with ADHD and PTSD with an age at onset of ADHD after the age at trauma, hereafter called *ADHD after trauma*. Individuals who displayed an age at onset of ADHD the same year as the age at trauma or had missing data were excluded in the analyses comparing *ADHD before trauma* and *ADHD after trauma*.

      Statistical methods. Non-parametric tests were used throughout the study, based on the skewed distribution of the dependent variables and as a way to minimize the effects of the interdependence in the sample owing to the correlation between twins. Median comparisons were made using Kruskal-Wallis or Mann-Whitney U tests, depending on the number of groups. When using the Kruskal-Wallis test, the Mann-Whitney U test was used for post hoc analyses. Distributions of categorical variables were compared with Pearson's χ^2 test. Between group effect sizes were calculated and the operational definitions of effect sizes for ϕ and r defined by Cohen (1988, p. 25 and p. 79) were used, where 0.1 is consider as a small, 0.3 as a medium, and 0.5 as a large effect size. All tests were two-tailed and alpha was set at .001. All analyses were conducted using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY).

Ethical considerations

      The CATSS study was designed in accordance with the Helsinki declaration and approved by the ethical review board of Karolinska Institutet (Dnr: 02-289). All participants consented to the study after receiving written and oral information. All analyses were performed using anonymized data files.

Results

Association between trauma exposure and ADHD prevalence

      Trauma exposure was associated with a higher prevalence of ADHD, as presented in Table 2. However, the between group effect size was small.

Table 2.

Prevalence of ADHD in individuals with and without trauma exposure.

	Trauma-exposed $n = 4,280$	Non-trauma-exposed $n = 12,021$	Test statistics	P-value	Effect size
Displaying ADHD n (% of group)	717 (16.8 %)	1,162 (9.7 %)	$\chi^2(1) = 155.40$	< .001	$\phi = .098$

ADHD = Attention-deficit/hyperactivity disorder.

Association between PTSD and ADHD prevalence

When further examining the trauma-exposed individuals, subsequent PTSD was significantly associated with higher prevalence of ADHD with a small between group effect size, as presented in Table 3.

Table 3.

Prevalence of ADHD in trauma-exposed individuals with and without subsequent PTSD.

	Trauma-exposed with subsequent PTSD $n = 389$	Trauma-exposed without subsequent PTSD $n = 3,891$	Test statistics	P-value	Effect size
Displaying ADHD n (% of group)	133 (34.2 %)	584 (15.0 %)	$\chi^2(1) = 93.30$	$< .001$	$\phi = .15$

ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.

ADHD in non-trauma-exposed individuals compared to ADHD in trauma-exposed individuals with and without PTSD

The three ADHD groups *ADHD only, ADHD+trauma* and *ADHD+PTSD* (see Figure 1) were compared on several aspects of ADHD, as presented in Table 4. The age at onset of ADHD did not differ significantly between the three ADHD groups. When compared on total A-TAC ADHD score (indicating ADHD severity), the three ADHD groups were found to differ significantly. Post hoc analyses revealed that the *ADHD only* group displayed a significantly lower mean score compared to the *ADHD+trauma* group, which in turn displayed a significantly lower mean score than the *ADHD+PTSD* group. The between group effect sizes were small. The three ADHD groups were thereafter compared on the two modules of the ADHD domain of A-TAC: 1) concentration and attention and 2) impulsiveness and activity, as presented in Table 4. The three groups differed significantly on the concentration and attention module as well as on the impulsiveness and activity module and post hoc analyses mirrored to a large degree the findings from the total A-TAC score analyses. However, the *ADHD only* group did not differ significantly from the *ADHD+trauma* group regarding mean score on the impulsiveness and activity module. All significant differences resulted in small between group effect sizes.

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Table 4.

Medians, mean values and standard deviations on aspects of ADHD for the three ADHD groups.
Significant differences on post hoc analyses are marked with † and ‡ (see legend).

	ADHD only $n = 1,162$	ADHD+trauma $n = 584$	ADHD+PTSD $n = 133$	Test statistics	P-value
Age at onset of ADHD	$Mdn = 5.5$ $M = 5.04 \pm 2.00$	$Mdn = 5.0$ $M = 4.75 \pm 2.04$	$Mdn = 5.0$ $M = 4.80 \pm 1.94$	$H(2) = 6.74$	.034
Total A-TAC ADHD score	$Mdn = 8.5$ $M = 9.18 \pm 2.97$	$Mdn = 9.0$ $M = 9.90 \pm 3.40$ †	$Mdn = 11.5$ $M = 11.82 \pm 3.69$ †, ‡	$H(2) = 72.47$	< .001
A-TAC score on the concentration and attention module	$Mdn = 4.5$ $M = 4.73 \pm 2.05$	$Mdn = 5.0$ $M = 5.12 \pm 2.11$ †,	$Mdn = 6.0$ $M = 5.86 \pm 2.09$ †, ‡,	$H(2) = 40.23$	< .001
A-TAC score on the impulsivity and activity module	$Mdn = 4.0$ $M = 4.45 \pm 2.21$	$Mdn = 4.5$ $M = 4.78 \pm 2.40$	$Mdn = 6.0$ $M = 5.95 \pm 2.65$ †, ‡,	$H(2) = 40.97$	< .001

† = Significant difference on post hoc comparison with the *ADHD only* group.
‡ = Significant difference on post hoc comparison with the *ADHD+trauma* group.
A-TAC = The Autism-Tics, AD/HD, and other Comorbidities inventory.
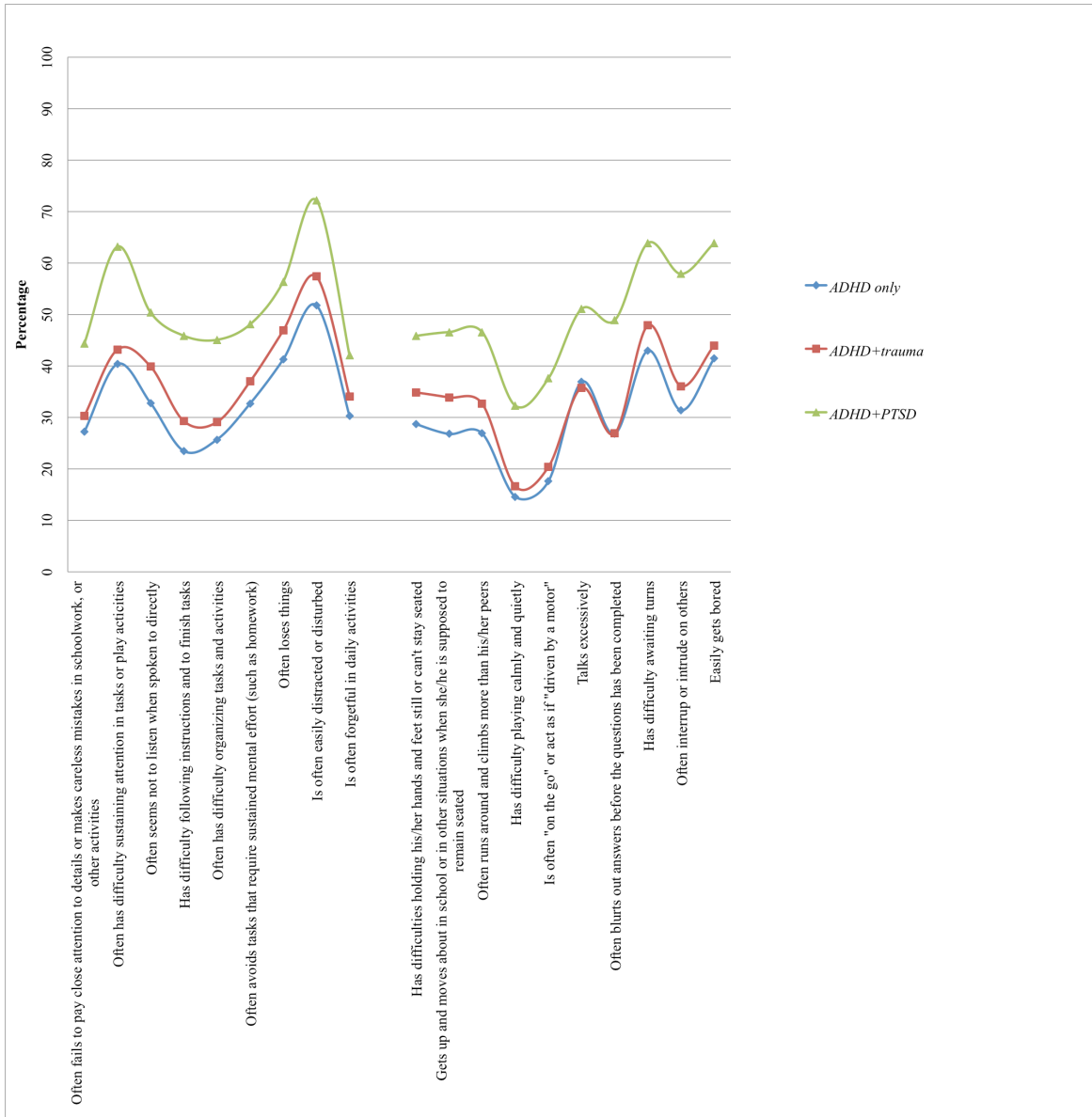ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.
ADHD only = Individuals displaying ADHD without trauma exposure.
ADHD+trauma = Individuals displaying ADHD who were trauma-exposed without subsequent PTSD.
ADHD+PTSD = Individuals displaying ADHD who were trauma-exposed with subsequent PTSD.

In order to further analyse patterns of confirmed items on the ADHD domain of the A-TAC, the three ADHD groups were compared regarding percentage of individuals who confirmed each of the 19 A-TAC ADHD items. The percentages of individuals who confirmed each item in the three ADHD groups are presented in Figure 2. The figure suggests that there are similar patterns for the three ADHD groups concerning the frequency of individuals confirming each item.

ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.
A-TAC = The Autism-Tics, AD/HD, and other Comorbidities inventory
ADHD only = Individuals displaying ADHD without trauma exposure.
ADHD+trauma = Individuals displaying ADHD who were trauma-exposed without subsequent PTSD.
ADHD+PTSD = Individuals displaying ADHD who were trauma-exposed with subsequent PTSD.

Figure 2. Percentage of individuals within each of three ADHD groups who confirmed each item on the ADHD domain of A-TAC. Items on the concentration and attention module are presented to the left and items on the impulsivity and activity module are presented on the right.

Differences in ADHD with comorbid PTSD based on whether the onset of ADHD predates or postdates the trauma exposure

After dividing the *ADHD+PTSD* group into the subgroups *ADHD before trauma* and *ADHD after trauma*, the two groups were compared on age at onset of ADHD, total ADHD A-TAC score, A-TAC score on the concentration and attention module, and A-TAC score on the impulsivity and activity module, as presented in Table 5. The two subgroups did not show any significant differences on any of the examined aspects of ADHD.

Table 5.

Medians, mean values and standard deviations on aspects of ADHD for the two subgroups of comorbid ADHD and PTSD.

	ADHD before trauma $n = 42$	*ADHD after trauma* $n = 55$	Test statistics	P-value	Effect size
Age at onset of ADHD	$Mdn = 4.5$ $M = 4.10 \pm 2.05$	$Mdn = 5.0$ $M = 5.12 \pm 1.58$	$z = -2.43$	.015	$r = -.25$
Total ADHD A-TAC score	$Mdn = 11.5$ $M = 12.17 \pm 3.32$	$Mdn = 12.5$ $M = 12.37 \pm 3.70$	$z = -0.21$	.835	$r = -.02$
A-TAC score on the concentration and attention module	$Mdn = 6.5$ $M = 6.26 \pm 1.94$	$Mdn = 6.5$ $M = 5.99 \pm 2.10$	$z = -0.45$	.651	$r = -.05$
A-TAC score on the impulsivity and activity module	$Mdn = 5.5$ $M = 5.90 \pm 2.57$	$Mdn = 7.0$ $M = 6.38 \pm 2.62$	$z = -1.02$	.309	$r = -.10$

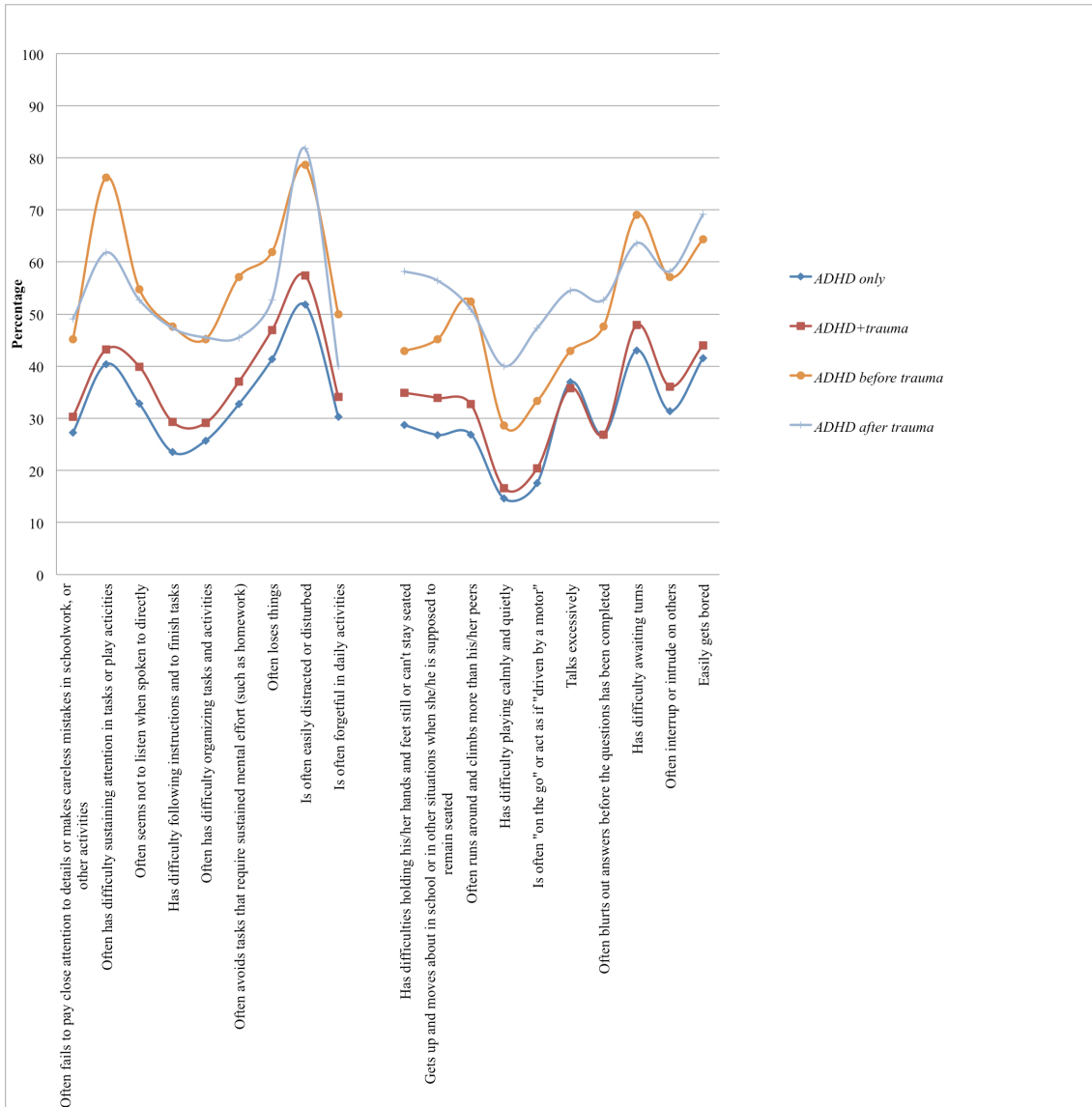A-TAC = The Autism-Tics, AD/HD, and other Comorbidities inventory
ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.
ADHD before trauma = Individuals with ADHD and PTSD with an age at onset of ADHD before the age at trauma
ADHD after trauma = Individuals with ADHD and PTSD with an age at onset of ADHD after the age at trauma

In order to further examine the patterns of confirmed items on the ADHD domain of the A-TAC, the percentages of individuals confirming each of the 19 A-TAC ADHD items in each group were compared (see Figure 3). The *ADHD before trauma* and *ADHD after trauma* groups were compared to the two ADHD groups *ADHD only* and *ADHD+trauma*. The figure indicates that the *ADHD before trauma* and the *ADHD after trauma* groups to a large extent share a similar pattern of confirmed items.

ADHD = Attention-deficit/hyperactivity disorder.
PTSD = Posttraumatic stress disorder.
A-TAC = The Autism-Tics, AD/HD, and other Comorbidities inventory
ADHD only = Individuals displaying ADHD without trauma exposure.
ADHD+trauma = Individuals displaying ADHD who were trauma-exposed without subsequent PTSD.
ADHD before trauma = Individuals with ADHD and PTSD with an age at onset of ADHD before the age at trauma.
ADHD after trauma = Individuals with ADHD and PTSD with an age at onset of ADHD after the age at trauma.

Figure 3. Percentage of individuals within each of four ADHD groups who confirmed each item on the ADHD domain of A-TAC. Items on the concentration and attention module are presented to the left and items on the impulsivity and activity module are presented on the right.

Discussion

The overall aim of this study was to assess the symptomatic associations between ADHD symptomatology, trauma exposure, and PTSD symptomatology. The overall finding indicates a strong association between ADHD symptomatology, trauma

exposure and PTSD symptomatology. The findings for each specific research question are presented and compared to earlier studies below.

Trauma exposure is positively associated with ADHD

The results of this study indicate that trauma exposure is associated with an elevated prevalence of ADHD. While only 9,7 % of the non-trauma-exposed individuals displayed ADHD, 16,8 % of the trauma-exposed individuals did. However, the effect size of the difference was small. This finding confirms the results of studies on similar research questions. Since questions on physical and emotional abuse/neglect as well as sexual abuse were included in the trauma definition in this study, the finding that trauma exposure is associated with an increased risk of ADHD can be seen as consistent with earlier findings showing an increased prevalence of ADHD in individuals who have experienced physical abuse (Fuller-Thomson et al., 2014), sexual abuse (Sonnby et al., 2011; Briscoe-Smith & Hinshaw, 2006) and maltreatment (Capusan et al., 2016; Ouyang et al., 2008). However, when interpreting these results one should bear in mind that earlier studies has suggested that the association between trauma exposure and ADHD might be mediated by PTSD symptoms (Husain et al., 2008; Spencer et al., 2015).

PTSD is positively associated with ADHD

The results of this study indicate that the development of PTSD after trauma exposure is associated with an elevated prevalence of ADHD. Amongst trauma-exposed individuals without PTSD, 15,0 % displayed ADHD compared to 34,2 % of trauma-exposed individuals with PTSD, resulting in a small between group effect size. This finding is in line with a large body of earlier research, showing a strong connection between ADHD and PTSD in both children (Biederman et al., 2014; Spencer et al., 2015, Husain et al., 2008) and adults (Kessler, 2006).

ADHD in combination with trauma exposure and comorbid PTSD is associated with more severe ADHD symptomatology

Through further examination of the individuals with ADHD, the results of this study indicate that ADHD in combination with trauma exposure is associated with more severe ADHD symptomatology compared to ADHD without trauma exposure. Furthermore, ADHD in combination with PTSD was associated with more severe ADHD symptomatology compared to ADHD in trauma-exposed individuals without PTSD. All between group effect sizes were small. These findings are somewhat inconsistent with earlier research showing similar numbers of ADHD symptoms in individuals with comorbid ADHD and PTSD and individuals with ADHD without comorbid PTSD (Biederman et al., 2013; Ansthel et al. 2013). However, the earlier studies found indications of greater clinical severity in the individuals with comorbid ADHD and PTSD groups on other aspects, such as psychiatric comorbidity or psychosocial dysfunction (Biederman et al., 2013; Ansthel et al. 2013). These aspects of

greater clinical severity in individuals with comorbid ADHD and PTSD could be associated with the difference in ADHD severity found in this study.

When further examining the differences between the three ADHD groups (*ADHD only*, *ADHD+trauma*, and *ADHD+PTSD*), the patterns of confirmed items suggested that the difference in ADHD severity was quantitative (i.e., an increased general prevalence of ADHD symptoms), rather than qualitative (i.e., increased prevalence of some ADHD symptoms). These findings suggest that ADHD in combination with trauma exposure/PTSD should not be conceptualized as a different form of ADHD, but rather as a more severe form of ADHD than ADHD in non-trauma-exposed individuals or trauma-exposed individuals without PTSD.

The significant differences in ADHD severity between the three ADHD groups in this study might give the appearance of a causal relation between trauma exposure/PTSD and more severe ADHD symptomatology. However, it is important to bear in mind that the non-experimental and cross-sectional design of this study entails that causal hypotheses neither can be confirmed nor rejected. The finding that ADHD in combination with trauma exposure and comorbid PTSD is associated with more severe ADHD symptomatology could be explained by several models of explanation, and none of them have been formally tested in this study. These models of explanation include (1) that trauma exposure and PTSD could enhance ADHD symptoms (Capusan et al., 2016), (2) that individuals with ADHD could have a higher risk for parental abuse or maltreatment because of the increased risk to have parents with ADHD (Barkley, 2015), (3) that more severe ADHD symptomatology could be associated with higher risk of more severe traumas (Ford et al., 2000), (4) that individuals with ADHD might have an elevated vulnerability to develop PTSD after trauma exposure (Spencer et al., 2017), (5) that individuals that are vulnerable for ADHD could be vulnerable for PTSD as well (Spencer et al., 2017), and finally (6) that symptoms registered as ADHD symptoms could represent PTSD symptoms mimicking those of ADHD (Weinstein et al. 2000). Further research using other designs is needed to examine the relative explanatory values of these models.

The order of trauma and onset of ADHD is not associated with severity of ADHD symptomatology in comorbid ADHD and PTSD

The results of this study do not indicate that the order of trauma vs. onset of ADHD affects severity of ADHD symptomatology or type of ADHD symptomatology in individuals with comorbid ADHD and PTSD. This difference has not been examined explicitly in earlier studies of comorbid ADHD and PTSD. When examining the patterns of confirmed ADHD items, the two groups did not seem to show different patterns of ADHD symptomatology.

If individuals who developed ADHD after the trauma exposure would have displayed a different ADHD profile from individuals who displayed ADHD symptoms before the trauma exposure, this difference could hypothetically suggest that there would be a way to distinguish individuals whose hyperactive and inattentive symptoms result in an increased risk for trauma exposure from individuals who suffer from enhanced ADHD-like symptoms following trauma exposure. However, the findings of this study suggest that such a division of individuals with comorbid ADHD and PTSD would not be meaningful. Based on the multitude of models of explanations for the

associations between ADHD and PTSD mentioned above, such a division is probably too simplistic to fully make sense of the complex processes affecting an individual's risk for ADHD, trauma exposure, and PTSD.

Limitations

This study has a great strength in the large size of the sample (N = 16,340), which is considerably larger than many other studies comparing ADHD with and without trauma exposure/PTSD. However, there are some limitations in this study worth mentioning.

The use of twin subjects. Using the Swedish Twin Registry as a sample has several apparent advantages in form of representation and sample size. However, some studies have reported that twins might have an increased risk for ADHD compared to singletons (Levy, Hay, McLaughlin, Wood, & Waldman, 1996), whereas other studies have found little evidence for such differences (Robbers et al., 2010; Simonoff et al., 1997). The findings by Levy et al. (1996), in combination with the non-responder analyses by Anckarsäter et al. (2011) indicating an higher prevalence of ADHD in non-responders of the CATSS study, suggests that there could have been an underrepresentation of ADHD in the studied sample.

As mentioned under *Analytical procedures*, non-parametric tests were used throughout in this study in order to minimize the effects of the interdependence in the sample owing to the correlation between twins. However, since the examined variables have shown very high heritability (Barkley, 2015; Larsson et al., 2013), there is a risk that the use of a twin sample could have led to a somewhat different result than if a singleton sample would have been used.

Assessment of ADHD symptomatology. When using the A-TAC for assessment of ADHD, two validated cut off values has been suggested, one low (6.0) and one high (12.5) (Larson et al., 2010). The low cut off was used in this study, which has yielded a sensitivity/specificity of 0.91/0.73 in an earlier study (Larson et al., 2010). The high cut off of 12.5, which has yielded a lower sensitivity and higher specificity of 0.56/0.93 in an earlier study (Larson et al., 2010), has in some studies been recommended for large-scale epidemiological studies (Larson et al., 2010; Larson et al., 2013). However, the high cut off was considered to result in an excessively large number of false negative cases in the studied sample and was therefore not used in this study. Since the overall aim of this study was to assess the associations with ADHD *symptomatology* (rather than a clinical diagnosis of ADHD), the low cut off was considered to give the best balance between sensitivity and specificity needed to fulfil the aim of the study. However, this might have resulted in a higher rate of false positive cases among individuals labelled with the proxy for ADHD diagnosis used in this study.

Assessment of trauma exposure. The questionnaire assessing trauma exposure used in this study has not been validated. Furthermore, screening for trauma exposure was parent-reported in this study, which might have introduced a bias in the assessment. Studies have found significant discrepancies between parent and child reports of the number of traumatic events experienced by the child, resulting in an underestimation of the child's level of exposure as well as the level of impact of the trauma (Schreier, Ladakakos, Morabito, Chapman, & Knudson, 2005; Stover, Hahn, Im, & Berkomitz, 2010). These findings were confirmed by Tingskull et al. (2015) in a Swedish study on

1,174 12-year old children and their parents. Parents has also been shown to underreport the child's exposure to domestic violence, which has been attributed to parental denial or minimizing the presence of children during incidents of violence by suggesting that the children for example were asleep or playing outdoors (Jaffe, Wolfe, & Wilson, 1990). Thus, there might have been an underreporting of trauma exposure in this study.

Assessment of PTSD symptomatology. Relying on parental report of PTSD symptoms poses some methodological difficulties. An event that is perceived as traumatic by the child may also be traumatic to the parent, either because the parent was an indirect participant in the same trauma or because the trauma's impact on the child may be, in itself, distressing to the parent (Shemesh et al., 2005). The use of parental reports of PTSD symptoms in children as a diagnostic instrument has been criticized and studies have found a moderate to low correlation between parent and child report of PTSD symptoms in the child (Shemesh et al., 2005; Chaffin & Shultz, 2001; Gerring et al., 2001). Shemesh et al. (2005) found no significant association between parent's report of PTSD symptoms in their children and the "best estimate" clinical diagnosis of PTSD in the child. However, this does not necessarily indicate that child self-report measures are superior to parent-report measures when assessing the effects of trauma, considering that children might lack the necessary cognitive and verbal abilities to recognize and express some trauma-related symptoms (Broome, 2009). When comparing child-reported and parent-reported measures of depression and anxiety, Muris, Meesters and Spinder (2003) found that children tend to be better at reporting internalizing symptoms and distress than their caregivers, whereas the opposite is true for externalizing symptoms. However, there is a risk that the use of parental report might have lead to an inaccurate reporting of PTSD symptoms in this study.

Neither the revised and shortened version of the PCL-C nor the cut off used as proxy for PTSD diagnosis in this study has been validated. The lack of items assessing the avoidance criterion needed for PTSD diagnosis, and the cut off of one intrusion symptom and one symptom of increased arousal might have resulted in an inaccurate assessment of PTSD in the sample. This hypothesis is supported by the findings of Giacona et al. (1995), who found that while 70.3 % of trauma-exposed individuals met the intrusion criterion and 56.4 % met the increased arousal criterion, only 14.5 % met all criteria for PTSD diagnosis. Thus, the cut off for PTSD used in this study might have resulted in an elevated rate of false positive cases among individuals labelled with the proxy for PTSD diagnosis.

Conclusions

Despite some limitations, this study adds to the understanding of the association between ADHD symptomatology, trauma exposure, and PTSD symptomatology in children. The results suggest that ADHD and PTSD are two disorders with a strong association and that trauma-exposed individuals are at risk for a more severe ADHD symptomatology. Furthermore, the results suggest that it might not be fruitful to divide individuals with comorbid ADHD and PTSD into individuals with onset of ADHD before vs. after the trauma, at least not regarding their severity of ADHD symptomatology. This conclusion might guide clinicians not to retreat to dichotomous models of "true" ADHD leading to trauma exposure vs. ADHD-like symptoms that follows trauma exposure. Instead, the conclusion of this study implies that the

associations between ADHD symptomatology, trauma exposure and PTSD symptomatology are more complex and might be multidirectional. Further research is needed to fully understand the processes behind the associations between childhood adversity and ADHD, as well as how to best diagnose and treat trauma-exposed children with ADHD.

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