Attention control therapy for acute stress disorder: A randomized controlled trial

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Abstract

Background: A subset of people exposed to traumatic events develop acute stress disorder (ASD), and approximately half of people with ASD develop posttraumatic stress disorder (PTSD). This randomized controlled trial examined the efficacy of internet-delivered attention control therapy (ACT), previously shown to reduce PTSD symptoms, as an adjuvant to treatment as usual in the community for patients with ASD.

Methods: About 119 participants with ASD were randomly assigned to ACT or treatment as usual in the community within the first month following their traumatic event. PTSD symptoms and attention patterns were measured.

Results: A significant reduction in stress-related symptoms was noted across participants with no difference between the two groups. Approximately half of the participants developed PTSD 2 months after the trauma. High attention bias variability was associated with elevated PTSD symptoms. However, attention bias variability did not change due to the therapy sessions.

Conclusions: Internet-delivered ACT was no more effective in reducing risk for PTSD in participants with ASD than treatment as usual in the community. Although elevated attention bias variability was detected in the patients with ASD, ACT failed to engage this cognitive target. Finally, ACT-based prevention research should proceed with caution given the possibility that this intervention might be associated with symptom worsening as indexed by the Clinical Global Impression scale.

KEYWORDS
acute stress disorder, attention control therapy, cognitive bias modification, early intervention, PTSD, secondary prevention

1 INTRODUCTION

Acute stress disorder (ASD) involves stress-related symptoms occurring 3–30 days following a traumatic event (American Psychiatric Association, 2013). ASD afflicts 10–20% of trauma exposed individuals and 50–70% of those with ASD later develop posttraumatic stress disorder (PTSD; Bryant, 2003; Dai et al., 2018). Meta-analyses support the efficacy of trauma-focused cognitive behavioral therapy (CBT) to prevent PTSD among patients with ASD (Bisson, Roberts, Kitchener, & Kenardy, 2009; Kliem & Kröger, 2013; Kornør et al., 2008), and is the indicated therapy of choice for ASD by the US Department of Veterans Affairs and the Department of Defense (2017). However, CBT requires extensively trained therapists, is quite expensive and entails high dropout rates (Hembree et al., 2003; Imel, Laska, Jakupcak, & Simpson, 2013). In addition, many treated patients with ASD still develop PTSD (Kornør et al., 2008), creating a need for alternative treatments. Here, we test the efficacy of attention control therapy (ACT), a novel computerized intervention for PTSD (Badura-Brack et al., 2015; Lazarov et al., 2019), in reducing risk for PTSD among patients with ASD. The delivery of ACT does
not require highly trained therapists, it is less emotionally demanding than many psychotherapies, and has a high dissemination potential, especially for individuals who are physically disabled or immobile following trauma (Amstadter, Broman-Fulks, Zinzow, Ruggiero, & Cerceo, 2009).

Severe traumatic stress and PTSD may involve imbalance in patients’ threat monitoring system, reflected in intense fluctuations between threat vigilance and threat avoidance (Schoorl, Putman, Van Der Werff, & Van Der Does, 2014; Shechner & Bar-Haim, 2016). Such fluctuations, coined attention bias variability (ABV), are elevated in patients with PTSD (Alon, Naim, Pine, Bliese, & Bar-Haim, 2019; Bardeen, Tull, Daniel, Evenden, & Stevens, 2016; Iacoviello et al., 2014; Naim et al., 2015; Swick & Ashley, 2017), and, therefore, mark a potential target for intervention. However, ABV levels in ASD populations have not been studied yet.

ACT is a computerized protocol based on a probe detection task thought to normalize ABV levels (Badura-Brack et al., 2015). In ACT, response targets appear with equal probability at neutral and threat locations on a screen, prompting patients to ignore these cues and spread their attention equally across neutral and threat information. Originally, ACT was used as a control condition to attention bias modification (ABM) that shifts attention away from threat. However, accumulating research suggests that ACT may reduce PTSD symptoms.

Four randomized trials directly contrasted the efficacy of ABM and ACT for PTSD (Badura-Brack et al., 2015; Lazarov et al., 2019; Schoorl, Putman, & Van Der Does, 2013). Schoorl et al. (2013) found ACT and ABM to be equally effective for PTSD symptoms. The other three trials found ACT to be more effective than ABM (Badura-Brack et al., 2015; Lazarov et al., 2019). Badura-Brack et al. (2015) reported that ACT but not ABM reduced ABV in a way that partially mediated clinical improvement. Although the exact mechanism by which ACT reduces PTSD symptoms is not clear, trials suggest that ACT rather than ABM may be the intervention of choice for PTSD among computerized attention treatments.

The current trial examines the efficacy of internet-delivered ACT as an adjuvant to treatment-as-usual (TAU) in the community compared with TAU in reducing stress-related symptoms and risk for PTSD in patients with ASD. We expected: (a) adjuvant ACT to be more effective than TAU only in reducing PTSD symptoms and risk for PTSD; (b) ABV and ASD symptom severity to positively correlate; and (c) ABV to reduce following ACT.

2 | METHODS

2.1 | Participants

A CONSORT diagram appears in Figure 1. Participants were 119 survivors of traumatic events admitted to the Tel-Aviv Medical Center’s emergency department between February 2016 and February 2019 ($M_{\text{age}}=37.45$ years, standard deviation [SD] = 12.86, range= 18–69). Participants were randomized to online ACT or TAU (see below). Inclusion criteria were: (a) a traumatic event in the past 30 days; (b) scores $\geq 7$ on the ASD inventory (ASDI; Bryant, Harvey, Dang, & Sackville, 1998); and (c) fluency in Hebrew. Exclusion criteria were: (a) prior PTSD diagnosis, to ensured that the trauma symptoms under investigation are caused by the recently experienced trauma rather than reflecting PTSD symptoms associated with an earlier trauma; (b) psychotic or bipolar disorders; (c) epilepsy or brain injury; (d) suicidal ideation; (e) drugs or alcohol misuse; (f) concurrent psychotherapy; (g) pharmacological treatment that is not stabilized over the past 3 months; (h) special populations (i.e., pregnant women, enlisted soldiers, and cognitively impaired); and (i) injury that required more than 3 days of hospitalization.

It should be noted that potential participants were contacted with an initial phone screen within the first month of hospital discharge. Full clinical assessments were conducted for participants who: (a) provided informed consent to participate; (b) reported high level of ASD symptoms; and (c) did not report exclusion criteria. The Institutional Review Boards of Tel Aviv University and the Sourasky Medical Center approved the study. Clinicaltrials.gov identifier: NCT02591485.

2.2 | Measures

2.2.1 | Clinical status

The ASD Interview (Bryant et al., 1998) was used to determine the presence of ASD. The Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) was used to determine comorbidity. Diagnostic interviews were conducted over the phone by two independent evaluators, graduate level clinical psychology students trained to 85% reliability with an experienced clinical psychologist, all blind to group assignment.

The ASD Interview (Bryant et al., 1998) is a diagnostic interview for ASD modified according to Diagnostic and Statistical Manual of Mental Disorders-fifth edition (DSM-5) criteria. A total score $\geq 7$ leads to an ASD diagnosis (Bryant, personal communication, 2016). The ASDI possesses strong test–retest reliability, and good internal consistency, sensitivity, and specificity (Bryant et al., 1998).

MINI is a structured diagnostic interview for DSM-IV and ICD-10 psychiatric disorders. The MINI has good inter-rater reliability, sensitivity, and specificity, and good test–retest reliability (Sheehan et al., 1998).

2.2.2 | Primary outcomes

Primary outcomes were: (a) total PTSD severity score; and (b) categorical diagnosis of PTSD based on the Clinician-Administered PTSD Scale (CAPS-5; Weathers et al., 2013) at posttreatment. The CAPS is a structured interview used to diagnose PTSD according to DSM-5 criteria (Bovin et al., 2016; Weathers et al., 2018). Cronbach’s $\alpha$ in the current sample = .89.
2.2.3 | Secondary outcome

The PTSD Checklist 5 (PCL-5; Weathers et al., 2013) is a self-report inventory assessing severity of PTSD symptoms corresponding to DSM-5 criteria. Scores can range 0–80 with higher scores reflecting greater severity (Armour et al., 2015; Bovin et al., 2016; Liu et al., 2014). Change from pre- to posttreatment in total PCL-5 scores served as a secondary outcome. Cronbach’s α in the current sample was .78 and .89 at pre- and posttreatment, respectively.

2.2.4 | Additional clinical outcomes

The Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001) is a self-report depression rating scale consisting of nine items on which the diagnosis of DSM-IV of major depression is based. Scores can range 0–27 with higher scores reflecting greater depression. The PHQ-9 has good validity, test–retest reliability, and internal consistency (Kroenke et al., 2001). Cronbach’s α in the current sample was .71 and .86 at pre- and posttreatment, respectively.

The Sheehan Disabilities Scale (SDS; Sheehan, 1983) is a functioning impairment three-items scale for the domains of work, social, and family life. Each item ranges 0–10 in severity. The SDS has high reliability and construct validity (Leon et al., 1993) and was collected at pre- and posttreatment.

Clinical Global Impression Severity and Improvement scales (CGI-S & CGI-I; Guy, 2000) were used to assess participants’ global clinical condition. The CGI-S and the CGI-I are single-items, assessing severity and improvement of illness, respectively. CGI-S was scored by the independent evaluators, whereas the CGI-I was self-reported by participants. Both measures were collected at posttreatment.
The measures above were translated into Hebrew and back-translated to English. The English back-translation was compared with the original. Words or phrases where the retranslated and the original versions were not directly matched were discussed by two Hebrew–English bilingual researchers until a final agreed upon Hebrew version was formulated.

### 2.3 | Treatments

#### 2.3.1 | Attention control therapy

ACT involved six, weekly, internet-delivered, word-based dot-probe sessions as in Badura-Brack et al. (2015). Each session included 160 trials (128 threat-neutral and 32 neutral-neutral). General threat words were used, paired with neutral words with the same number of letters and use frequency in Hebrew. Each trial began with a centrally presented fixation-cross (500 ms), followed by a pair of words presented simultaneously above and below the fixation (1,000 ms). Then, a target ("E" or "F") appeared in the location previously occupied by one of the words. Participants had to discriminate probe type via button press. Target probes remained on the screen until response, after which the next trial began. Target probes appeared with equal probability at the locations of threat and neutral words, with the intention of attenuating fluctuations in attention toward and away from threat (Iacoviello et al., 2014; Naim et al., 2015). ABV was calculated per Naim et al. (2015) to reflect within-session variability in threat-related attention bias normalized to individual task performance (Iacoviello et al., 2014; Naim et al., 2015; Price et al., 2015). Test–retest reliability of ABV was modest and similar to the reliability reported in previous studies (Naim et al., 2015), with intersession correlations ranging .38–.64, all $p < .03$. Importantly, in our design, we prioritized clear interpretation of clinical effects over interpretation of mechanism-related process (i.e., change in ABV). Therefore, attention measurements were applied only for the ACT group. Such measurements in the TAU group would have induced a low dose ACT (i.e., two sessions). Finally, participants were free to seek treatment in the community for their posttraumatic symptoms. Indeed, 15 participants from the ACT group (25%) reported they had started treatment in the community during the study period (eight were treated by medications, four received psychosocial treatment, and three received a combination of both).

#### 2.3.2 | Treatment-as-usual

Following enrollment to TAU, participants were informed that they would be contacted by phone for a clinical reassessment in 2 months’ time. Participants had no contact with the research team during this period. The follow-up assessment coincided with the posttreatment assessment of the ACT group. Fourteen participants from the TAU group (23.7%) had started treatment during that period (seven with medications, six a psychosocial treatment, and one with a combination of both).

### 2.4 | Procedure

We employed a systematic outreach to survivors of traumatic events who were admitted to the emergency room of a large metropolitan hospital. Logged records of trauma survivors were first filtered by age, number of hospitalization days (maximum of 2 days), and hospitalization that involved a potentially traumatic event (i.e., car accident, work accident, physical assault, and terror attack). Eligible survivors ($N = 6,473$) were contacted via telephone. Of these, 5,139 did not continue to the ASD interview (declined participation or did not meet inclusion criteria). Of the remaining eligible survivors, 1,334 agreed to a full diagnostic interview—1,215 did not meet the inclusion criteria and 119 were enrolled in the study and randomly assigned to either ACT or TAU. Randomization was conducted using a computer-generated set of random allocations overseen by a support researcher who did not perform data collection and was not involved in the study in any other capacity. The allocation set was prepared in advance of the start of the study and allocations were consecutively allotted irreversibly to each new patient once included in the study.

Participants assigned to ACT ($n = 60$) were provided access to a website through which therapy was delivered from home (six sessions, once a week over 6 weeks). Compliance was monitored noting: (a) starting a session; (b) number of trials completed; and (c) performance accuracy. Weekly electronic reminders of the upcoming session were sent to participants in the ACT condition, as well as provision of a contact person for guidance and technical support.

In the TAU condition ($n = 59$), participants were evaluated again 2 months after enrollment and had no contact with the research team during the ensuing weeks. Self-reported measures (PCL-5, PHQ-9, and the SDS) were administered pre- and posttherapy. CAPS-5 interviews and a clinical global status (CGI-S/I) were applied only post therapy.

### 2.5 | Data analyses

χ² and independent sample t tests were used to compare descriptive characteristics between the study groups. Intervention effects were tested with random-effects time-series models in generalized estimating equations (Zeger & Liang, 1986; Zeger, Liang, & Albert, 1988). This enabled consideration of correlations between repeated measurements and addressed missing data via estimated marginal means relying on the entire sample of randomized participants, taking into account all data collected at any time point including missing data. The generalized estimating equations models examined Time (pretreatment and posttreatment) by Group (ACT and TAU) effects on PTSD, depression, and SDS. These analyses specified an unstructured correlation matrix to model the correlations between participant-specific intercepts and change slopes in outcomes. The interaction terms between Time and Group (regressed on symptoms) reflect the outcomes of interest as recommended for clinical trials (Vens & Ziegler, 2012). Differences between groups in PTSD severity and diagnostic occurrence measured by CAPS-5 at posttreatment, were examined using an
independent sample t test and the $\chi^2$ test, respectively. Associations between the cognitive target (ABV) and symptoms at pre- and post-treatment were examined in the ACT group using simple correlations. Change in ABV over therapy sessions was tested using analysis of variance (ANOVA) with ABV as the dependent variable and session as a within-subject variable. Finally, to examine whether starting active treatment in the community after the traumatic event had affected clinical outcomes, generalized estimation equations analyses were conducted with Treatment (received and not received) and Group (ACT and TAU) as between-subject variables and Time (pretreatment and posttreatment) as a within-subject variable on PCL-5, PHQ-9, and SDS. In addition, ANOVAs examined Treatment-by-Group effects on CAPS-5 and CGI-S/I. A significance level of $\alpha = .05$ was used to detect effects. All tests were two-tailed.

The current study was powered to detect a small between-groups effect size of .25 based on small-to-medium effect sizes emerging from meta-analyses of early administered CBT to reduce risk for PTSD (Kliem & Kröger, 2013) and of computerized ABM trials (Linetzky, Pergamín-Hight, Pine, & Bar-Haim, 2015). With an $\alpha$ set at .05 and power (1-beta) set to .80 power, a sample of at least 45 participants per group would be required to detect an effect of .25. We further considered a dropout rate of ~20% (see Bryant et al., 2008; Kliem, & Kröger, 2013), and thus set out to enroll 60 participants per group.

## RESULTS

All randomized participants were included in analyses. Descriptive statistics for all demographic, clinical, and cognitive bias variables appear in Tables 1 and 2, respectively. The ACT and TAU groups did not differ in demographic characteristics, all $p > .09$, or clinical measures at pretreatment, all $p > .18$.

### 3.1 Compliance with ACT

Participants attended, on average, more than five of the six ACT sessions ($M = 5.32$, $SD = 1.62$, range = 1–6; 50 participants completed all sessions). Mean number of completed trials per session was 159.42 ($SD = 3.76$) out of 160. Finally, mean accuracy was 97% ($SD = 7%$). Together these results indicate very good compliance with the training task.

### 3.2 Clinical outcomes

#### 3.2.1 Primary outcomes: PTSD symptoms severity and PTSD diagnosis posttreatment

Participants receiving ACT and TAU did not differ in PTSD symptoms severity, $t(106) = 1.2$, $p = 0.23$, $d = .23$ or PTSD diagnosis rates, $\chi^2 = 0.90$, $p = .34$, at posttreatment, 2 months following trauma exposure. The rates of PTSD diagnosis in patients with ASD in the current sample for the ACT and TAU groups (58.2% and 49.1%, respectively), resemble those commonly reported in ASD populations (e.g., Bryant et al., 2015).

#### 3.2.2 Secondary outcome: Change in self-reported PTSD symptoms

Generalized estimating equations of PTSD symptoms change (PCL-5), yielded a main effect of time, Wald $\chi^2 = 33.24$, $p < .0001$, indicating decrease in PTSD severity from pre- to posttreatment in both groups. The time-by-group interaction, and the main effect of group were nonsignificant, $p > .36$.

#### 3.2.3 Change in additional clinical measures

Generalized estimating equations of change in depression symptoms (PHQ-9) and disability of functioning (SDS), yielded main effects of time,
Abbreviations: ABV, attention bias variability; ASDI, Acute Stress Disorder Interview; CAPS, The Clinician-Administered PTSD Scale; CGI-S/I, Clinical Global Impression Severity/Improvement; PCL-5, PTSD Checklist; PHQ-9, Patient Questionnaire-9; PTSD, posttraumatic stress disorder; SDS, The Sheehan Disability Scale.

* t(106) = 2.13, p < .04, d = .41.

<table>
<thead>
<tr>
<th></th>
<th>ACT group</th>
<th>TAU group</th>
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<tbody>
<tr>
<td></td>
<td>Pre treatment</td>
<td>Post treatment</td>
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<tr>
<td>ASDI</td>
<td>9.43</td>
<td>(1.70)</td>
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<tr>
<td>PCL-5</td>
<td>47.52</td>
<td>(10.09)</td>
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<td>PHQ-9</td>
<td>17.58</td>
<td>(4.07)</td>
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<td>SDS</td>
<td>21.28</td>
<td>(6.59)</td>
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<td>CAPS severity</td>
<td>–</td>
<td>27.6</td>
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<tr>
<td>CAPS diagnosis</td>
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<td>58.2%</td>
</tr>
<tr>
<td>CGI-S</td>
<td>–</td>
<td>3.8</td>
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<tr>
<td>CGI-I</td>
<td>–</td>
<td>3.11</td>
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<tr>
<td>ABV session 1 vs. session 6</td>
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<td>(0.04)</td>
</tr>
<tr>
<td>Mean ABV across all sessions</td>
<td>0.08</td>
<td>(0.03)</td>
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Wald $\chi^2 = 111.86$ and $119.5$, respectively, $p < .0001$, indicated a general decrease in depression and disability of functioning from pre- to posttreatment. The main effect of group and the time-by-group interaction were not significant, $p > .11$. In addition, participants in the two groups showed similar amounts of improvement pre- to posttreatment in CGI-I, t(105) = 0.91, $p = .37$, $d = .17$. Contrary to our expectations clinician-determined CGI-S was in fact better in the TAU group relative to the ACT group at posttreatment, t(106) = 2.13, $p < .04$, $d = .41$.

### 3.3 Associations between threat-related attention bias variability and clinical symptoms

Mean ABV across sessions significantly correlated with self-reported PTSD symptoms (PCL-5) at pre- ($r = .34$, $p < .01$) and at posttreatment ($r = .31$, $p < .03$), and revealed a nonsignificant correlation with clinician-evaluated PTSD symptoms (CAPS), $r = .24$, $p = .08$, at posttreatment (Figure 2). Mean ABV did not correlate with depression symptoms (PHQ-9) or with disability of functioning (SDS) neither pre- nor posttreatment, $p > .18$. ABV was high ($M = 0.08$; $SD = 0.04$) in our ASD sample and similar in magnitude to ABV previously reported in patients with PTSD (Naim et al., 2015). However, mean ABV was significantly higher in patients that ended-up with PTSD relative to patients who did not have PTSD at posttreatment, t(53) = 2.53, $p < .02$, $d = .73$.

### 3.4 Cognitive target engagement

Within the ACT group, analyses revealed no change in ABV over therapy sessions, F(5, 240) = 0.61 and 0.97, respectively, $p > .44$, $\eta^2_p < .02$, indicating that our internet-delivered ACT did not engage the targeted cognitive mechanism.

### 3.5 Seeking treatment in the community and clinical outcome

Generalized estimating equations indicated higher self-reported PTSD severity scores (PCL-5) across groups for those who sought and received treatment in the community, Wald $\chi^2 = 6.27$, $p < .05$. However, the main effects of treatment in the community for all the other symptom severity measures (CAPS-5, PHQ-9, SDS, and CGI-S/I) were all nonsignificant, $p > .09$. And, treatment-by-time-by-group and treatment-by-group interactions were nonsignificant for all outcome measures.

### 4 DISCUSSION

This is the first randomized trial to examine the efficacy of internet-delivered ACT for ASD. Contrary to our expectations, ACT appeared no more effective in reducing risk for PTSD than TAU. As in previous nonclinical studies of ASD outcome, about half of the participants remitted (e.g., Bryant et al., 2015). These remission rates are lower than rates of remission in studies of CBT ASD, which typically ~70–80% (e.g., Bryant et al., 2008). Nonsignificant group differences were also found in stress-related symptoms, depression, and disability of functioning, with patients in both groups showing reductions from pre- to posttreatment. The results also raise questions about possible ACT-related deleterious effects in ASD, since CGI-S
scores indicated more severe symptoms in the ACT than TAU group. Moreover, the ACT group had 9% more PTSD cases at posttreatment. In line with previous reports, ABV correlated with stress-related symptoms (e.g., Alon et al., 2019; Naim et al., 2015), suggesting a possible intervention target. However, our internet-delivered ACT appeared ineffective in engaging this target, which remained elevated across therapy sessions.

Although effective for PTSD (Badura-Brack et al., 2015; Lazarov et al., 2019), the current study found no support for internet-delivered ACT in treating ASD. Several factors could explain discrepant findings of ACT efficacy in ASD and PTSD. First, ACT may be a time-dependent intervention, and only treat trauma-related symptoms once these have become chronic and stable (e.g., Badura-Brack et al., 2015; Lazarov et al., 2019). Second, the current study contacted patients who presented to a medical emergency unit, unlike prior studies in PTSD, which recruited treatment-seeking patients. Differences in motivation for treatment might account for differences in efficacy (Shalev, Ankri, Peleg, Israeli-Shalev, & Freedman, 2014). Targeting other populations could complicate future work, since outreach designs are typical in ASD research given a critical time window for ASD interventions. Third, in contrast with previous studies (Badura-Brack et al., 2015), the current ACT failed to reduce ABV and thereby engage a possible cognitive target for the therapy. Prior work suggests that failing to engage an attention target predicts poor clinical response to ABM (Grafton et al., 2017; MacLeod & Grafton, 2016). This is particularly true when using internet-based protocols (Linetzky et al., 2015). Although patients in the current study complied well with the ACT protocol, it is still possible that the home environment in which therapy occurred impaired bias modification (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). Various factors that are closely controlled in the clinic (e.g., external noise, posture and distance to the screen, illumination, and competing tasks) can interrupt training gains when performing ACT from home. In addition, physically arriving to the clinic may interact with ACT effects to induce clinical benefits that are not present with home treatment (e.g., friendly interactions with therapists, traveling to the clinic provides opportunities to disprove negative beliefs about the world). It remains to be seen whether ACT delivered in the clinic, where previous randomized controlled trials for PTSD succeeded (Badura-Brack et al., 2015; Lazarov et al., 2019), may be more effective in cognitive target engagement and symptom reduction in ASD. Fourth, another explanation for the ineffectiveness of ACT for ASD may be associated with the high rates of natural recovery in ASD (Bryant, 2011; Zhou, Zhang, Wei, Liu, & Hannak, 2016). The high diversity in response to trauma on the one hand (Galatzer-Levy, Huang, & Bonanno, 2018), and the spontaneous recovery of about half the patients on the other hand, makes it difficult to reveal therapeutic effects of early interventions (Bisson et al., 2009; Kliem & Kröger, 2013; Qi, Gevonden, & Shalev, 2016).

As in previous studies (Alon et al., 2019; Naim et al., 2015; Price et al., 2015) ABV showed moderate-to-good retest reliability. ABV is thought to reflect the natural plasticity (i.e., fluctuations between threat vigilance and threat-avoidance) that is built into the threat monitoring system, rather than an index of a highly stable trait. Therefore, significant, but not highly robust, stability in ABV over time is expected. The current results also reveal for the first time that ABV is elevated in patients with ASD, and that there is a correlation between ABV and stress-related symptoms severity. This association strengthens the notion that ABV may offer a cognitive marker for trauma-related disorders and could serve as a potential target for intervention (e.g., Alon et al., 2019; Bardeen et al., 2016; Iacoviello et al., 2014; Naim et al., 2015; Swick & Ashley, 2017). Participants who had PTSD at posttreatment also showed higher ABV during the training sessions relative to patients with ASD who remitted at posttreatment. This implies that ABV may also serve as a precursor marker in the acute phase following trauma to identify those who are at greater risk to develop PTSD out of patients with ASD.

The results of the current study should be viewed in light of several limitations. First, ABV was collected only from participants in the ACT condition and, therefore, do not afford direct comparison with the TAU condition. We purposefully prioritized interpretation of clinical outcome over interpretation of underlying mechanisms. Therefore, we refrained from providing two ACT sessions (for the

![Figure 2](image-url)

**FIGURE 2** (a and b) Scatter plots and simple slope line between PTSD symptoms scores (PCL) at pre- and posttreatment and mean of ABV across all sessions within the attention control therapy (ACT) group. PTSD diagnosis is represented by the different colors of the dots. ABV, attention bias variability; PCL-5, PTSD Checklist; PTSD, posttraumatic stress disorder.
sake of measurement) to patients in the TAU condition, not opening interpretation of potential clinical findings to dose–response explanations. Future studies could overcome this limitation by applying alternative and independent measurements of threat-related ABV. Second, we measured cognitive and clinical outcomes once, shortly after the intervention and only 2–3 months posttrauma. It remains to be seen in future research whether the observed overall reduction in clinical symptoms, and the stable pattern of ABV is sustained over longer time periods. Third, cause of trauma in the current sample was primarily of motor vehicle accidents (MVA), limiting our ability to investigate trauma type-by-group interactions. This aspect could be followed more systematically in future research. Fourth, for ethical reasons, the current study did not prevent participants in either group from individually obtaining therapy in the community. While this approach carries high ecological validity, it also hinders the capacity to evaluate the full potential of ACT in this context. It should be noted that, in the current sample, patients who sought additional treatment had more severe PTSD symptoms than patients who did not. However, there is no clear indication for benefit of community care over natural recovery given similar symptoms reduction in both conditions. These findings highlight the need to further improve the available treatments for ASD in the community.

In conclusion, the present study is first to examine the efficacy of ACT among individuals with ASD following a recent traumatic event. Contrary to our expectations, internet-delivered ACT proved no more effective in reducing risk for PTSD than TAU in the community. Although, the current findings indicate that attentional fluctuations toward and way from threat in ASD, our ACT protocol failed to engage these fluctuations. Future research could focus on clinic-delivered ACT that may be more efficacious in modifying ABV in patients with ASD.

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CONFICT OF INTERESTS
The authors declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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REFERENCES


SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.