Attention-Deficit/Hyperactivity Disorder-Related Self-Reported Symptoms Are Associated With Elevated Concussion Symptomatology

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The aim of the present investigation was to provide insight into how postconcussion symptomatology may be altered in individuals exhibiting attention-deficit/hyperactivity disorder (ADHD)—related behaviors and examine factors that may be responsible for driving such relationships. A total of 99 individuals were assessed during the subacute phase of concussion recovery. Inattentive symptomatology, but not diagnosis of ADHD, was related to greater concussion-symptom severity and overall symptoms endorsed. Cluster and factor analyses highlighted that the relationship between ADHD symptomatology and concussion symptomatology was not a function of overlapping constructs being assessed (i.e., concussion-related symptomatology was not a proxy of ADHD-related symptomatology). These relationships were not mediated by parental observations of impairments in behaviors associated with executive functioning (i.e., executive dysfunction was not driving the greater concussion-related symptomatology associated with ADHD-related symptomatology). These findings highlight the importance of moving beyond categorical frameworks of ADHD to, instead, consider the continuum of underlying behaviors.

Keywords: SCAT5, ADHD-IV, executive function, BRIEF, subacute phase

Each year, an estimated 1.6–3.8 million individuals in the United States suffer concussive injuries (Langlois et al., 2006; Reid et al., 2020), with a sizable portion of these injuries occurring in individuals 18 years of age and younger (Kerr et al., 2019; P. Collings et al., 2017; Cook et al., 2017; Elbin et al., 2013; Houck et al., 2019; Iaccarino et al., 2018), such associations are not always observed, and a recent systematic review found no clear association for the presence of ADHD being related to differences in clinical outcomes (Cook et al., 2020).

Although a concussion is defined as transient neurological dysfunction in the absence of gross structural injury caused by a direct blow to either the head, neck, or body through which biomechanical forces are transmitted to the brain (McCrory et al., 2017), the heterogeneity and nonspecificity of symptom presentation makes concussion assessment and prognosis a challenging task (Kirkwood, 2006; Rosenbaum & Lipton, 2012). Patient-reported symptom inventories, such as the Sport Concussion Assessment Tool (SCAT), remain a staple of clinical assessments due to their ease of administration and the strong predictive relationship between greater acute/subacute symptom severity and prolonged concussion recovery across a number of clinical settings (Eagle et al., 2020; Eisenberg et al., 2013; Fehr et al., 2019; Putukian et al., 2021; Rabinowitz et al., 2015). However, despite an emerging body of evidence indicating that individuals with ADHD report greater concussion-related symptom severity compared with their non-ADHD counterparts (Biederman et al., 2015; Collings et al., 2017; Cook et al., 2017; Elbin et al., 2013; Houck et al., 2019; Iaccarino et al., 2018), such associations are not always observed, and a recent systematic review found no clear association for the presence of ADHD being related to differences in clinical outcomes (Cook et al., 2020).

One reason for such findings may be that clinical decision making is made more difficult given the similarity of ADHD and concussion-related symptoms, which may result in individuals being prematurely cleared to return to school, work, or play as residual impairments/symptoms are misattributed to a prior diagnosis of ADHD (Cook et al., 2020; Mautner et al., 2015). This is further compounded by the overrepresentation of ADHD within concussed individuals. Indeed, ADHD is considered a risk factor for concussive injuries given evidence indicating that individuals with ADHD are not only more likely to be concussed but also incur...
It is also important to consider that perceived stigma associated with assessments and treatments for mental health—particularly among populations such as elite athletes—may lead to fewer individuals actually being diagnosed or reporting being diagnosed with ADHD (Han et al., 2019). Thus, there may be individuals who exhibit impairments in ADHD-related behaviors but who do not identify with or have not been diagnosed with ADHD as well as individuals who exhibit impairments in ADHD-related behaviors that are below the diagnostic threshold for ADHD. Conversely, individuals who do not exhibit ADHD-related symptomatology may, nevertheless, have received an ADHD diagnosis—and identify as having ADHD—in hopes of incurring potential attentional/performance enhancement through ADHD medications (Buckman et al., 2013; Parr, 2011; Putukian et al., 2011). There is also the situation in which an individual may have a legitimate diagnosis of ADHD but has symptomatology that is well controlled and minimal as a result of their treatment regimen. Therefore, the dichotomous categorization of having ADHD or not may have little predictive importance for alterations in concussion symptomatology in comparison with focusing on the associated underlying behaviors of ADHD. As a substantial criticism of the extant literature is the focus only on such dichotomous categorization while ignoring ADHD subtype (Cook et al., 2020); moving toward focusing on the underlying symptomatology associated with ADHD using a dimensional nosological framework substantially advances this area of research to bring it in line with more modern perspectives of ADHD (McLennan, 2016).

The present investigation, thus, aimed to determine the independent contribution of ADHD diagnosis and ADHD-related symptomatology for explaining the variance of concussion-related symptomatology. It may be that greater symptom severity following a concussion occurs partly because the concussion-related symptomatology being endorsed is actually a proxy of ADHD-related symptomatology, given the overlap between ADHD and concussion-related symptoms (Cook et al., 2020; Mautner et al., 2015). Therefore, questions on patient-reported concussion symptom inventories relating to attention, concentration, irritability, and emotion regulation may coload with ADHD symptomatology, leading to greater reported concussion symptom severity. Indeed, some evidence in support of such an assertion is drawn from investigations by Elbin et al. (2013) as well as Brooks et al. (2016), who both observed greater symptom severity at baseline on a concussion symptom survey for individuals with ADHD—or individuals who reported having problems with attention deficit disorder (ADD)/hyperactivity—relative to those without ADHD/ADHD-related problems. Alternatively, it may be that this apparent link between ADHD and concussion-related symptom severity is the result of a central deficit in executive function. Indeed, a core cognitive deficit in executive functioning is believed to underlie the manifestation of ADHD (Antshel et al., 2014; Barkley, 1997) with children with ADHD exhibiting higher rates of behaviors associated with poor executive functioning as assessed using the Behavior Rating Inventory of Executive Functioning (BRIEF; McCandless & O’Laughlin, 2007; Topik et al., 2008; Zarrabi et al., 2015). Deficits in executive functioning are also characteristic following concussive injuries (McGowan et al., 2018, 2019) with pediatric, high-school, and collegiate athletes exhibiting poorer executive functioning as assessed using the BRIEF following a concussive injury relative to nonconcussed controls and normative data (Donders & Strong, 2016; Ivanisevic et al., 2021; Seichepine et al., 2013). This apparent link may, then, manifest as individuals with poorer executive function exhibiting greater ADHD symptomatology and being more likely to present with elevated concussion-related symptom severity on objective symptom reports. Thus, elevated concussion symptom severity for individuals with ADHD-related symptomatology may be mediated by a shared deficit in executive function. Collectively, such an understanding of how ADHD diagnosis and ADHD-related symptomatology are associated with concussion symptomatology—as well as insights into factors that may be responsible for driving such relationships—is of critical importance for advancing research in this area and contributing to the development of superior clinical practices.

Method

Participants

Analyses were conducted on a sample of 99 individuals (mean age: 13.6 ± 2.6 years [7.0–18.0], 45 female) that were evaluated at a pediatric sports medicine clinic during the subacute phase of concussion recovery—defined as the period between 4 and 44 days (6 weeks) following injury. Concussion diagnoses were confirmed by an attending physician using guidelines established by the Consensus Statement on Concussion in Sport (McCrorry et al., 2017) and the American Academy of Neurology (Giza et al., 2013). Upon arriving at the clinic, demographic information (e.g., age, sex, and ethnicity), injury characteristics, concussion symptomatology using the Sport Concussion Assessment Tool, and medical histories were collected from patients or parents/legal guardians who accompanied the patients. Parents/legal guardians in consultation with participants were also asked to complete the ADHD-IV rating scale and BRIEF questionnaire, keeping any changes in behavior since the injury in mind. All experimental protocols were approved by the Health Sciences Institutional Ethics Review Board, and all methods were carried out in accordance with those protocols and relevant guidelines and regulations regarding the use of human subjects.

Measures

SCAT5 Symptom Inventory

Concussion symptomatology was assessed using the SCAT, fifth edition (SCAT5) symptom inventory (Echemendia et al., 2017). The SCAT5 symptom inventory is a subjective assessment of symptom severity on a 7-point Likert scale ranging from 0 (none) to 6 (severe) across 22 items. Parents/legal guardians were provided with the symptom inventory and asked to respond to each item in consultation with participants, keeping any changes in behavior since the injury in mind. The total number of symptoms endorsed was calculated by summing all symptoms with a severity greater than 0 (maximum score of 22). The severity of symptoms endorsed was calculated by summing the severity for each of the 22 symptoms (maximum score of 132). The SCAT5 symptom inventory has shown adequate convergent validity (Asken et al., 2020; Gioia et al., 2009) and test–retest reliability (Hänninen et al., 2021; Register-Mihalik et al., 2013).
ADHD-IV Rating Scale

The ADHD symptomatology was assessed using the ADHD-IV rating scale (ADHD-IV; DuPaul et al., 1998). The ADHD-IV rating scale is a subjective assessment of the frequency of ADHD-related behaviors on a 4-point Likert scale ranging from 0 (never or rarely) to 3 (very often) across 18 items. The behaviors included in the survey are derived from the diagnostic criteria for ADHD established in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition revised (American Psychiatric Association, 2000; DuPaul et al., 1998). Parents/legal guardians were provided with the rating scale and asked to respond to each item in consultation with participants. Scores were summed into inattentive and hyperactive–impulsive symptom domains and transformed into symptom percentile scores based upon normative data based on age and biological sex (DuPaul et al., 1998). This assessment has demonstrated strong psychometric properties using parent report, including interrater reliability, test–retest reliability, and construct validity (Wyrwich et al., 2016; Zhang et al., 2005).

Behavior Rating Inventory of Executive Functioning

Executive functioning was assessed using the BRIEF (Gioia et al., 2000; Gioia & Isquith, 2011). The BRIEF questionnaire is a subjective assessment of parental observations of behaviors associated with executive functioning on a 3-point Likert scale ranging from 1 (never) to 3 (often) across a total of 86 behaviors (Vriezen & Pigott, 2002). Parents/legal guardians were provided the questionnaire and asked to respond to each item. Item scores were summed into composite scores and transformed into t scores based upon normative data (Gioia et al., 2000; Gioia & Isquith, 2011). The BRIEF has high test–retest reliability (Gioia & Isquith, 2011) as well as strong clinical utility in detecting impairments in executive functioning associated with psychological conditions and is modestly correlated with performance on cognitive tasks reliant upon executive control (McCandless & O’Laughlin, 2007; Roth et al., 2014). For the purposes of this investigation, executive function was quantified using the global executive composite t score.

Statistical Analyses

All data analyses were performed in R (version 4.0; R Core Team, 2019) utilizing a family-wise alpha level of $p = .05$. To examine the relationship between ADHD and concussion-related symptomatology, hierarchical linear regression analyses were performed to determine the independent contribution of ADHD-related symptomatology (as assessed using percentiles for inattentive symptoms, hyperactive–impulsive symptoms, and total symptoms) for explaining variance in concussion-related symptomatology (as assessed using the total number of symptoms endorsed and the severity of symptoms endorsed). Each analysis was conducted separately relative to an initial model accounting for descriptive and potential confounding factors (i.e., age, biological sex [0 = female and 1 = male], ethnicity [0 = White and 1 = non-White], presence of a learning disorder [0 = no and 1 = yes], the number of days since the concussion, whether the concussion resulted in a loss of consciousness [0 = no and 1 = yes], the number of previous concussions, and the use of ADHD-related medications [0 = no and 1 = yes]). The findings remained unchanged when presence of a learning disorder and the use of ADHD-related medications were excluded from the initial models. All variables and analyses residuals were screened for normality and homoscedasticity using histograms, Q–Q plots, Shapiro–Wilk tests (Shapiro & Wilk, 1965), and Studentized Breusch-Pagan tests (Koenker, 1981), and no data transformations were required or applied.

To assess the extent to which the relationship between ADHD and concussion-related symptomatology might manifest as a result of overlapping constructs being assessed (i.e., questions on the concussion symptom survey might be getting at ADHD-related symptomatology), a two-part approach was employed. First, cluster analysis was performed to determine related clusters of questions across the 22 questions from the SCAT5 and the 18 questions from the ADHD-IV rating scale based upon Euclidean distance. Ward hierarchical clustering using the “pvelust” (version 2.2; Suzuki & Shimodaira, 2006) R package was computed with multiscale bootstrap resampling ($n = 1,000$) to calculate $p$ values for each cluster. Second, exploratory maximum likelihood factor analyses were conducted using varimax rotation on the questions from the SCAT5 and ADHD-IV rating scale using the “stats” (version 4.0; R Core Team, 2019) R package. The optimal number of factors to extract was identified using a scree plot and requiring all factors to have eigenvalues greater than 1.0. Prior to analyses, all questions across the SCAT5 and ADHD-IV rating scale were scaled to a common scale, and assessment of the correlation between questions indicated that the data were appropriate for factor analysis as evidenced by a Kaiser–Meyer–Olkin measure of sampling adequacy of 0.84 and the Barlett test of sphericity: $\chi^2(780) = 2,848.2; p < .001$.

To assess the extent to which the relationship between ADHD and concussion-related symptomatology might manifest as a result of an underlying deficiency in executive function, mediation analysis was performed using the “mediation” (version 4.4.7; Tingley et al., 2014) and “Rmimic” (version 1.0.3; Pontifex, 2020) R packages with unstandardized indirect effects computed using 1,000 quasi-Bayesian approximation-based samples. This approach enabled the estimation of the mediating influence of executive function (as assessed using the BRIEF global executive composite t score) on the relationship between ADHD-related symptomatology for explaining variance in concussion-related symptomatology. Each analysis was conducted separately and included descriptive and potential confounding factors.

Results

The racial and ethnic distribution of the sample was zero (0%) American Indian or Alaska Native, one (1.0%) Asian, 31 (31.3%) Black or African American, zero (0%) Native Hawaiian or other Pacific Islander, 61 (61.6%) White or Caucasian, and four (4.0%) biracial or of other ethnicities, with two (2.0%) individuals identifying as being of Hispanic origin. Demographic data are provided in Table 1.

The Relationship Between ADHD Symptomatology and the Number of Concussion Symptoms Endorsed

ADHD Diagnosis

Hierarchical regression analysis indicated that being diagnosed with ADHD was unrelated to the number of concussion-related symptoms endorsed ($B = -1.62; 95\%$ confidence interval [CI] [–6.7, 3.46]; SE $B = 2.56, \beta = -0.09$) after accounting for the influence of descriptive and potential confounding factors,
\[ R^2_{\text{change}} < .01; F_{\text{change}(9, 89)} = 0.4; p = .5; f^2 < 0.01; 95\% \text{ CI [0.0, 0.04]} \]

**ADHD Symptomatology**

Individuals with greater inattentive ADHD-related symptomatology exhibited an increased number of concussion-related symptoms endorsed \( (B = -1.62; 95\% \text{ CI} [-6.7, 3.46]; SE B = 2.56; \beta = -0.09) \) after accounting for the influence of descriptive and potential confounding factors, \( R^2_{\text{change}} = .073; F_{\text{change}(9, 89)} = 9.0; p = .004; f^2 = 0.09; 95\% \text{ CI [0.01, 0.24]} \). No such relationship was observed for hyperactive–impulsive symptomatology, and the observed relationship with total ADHD-related symptomatology was weaker as a result (Figure 1 and Table 2).

The Relationship Between ADHD Symptomatology and the Severity of Concussion Symptoms Endorsed

**ADHD Diagnosis**

Hierarchical regression analysis indicated that being diagnosed with ADHD was unrelated to the severity of concussion-related symptoms \( (B = -4.90; 95\% \text{ CI} [-24.6, 14.83]; SE B = 9.93; \beta = -0.07) \) after accounting for the influence of descriptive and potential confounding factors, \( R^2_{\text{change}} < .01; F_{\text{change}(9, 89)} = 0.2; p = .6; f^2 < 0.01; 95\% \text{ CI [0.0, 0.03]} \).

**ADHD Symptomatology**

Individuals with greater inattentive ADHD-related symptomatology exhibited an increased severity of concussion-related symptoms \( (B = 0.24; 95\% \text{ CI [0.08, 0.40]]; SE B = 0.08; \beta = 0.31) \) after accounting for the influence of descriptive and potential confounding factors, \( R^2_{\text{change}} = .074; F_{\text{change}(9, 89)} = 9.2; p = .003; f^2 = 0.09; 95\% \text{ CI [0.01, 0.24]} \). No such relationship was observed for hyperactive–impulsive symptomatology, and the observed relationship with total ADHD-related symptomatology was weaker as a result (Figure 1 and Table 2).

Was This Relationship the Result of Potential Overlapping Constructs?

**Cluster Analysis**

Ward hierarchical clustering extracted two clusters of questions that were highly supported by multiscale bootstrap resampling. This clustering approach separated all the questions on the SCAT5 from questions on the ADHD-IV rating scale, \( p = .01 \) (Figure 2). No additional clusters were identified as highly supported by multiscale bootstrap resampling \((p’s \geq .13)\).

**Factor Analysis**

The exploratory factor analysis revealed a three-factor solution, which accounted for 50.0% of the total variance, \( \chi^2(663) = 1,054.0; p < .001 \). The three factors extracted aligned with the broader measures such that the primary factor (conclusion) accounted for 25.8% of the variance with an eigenvalue of 10.3 and represented all the questions on the SCAT5 (Figure 2). The second factor (inattention) accounted for 13.8% of the variance with an eigenvalue of 5.5 and represented the inattentive questions on the ADHD-IV rating scale, whereas the third factor (hyperactive–impulsive) accounted for 10.3% of the variance with an eigenvalue of 4.1 and represented the hyperactive–impulsive questions on the ADHD-IV rating scale (Figure 2). No ADHD-IV rating scale questions exhibited cross loadings with the concussion factor of more than 0.31.

Was This Relationship Mediated by a Deficiency in Executive Function?

**Total Number of Concussion Symptoms**

Analyses observed that the relationship between inattentive ADHD-related symptomatology and the increased number of concussion-related symptoms endorsed did not appear to be mediated by a global deficit in executive function (propotion mediated < 0.1%; 95% CI [0.0%, 20.6%]; average causal mediation...
### Table 2  Summary of Hierarchical Regression Analyses

<table>
<thead>
<tr>
<th>Model</th>
<th>$R^2$</th>
<th>$R^2$ change</th>
<th>$f^2$ [95% CI]</th>
<th>$B$ [95% CI]</th>
<th>SE $B$</th>
<th>β</th>
<th>$t$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total number of SCAT5 symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD diagnosis</td>
<td>.13</td>
<td>&lt;.01</td>
<td>0.0 [0.0, 0.04]</td>
<td>−1.62 [−6.70, 3.46]</td>
<td>2.56</td>
<td>−0.09</td>
<td>0.53</td>
</tr>
<tr>
<td>ADHD-IV inattentive</td>
<td>.20</td>
<td>.073**</td>
<td>0.09 [0.01, 0.24]</td>
<td>0.06 [0.02, 0.10]</td>
<td>0.02</td>
<td>0.31</td>
<td>3.0**</td>
</tr>
<tr>
<td>ADHD-IV hyperactive–impulsive</td>
<td>.14</td>
<td>.014</td>
<td>0.02 [0.0, 0.08]</td>
<td>0.04 [−0.02, 0.09]</td>
<td>0.03</td>
<td>0.13</td>
<td>1.2</td>
</tr>
<tr>
<td>ADHD-IV total</td>
<td>.18</td>
<td>.055*</td>
<td>0.07 [0.01, 0.20]</td>
<td>0.06 [0.01, 0.10]</td>
<td>0.02</td>
<td>0.26</td>
<td>2.6*</td>
</tr>
<tr>
<td><strong>SCAT5 symptom severity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>ADHD diagnosis</td>
<td>.13</td>
<td>&lt;.01</td>
<td>0.0 [0.0, 0.03]</td>
<td>−4.90 [−24.6, 14.83]</td>
<td>9.93</td>
<td>−0.07</td>
<td>0.5</td>
</tr>
<tr>
<td>ADHD-IV inattentive</td>
<td>.21</td>
<td>.07**</td>
<td>0.09 [0.01, 0.24]</td>
<td>0.24 [0.08, 0.40]</td>
<td>0.08</td>
<td>0.31</td>
<td>3.0**</td>
</tr>
<tr>
<td>ADHD-IV hyperactive–impulsive</td>
<td>.13</td>
<td>&lt;.01</td>
<td>0.0 [0.0, 0.04]</td>
<td>0.07 [−0.15, 0.29]</td>
<td>0.07</td>
<td>0.60</td>
<td>0.5</td>
</tr>
<tr>
<td>ADHD-IV total</td>
<td>.18</td>
<td>.05*</td>
<td>0.07 [0.01, 0.19]</td>
<td>0.21 [0.04, 0.37]</td>
<td>0.08</td>
<td>0.26</td>
<td>2.5*</td>
</tr>
</tbody>
</table>

*Note: $R^2$ reflects the adjusted $R^2$ value. All models reflect the addition of the ADHD-IV diagnosis or percentile to a model including age, biological sex (0 = female and 1 = male), ethnicity (0 = White and 1 = non-White), presence of a learning disorder (0 = no and 1 = yes), the number of days since the concussion, whether the concussion resulted in a loss of consciousness (0 = no and 1 = yes), the number of previous concussions, and the use of ADHD-related medications (0 = no and 1 = yes). The findings remain unchanged when presence of a learning disorder and the use of ADHD-related medications are excluded from the initial models. ADHD = attention-deficit/hyperactivity disorder; SCAT5 = Sport Concussion Assessment Tool 5th ed.

* $p < .05$. ** $p < .01$. 

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**Figure 1** — Scatterplots showing the relationship between ADHD-related diagnosis/symptoms and the total number of symptoms endorsed on the SCAT5 (top), as well as the severity of the symptoms endorsed on the SCAT5 (bottom). Note: A small amount of random jitter was introduced for all scatterplots graphically representing the data to prevent overplotting. ADHD = attention-deficit/hyperactivity disorder; SCAT5 = Sport Concussion Assessment Tool 5th ed.
Figure 2 — Dendrogram (left) showing the hierarchical relationships between questions on the ADHD-IV rating scale and the SCAT5 alongside a plot of factor loadings (right) for these questionnaires. ADHD = attention-deficit/hyperactivity disorder; SCAT5 = Sport Concussion Assessment Tool 5th ed.
effect = −0.02; 95% CI [−0.06, 0.01], p = .2; and average direct
effect = 0.09; 95% CI [0.04, 0.13], p = .002) while accounting for
the influence of descriptive and potential confounding factors.

Severity of Concussion Symptoms
Analyses observed that the relationship between inattentive
ADHD-related symptomatology and the increased severity of
concussion-related symptoms did not appear to be mediated by
a global deficit in executive function (proportion mediated <0.1%;
95% CI [0.0%, 47.1%]; average causal mediation effect = −0.05;
95% CI [−0.19, 0.09], p = .53; and average direct effect = 0.29;
95% CI [0.08, 0.50], p = .004) while accounting for the influence of
descriptive and potential confounding factors.

Discussion
The primary aim of the present investigation was to assess
how variance in concussion-related symptomatology may differ-
entially be explained by ADHD diagnosis and ADHD-related
symptomatology. Consistent with criticisms of the extant litera-
ture in this area (Cook et al., 2020), the use of a categorical
dichotomous framework to only consider if an individual was or
was not diagnosed with ADHD was observed to be a poor
predictor of concussion-related symptomatology. Specifically,
although the present sample exhibited a slight over-representation
of individuals diagnosed with ADHD (15.2%) relative to popula-
tion prevalence estimates, ADHD diagnosis was not observed to
relate to either the number of symptoms endorsed or the severity
of symptoms during the subacute phase of a concussive injury.
Thus, consideration of the presence or absence of ADHD diag-
nosis does not appear to provide any additional predictive value
for understanding concussion-related symptomatology after
accounting for the influence of descriptive (e.g., age, sex, ethnic-
ity) and potential confounding factors (e.g., presence of a learning
disorder, number of days since the concussion, whether the
concussion resulted in a loss of consciousness, the number of
previous concussions, and the use of ADHD-related medications).
Importantly, these findings remain even if potential factors that
may be collinear with ADHD diagnosis—such as the presence of a
learning disorder (8.1% of the sample) and the use of ADHD-
related medications (8.1% of the sample)—were excluded from
the initial models.

Novel to the present investigation was the utilization of a
symptom framework to, instead, consider how the continuum of
underlying behaviors associated with ADHD may be related to
concussion symptomatology. Findings observed a relationship
between subjective reports of ADHD symptomatology and con-
cussion symptomatology. An alternative hypothesis was that the link
between ADHD and concussion-related symptomatology may
potentially be explained by ADHD diagnosis and ADHD-related
symptoms. Specifi
cally, rather than only asking a binary question regarding the presence or absence of ADHD diagnosis, greater quality of information regarding ADHD symptomatology
may be provided by clinicians/practitioners incorporating Likert
ratings of inattentive behaviors associated with ADHD, such as
questions 1–9 of the ADHD-IV/V rating scale. Given the findings
reported herein, the use of such valid and reliable subscales of
inattentive ADHD-related symptomatology reflects a minimal
subset of questions necessary to quantify ADHD symptomatology
that is still predictive of concussion symptomatology.

Given the apparent relationship between ADHD and concus-
sion symptomatology, a secondary aim of the present investiga-
tion was to provide insight into factors that may be responsible for
driving this relationship. Although it has been speculated that this
relationship may simply be a manifestation of coloading of a
common set of symptoms across both ADHD and concussion
symptom inventories (Cook et al., 2020; Mautner et al., 2015), the
ADHD-IV rating scale and the SCAT5 do not appear to share an
underlying construct that might contribute to the greater endorse-
ment of concussion-related symptoms for those with more pro-
nounced ADHD-related symptoms. Specifically, both cluster and
factor analyses observed complete separation of items across these
inventories. Thus, the elevated concussion-related symptomatol-
yogy does not appear to be simply a proxy of ADHD-related
symptomatology. An alternative hypothesis was that the link
between ADHD and concussion-related symptom severity may
have been the result of a central deficit in executive function such
that individuals with poorer executive function would exhibit
greater ADHD symptomatology as well as be more likely to present
with elevated concussion-related symptom severity. Analysis of
the mediating influence of executive function (as assessed using the
BRIEF global executive composite t score) for the relationship
between ADHD and concussion symptomatology suggests this is
not likely to be the case, however. Specifically, the nature of the
indirect path between ADHD symptomatology and concussion
symptomatology with executive function as a mediator suggests
that executive function may actually be a suppressive variable in
this relationship. By including behaviors associated with executive
function within the model, the relationship between ADHD symp-
tomatology and concussion symptomatology gets stronger as
nuisance variability associated with executive functioning is sup-
pressed (MacKinnon et al., 2000). However, it is important to
note that the indirect relationship between variables was not
statistically significant. Given the small magnitude of the relation-
ships observed, Monte Carlo power analysis for indirect effects
(Schoemann et al., 2017) indicates that a sample of 5,000–18,000
participants would be necessary to achieve 80% power to control
for the probability of finding no mediating effect when a mediating/suppressive effect was actually present (i.e., Type II error or false
negative). Thus, although the current findings should be considered as preliminary, they provide initial estimates suggesting that, at
best, executive function would only partially mediate the relation-
ship between inattentive ADHD symptomatology and concussion
symptomatology—accounting for 20% of the relationship with the
considering ADHD-related symptomatology when evaluating an
individual following a concussive injury. Although capturing a
complete profile of ADHD-related symptomatology is preferable,
recognizing the limitations inherent within some clinical settings/
situations and being cognizant of potential concerns regarding
survey burden and questionnaire fatigue in such settings, the
specificity of this relationship to inattentive ADHD-related symp-
tomatology suggests that it would be feasible to focus just on the
inattentive subscales. Specifically, rather than only asking a binary
question regarding the presence or absence of ADHD diagnosis,
greater quality of information regarding ADHD symptomatology
may be provided by clinicians/practitioners incorporating Likert
ratings of inattentive behaviors associated with ADHD, such as
questions 1–9 of the ADHD-IV/V rating scale. Given the findings
reported herein, the use of such valid and reliable subscales of
inattentive ADHD-related symptomatology reflects a minimal
subset of questions necessary to quantify ADHD symptomatology
that is still predictive of concussion symptomatology.

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driving this relationship. Although it has been speculated that this
relationship may simply be a manifestation of coloading of a
common set of symptoms across both ADHD and concussion
symptom inventories (Cook et al., 2020; Mautner et al., 2015), the
ADHD-IV rating scale and the SCAT5 do not appear to share an
underlying construct that might contribute to the greater endorse-
ment of concussion-related symptoms for those with more pro-
nounced ADHD-related symptoms. Specifically, both cluster and
factor analyses observed complete separation of items across these
inventories. Thus, the elevated concussion-related symptomatol-
yogy does not appear to be simply a proxy of ADHD-related
symptomatology. An alternative hypothesis was that the link
between ADHD and concussion-related symptom severity may
have been the result of a central deficit in executive function such
that individuals with poorer executive function would exhibit
greater ADHD symptomatology as well as be more likely to present
with elevated concussion-related symptom severity. Analysis of
the mediating influence of executive function (as assessed using the
BRIEF global executive composite t score) for the relationship
between ADHD and concussion symptomatology suggests this is
not likely to be the case, however. Specifically, the nature of the
indirect path between ADHD symptomatology and concussion
symptomatology with executive function as a mediator suggests
that executive function may actually be a suppressive variable in
this relationship. By including behaviors associated with executive
function within the model, the relationship between ADHD symp-
tomatology and concussion symptomatology gets stronger as
nuisance variability associated with executive functioning is sup-
pressed (MacKinnon et al., 2000). However, it is important to
note that the indirect relationship between variables was not
statistically significant. Given the small magnitude of the relation-
ships observed, Monte Carlo power analysis for indirect effects
(Schoemann et al., 2017) indicates that a sample of 5,000–18,000
participants would be necessary to achieve 80% power to control
for the probability of finding no mediating effect when a mediating/suppressive effect was actually present (i.e., Type II error or false
negative). Thus, although the current findings should be considered as preliminary, they provide initial estimates suggesting that, at
best, executive function would only partially mediate the relation-
ship between inattentive ADHD symptomatology and concussion
symptomatology—accounting for 20% of the relationship with the
number of concussion symptoms and 47% of the relationship with concussion symptom severity—and, at worst, serves as a suppressive variable that strengthens the observed relationships. Regardless, the small magnitude of the indirect effect estimates suggest that a central deficit in executive function likely exhibits minimal influence on the relationship between ADHD symptomatology and concussion symptomatology.

Despite the strengths of the present investigation, the cross-sectional design employed only assessed symptomatology at a single point during the subacute phase of concussion recovery. As preinjury baseline measures were not available, we cannot determine the extent to which the findings reported herein are directly the result of concussive injury or may manifest as a result of some other factor. Indeed, as individuals with ADHD have higher rates of mental health comorbidities (e.g., depression, anxiety, and oppositional defiant disorders) compared with the general population (Angold et al., 1999; Spencer et al., 1999; Wilens et al., 2002), the elevated concussion-related symptomatology may be a byproduct of associations with these other disorders of mental health (Karr et al., 2020; Morgan et al., 2015). As such, further research utilizing longitudinal designs is necessary to better elucidate the extent to which ADHD symptomatology may be more informative for understanding differences in clinical outcomes than only considering ADHD diagnosis and how such relationships may be moderated by associations with other injury characteristics and postinjury profiles. Another limitation of the present investigation was the utilization of a slightly outdated ADHD rating scale designed to align with the diagnostic criteria for ADHD established in the Diagnostic and Statistical Manual of Mental Disorders (DSM) IV. However, the DSM-5 retained the same 18 core symptoms of ADHD assessed within the ADHD-IV rating scale aside from minor tweaks to add example behaviors for a few symptoms (Epstein & Loren, 2013). As such, there is no reason to believe that this limitation would have substantially impacted the findings reported herein.

Collectively, the findings reported herein suggest that ADHD-related symptomatology, but not ADHD diagnosis, is associated with an increase in concussion-related symptomatology. Furthermore, this relationship was not explained by an overlap in assess-

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