contribute to clinical recovery after sport-related concussion

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ABSTRACT

Objective: To determine the degree to which preinjury and acute postinjury psychosocial and injury-related variables predict symptom duration following sport-related concussion.

Methods: A total of 2,055 high school and collegiate athletes completed preseason evaluations. Concussed athletes (n = 127) repeated assessments serially (<24 hours and days 8, 15, and 45) postinjury. Cox proportional hazard modeling was used to predict concussive symptom duration (in days). Predictors considered included demographic and history variables; baseline psychological, neurocognitive, and balance functioning; acute injury characteristics; and postinjury clinical measures.

Results: Preinjury somatic symptom score (Brief Symptom Inventory–18 somatization scale) was the strongest premorbid predictor of symptom duration. Acute (24-hour) postconcussive symptom burden (Sport Concussion Assessment Tool–3 symptom severity) was the best injury-related predictor of recovery. These 2 predictors were moderately correlated ($r = 0.51$). Path analyses indicated that the relationship between preinjury somatization symptoms and symptom recovery was mediated by postinjury concussive symptoms.

Conclusions: Preinjury somatization symptoms contribute to reported postconcussive symptom recovery via their influence on acute postconcussive symptoms. The findings highlight the relevance of premorbid psychological factors in postconcussive recovery, even in a healthy athlete sample relatively free of psychopathology or medical comorbidities. Future research should elucidate the neurobiopsychosocial mechanisms that explain the role of this individual difference variable in outcome following concussive injury. ***Neurology*® 2016;86:1856–1863**

GLOSSARY

BESS = Balance Error Scoring System; **BSI-18** = Brief Symptom Inventory–18; **CI** = confidence interval; **DSM-V** = *Diagnostic and Statistical Manual of Mental Disorders, 5th edition*; **HR** = hazard ratio; **ImPACT** = Immediate Post-Concussion Assessment and Cognitive Testing; **MSVT** = Medical Symptom Validity Test; **SAC** = Standardized Assessment of Concussion; **SCAT3** = Sport Concussion Assessment Tool, 3rd edition; **SRC** = sport-related concussion; **SS** = standard score; **SWLS** = Satisfaction With Life Scale.

As the clinical and neurobiologic sequelae of sport-related concussion (SRC) are becoming better understood,[1–6] there is increasing interest in understanding individual differences in recovery. Identifying those at risk for prolonged recovery is critical to developing early interventions that maximize outcomes. Investigations into predictors of symptom recovery after SRC have largely focused on demographics, acute injury characteristics, and clinical assessment measures. Although findings have not been ubiquitous,[7–9] the most consistent independent predictor of recovery is early self-reported concussive symptoms.[10,11] Other variables historically thought to signify traumatic brain injury severity have not been predictive.[3] Consequently, acute symptom burden appears to be the strongest marker of concussion severity and recovery.[12]

Yet symptom burden likely conflates several distinct factors, including the consequences of the biomechanical forces during and neurologic processes after injury and one's premorbid predisposition to experience symptoms. The relevance of preinjury psychological factors in concussion recovery is accepted for civilian and military patients,[13] although studies of these

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populations have only estimated premorbid psychological functioning retrospectively.[14–17] One sample of personal injury claimants reporting chronic symptoms after mild cranial or cervical injuries has been published where preinjury psychiatric data were available. All 23 individuals had abnormal psychological profiles, with somatoform psychopathology most common.[18]

The aim of this longitudinal study was to comprehensively assess the degree to which preinjury and acute injury–related variables predict recovery after SRC. We employed preseason and postinjury assessment of psychological, neurocognitive, and balance functioning. We hypothesized that both premorbid psychological symptoms and acute postconcussive symptoms would predict symptom duration.

METHODS

Participants. This longitudinal study of SRC enrolled and followed contact and collision sport athletes from 9 high schools and 4 colleges in southeastern Wisconsin between August 2012 and March 2015.[19–21] Among the 2,055 athletes who completed preseason baseline assessments, 127 were concussed during the study and followed serially postinjury.

Standard protocol approvals, registrations, and patient consents. The study was approved by the Medical College of Wisconsin's Institutional Review Board. Adult athletes and parents of minor athletes completed written informed consent, and minor participants completed written assent, prior to their first evaluation.

Study design. The definition of concussion was extracted from that of the study sponsor, the US Department of Defense:

> mTBI is defined as an injury to the brain resulting from an external force and/or acceleration/deceleration mechanism from an event such as a blast, fall, direct impact, or motor vehicle accident which causes an alteration in mental status typically resulting in the temporally related onset of symptoms such as headache, nausea, vomiting, dizziness/balance problems, fatigue, insomnia/sleep disturbances, drowsiness, sensitivity to light/noise, blurred vision, difficulty remembering, and/or difficulty concentrating.[22]

All participants in the sample sustained a sport-related injury involving direct impact to the head and were diagnosed by a licensed athletic trainer.

The study protocol involved completion of a 90-minute preseason baseline examination and 60-minute postinjury examinations conducted within 24 hours of injury and at 8, 15, and 45 days postinjury (appendix e-1 on the *Neurology*® Web site at Neurology.org).

Main outcome measures. The primary outcome was self-reported duration of postconcussive symptoms (in days). Predictors of symptom duration were extracted from the baseline and postinjury clinical examinations, which consisted of demographics/health history, Wechsler Test of Adult Reading (baseline only),[23] Standardized Assessment of Concussion (SAC),[24] Sport Concussion Assessment Tool, 3rd edition (SCAT3) symptom checklist,[25] Brief Symptom Inventory–18 (BSI-18),[26] Balance Error Scoring System (BESS),[27] Green's Medical Symptom Validity Test (MSVT),[28] and Satisfaction With Life Scale (SWLS).[29] In addition, each athlete completed 2 of 3 computerized neurocognitive tests (these were pseudorandomly assigned as discussed in appendix e-1). Because data for Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) were available on the largest percentage of participants in the concussed study sample (70.6%), it was selected for analyses of computerized neurocognitive performance here.

Statistical analysis. As the primary aim of the study was to identify predictors of symptom duration, time to recovery was analyzed using a Cox proportional hazards model. As years of participation demonstrated a bimodal distribution, this variable was dichotomized at the valley of the distribution (≤9 years vs ≥10 years). Similarly, several clinical assessment variables had skewed or bimodal distributions. To facilitate interpretation of Cox regression output and avoid nonproportionality of hazards, we dichotomized (via median split) the SCAT3, BSI-18, SWLS, SAC, BESS, and ImPACT variables. The median score for each variable at baseline/24 hours was as follows: SCAT3 symptom severity 3/20, BSI-18 somatization standard score (SS) 97/100, BSI-18 depression SS 93/93, BSI-18 anxiety SS 96/96, BSI-18 global severity index SS 93/97, SWLS 28/28, SAC total score 27/26, BESS total score 13/13, ImPACT verbal memory 85.5/81.0, ImPACT visual memory 78.0/71.0, ImPACT visual motor speed 39.2/37.1, and ImPACT reaction time 0.56/.61. Psychiatric diagnostic history was excluded from analyses given its limited prevalence (n = 1). Sport was grouped into football, soccer, or other categories in order to avoid low-frequency categories. Analyses of ImPACT excluded 3 athletes with invalid baseline profiles. Inclusion of these athletes in other analyses did not alter the results (e.g., the mean difference in tables 1 and 2 hazard ratios [HRs] with vs without these 3 participants was 0.02).

Because of the large number of models being estimated, α was set to 0.01 to limit the rate of false discoveries. For all Cox regression models evaluated, we verified that the proportional hazards assumption was met. Initially, bivariate analyses were performed predicting symptom duration from each predictor. Because of our aim to identify and compare premorbid and injury-related predictors, we then underwent development of multivariable prediction models using premorbid and postinjury variables separately. We used forward stepwise elimination variable selection to find parsimonious multivariate models, with likelihood ratio tests used to determine whether model fit improved with additional predictors. Predictors considered for possible inclusion were those with p values from the unadjusted/bivariate analyses of ≤0.10. Finally, a parsimonious regression model aggregating the key preinjury and postinjury predictors was developed. In order to evaluate the contributions of theoretically related and empirically correlated preinjury and postinjury variables in predicting recovery, a mediation model incorporating both preinjury and postinjury symptom reporting variables in the prediction of symptom recovery was secondarily explored, as described below.

RESULTS

Sample characteristics. Sample demographics and acute injury characteristics are presented in table 1. The sample was 80.3% male and was distributed across sport as follows: 61.4% football, 23.6% soccer, 5.5% lacrosse, 3.1% wrestling, 3.1% ice hockey, 2.4% rugby, and 0.8% field hockey. All participants achieved passing scores on the MSVT. The median symptom duration was 5 days and 63.8% of the sample reported achieving symptom recovery within 1 week. The vast majority of participants

Table 1 Sample characteristics (n = 127)

	Mean ± SD or %
Demographics/history	
Male	80.3
College (vs high school)	63.8
Grade point average	3.23 ± 0.53
WTAR SS	101.05 ± 13.32
ADHD	9.6
Learning disability	4.8
Nonmigraine headaches	8.8
Migraine headaches	6.5
Psychiatric diagnosis	0.8
Prior concussions	
0	42.4
1	32.8
2	12.0
3+	12.8
Sport	
Football	61.4
Soccer	23.6
Field hockey	0.8
Wrestling	3.1
Lacrosse	5.5
Rugby	2.4
Ice hockey	3.1
Years of participation	8.40 ± 4.13
Preinjury (baseline) symptoms	
SCAT3 symptom severity	6.65 ± 10.40
BSI-18 somatization SS	97.23 ± 11.69
BSI-18 depression SS	93.85 ± 9.23
BSI-18 anxiety SS	93.32 ± 10.61
BSI-18 global severity SS	94.06 ± 12.19
Injury characteristics and acute symptoms	
Loss of consciousness	6.4
Posttraumatic amnesia	12.8
Retrograde amnesia	8.8
SCAT3 symptom severity	24.65 ± 18.79
BSI-18 somatization SS	103.85 ± 12.60
BSI-18 depression SS	95.54 ± 10.13
BSI-18 anxiety SS	93.23 ± 11.12
BSI-18 global severity SS	98.32 ± 11.88

Abbreviations: ADHD = attention-deficit/hyperactivity disorder; BSI-18 SS = Brief Symptom Inventory–18 Standard Score; SCAT3 = Sport Concussion Assessment Tool–3; WTAR SS = Wechsler Test of Adult Reading Standard Score (mean = 100; SD = 15).

(95.3%) reported being symptom-free by 1 month postinjury, and 2.4% of the sample required between 1 month and 45 days to achieve symptom remission. The point of recovery was unknown for 3 participants; 2 (1.6%) were symptomatic at their 45-day assessments and 1 (0.8%) was symptomatic at the day 14 assessment and was lost to attrition.

Preinjury predictors of symptom duration. Results of single predictor regression models for symptom duration using variables assessed at preseason examinations are presented in table 2. Baseline ratings of somatic symptoms (BSI-18 somatization; $p = 0.003$) was the only significant predictor ($\alpha = 0.01$), with high (vs low) baseline somatic symptom reporting associated with a 42% lower hazard of recovery (i.e., higher baseline somatic symptoms was associated with lower recovery rates). Sport ($p = 0.025$ football vs soccer) and years of participation ($p = 0.012$) predicted recovery duration more modestly, with athletes playing soccer (vs football) and those reporting more (≥10 vs <10) years of experience demonstrating more rapid recovery. Both of these small effects appeared to be accounted for by especially fast recovery in the more experienced cohort (inclusion of sport and years of participation in a single model rendered sport nonsignificant; see appendix e-1 for additional analysis). Concussion history, sex, level of competition/age group, attention-deficit/hyperactivity disorder, learning disability, and headache history were not associated with duration of symptom recovery. Among all preinjury variables considered for inclusion in the multivariable model, only one was independently associated with (longer) symptom recovery: (higher) baseline BSI-18 somatization subscale score (see table 2 and figure 1A).

Postinjury predictors of symptom duration. Results of single predictor models for symptom recovery using acute injury characteristics and acute (24-hour) postinjury variables as predictors are presented in table 3. Two predictors were significant: postconcussive (SCAT3) symptom severity ($p < 0.001$) and BSI-18 somatization score ($p = 0.003$). A trend ($p = 0.023$) was also observed for the BSI-18 global severity index. Consistent with prediction, individuals with high levels of acute (<24 hours) postconcussive (SCAT3) symptoms had a 51% reduction in the hazards of recovery (i.e., slower recovery). Individuals with high levels of acute postinjury (BSI-18) somatic symptoms had a 42% reduction in the hazards of recovery at any given time. Multivariable regression analyses, however, yielded a parsimonious model with only one predictor: 24-hour SCAT3 symptom severity score (HR = 0.49, $p < 0.001$; see table 3 and figure 1B).

Table 2 Preinjury (baseline) predictors of postconcussive symptom duration	HR	95% CI	*p* Value
Demographics/history			
Female	1.12	0.72-1.74	0.610
College (vs high school)	1.04	0.72-1.50	0.827
Grade point average	1.29	0.93-1.80	0.134
WTAR SS	1.01	0.99-1.02	0.406
Household SES	1.00	0.99-1.02	0.697
ADHD	1.36	0.75-2.48	0.310
Learning disability	2.32	0.84-6.38	0.104
Nonmigraine headaches	1.71	0.89-3.29	0.107
Migraine headaches	1.38	0.67-2.84	0.381
No. prior concussions	0.98	0.83-1.15	0.812
Sport			0.067
Football (vs soccer)	1.64	1.07-2.53	0.025
Football (vs other)	0.99	0.60-1.64	0.963
Soccer (vs other)	1.66	0.93-2.98	0.088
Years of participation	1.60	1.11-2.31	0.012
Symptoms			
SCAT3 symptom severity	0.93	0.64-1.36	0.706
BSI-18 somatization	0.58	0.40-0.83	0.003[a]
BSI-18 depression	0.71	0.48-1.06	0.092
BSI-18 anxiety	0.79	0.55-1.12	0.185
BSI-18 global severity	0.76	0.53-1.08	0.128
SWLS total	1.03	0.72-1.47	0.870
Cognition			
SAC total	1.25	0.86-1.83	0.241
ImPACT VERM	1.08	0.70-1.66	0.734
ImPACT VISM	1.04	0.67-1.61	0.865
ImPACT VMS	0.68	0.43-1.06	0.086
ImPACT RT	1.65	1.04-2.61	0.033
Balance			
BESS total	1.06	0.74-1.52	0.759

Abbreviations: ADHD = attention-deficit/hyperactivity disorder; BESS = Balance Error Scoring System; BSI-18 = 18-item Brief Symptom Inventory; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; RT = reaction time; SAC = Standardized Assessment of Concussion; SCAT3 = Sport Concussion Assessment Tool-3; SES = Hollingshead household socioeconomic status score; SWLS = Satisfaction With Life Scale; VERM = verbal memory; VISM = visual memory; VMS = visual motor speed; WTAR SS = Wechsler Test of Adult Reading Standard Score.

[a] $p < 0.01$ in univariate model and retained in the final parsimonious model using preinjury predictors of recovery (only preinjury somatic symptoms were uniquely predictive of symptom duration in multivariate analyses of baseline variables).

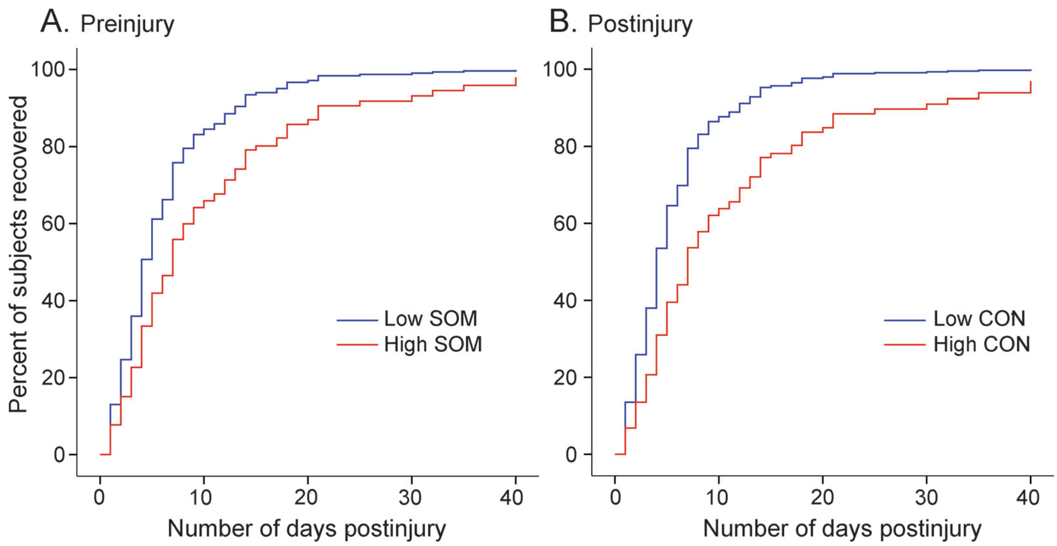
Final multivariate model of symptom duration. Finally, we undertook joint analysis of the 2 key variables identified in the previous models (i.e., preinjury BSI-18 somatization and acute postinjury SCAT3 concussive symptoms, referred to herein as preinjury somatization and postconcussive symptoms, respectively) in order to better understand their relationship in predicting postinjury symptom recovery. As preinjury somatization and postconcussive symptoms were moderately correlated (point biserial r of the dichotomized variables = 0.36; $p < 0.001$; Pearson r of log-transformed continuous scores = 0.51) and has been theorized to be strongly associated with concussive symptom reporting and prolonged recovery in nonsport samples,[18,30,31] we explored the potential for both moderation and mediation between these variables in the prediction of symptom recovery. Moderation was ruled out by a lack of interaction between the 2 predictors ($p = 0.208$). In the resulting model comprised of each variable's main effect, postconcussive symptoms remained a significant predictor at $p = 0.001$ (HR 0.53, 95% confidence interval [CI] 0.37–0.77), while the significance of preinjury somatization fell to $p = 0.035$ (HR 0.66, 95% CI 0.45–0.97).

Mediation of baseline somatic symptoms by acute postconcussive symptoms. Because the reduced predictive value of preinjury somatization after accounting for postconcussive symptoms suggested that postconcussive symptoms might be mediating the effect of somatization on symptom recovery, we next tested this mediation model. To clarify, this analysis tested the degree to which preinjury somatization played an important role in the prediction of postinjury symptom recovery through a direct effect on postinjury symptom reporting vs indirectly (through its effect on postconcussive symptoms). A visual illustration of the hypothesized model is depicted in figure 2. We took 2 complementary approaches to quantifying the mediated effect (i.e., indirect path of preinjury somatization to symptom recovery through postconcussive symptoms). First, we applied a method proposed by Iacobucci[32] that allows for estimation of the significance of a mediated effect when differing statistical models are needed to quantify each model path. This approach involves standardizing each path such that the mediated effect can be computed and interpreted as a z score. Although less frequently used than more traditional mediation analyses, this approach allowed us to retain the Cox model to the degree possible (i.e., for estimation of paths b and c),[32] with logistic regression used to quantify path a (dichotomized postconcussive symptoms predicted from preinjury somatization). Using this approach, the mediated effect ($a \times b$) was significant, $z = 5.64$, $p < 0.001$. Second, we applied a more popular mediation analysis using linear regression of the continuous predictor and outcome measures. As detailed in appendix e-1, this analysis also found strong evidence that postconcussive symptoms mediated the relationship between preinjury somatization and symptom duration.

DISCUSSION

This longitudinal study of SRC revealed 2 main findings about the predictors of

Figure 1 Cumulative incidence of concussion symptom recovery by preinjury somatization (SOM) and acute postconcussive (CON) symptoms



(A) Preinjury/baseline somatic symptoms (SOM_{PRE}) and (B) acute (<24 hours) postconcussive symptoms (CON_{POST}).

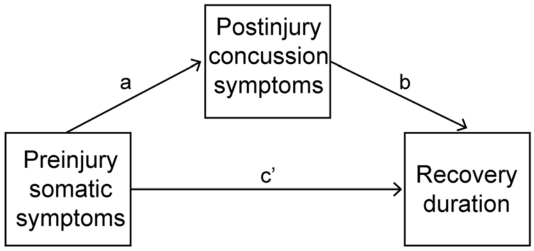
Table 3 Acute (<24 hours) postinjury predictors of symptom duration

	HR	95% CI	*p* Value
Acute injury characteristics			
Loss of consciousness	1.08	0.52–2.22	0.836
Posttraumatic amnesia	0.74	0.43–1.28	0.280
Retrograde amnesia	0.55	0.29–1.07	0.077
Symptoms			
SCAT3 symptom severity	0.49	0.34–0.70	<0.001[a,b]
BSI-18 somatization	0.58	0.40–0.84	0.003[a]
BSI-18 depression	0.84	0.58–1.21	0.346
BSI-18 anxiety	0.84	0.59–1.19	0.324
BSI-18 global severity	0.66	0.46–0.95	0.023
SWLS total	0.99	0.69–1.42	0.966
Cognition			
SAC total	1.20	0.84–1.72	0.314
ImPACT VERM	1.33	0.86–2.07	0.198
ImPACT VISM	1.39	0.89–2.15	0.147
ImPACT VMS	1.11	0.72–1.72	0.641
ImPACT RT	0.97	0.62–1.54	0.911
Balance			
BESS total	1.15	0.79–1.66	0.469

Abbreviations: BESS = Balance Error Scoring System; BSI-18 = 18-item Brief Symptom Inventory; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; RT = reaction time; SAC = Standardized Assessment of Concussion; SCAT3 = Sport Concussion Assessment Tool-3; SWLS = Satisfaction With Life Scale; VERM = verbal memory; VISM = visual memory; VMS = visual motor speed.
[a] $p < 0.01$ in single predictor models.
[b] p Value retained in the final multivariable model (only acute postconcussive symptom burden was uniquely predictive of symptom duration in multivariate analyses of postinjury variables).

postconcussive symptom recovery. First, acute postconcussive symptom severity is the strongest postinjury predictor of recovery time. Second, preinjury somatization symptoms play an important role in recovery by enhancing athletes' experience or reporting of postconcussive symptoms. This represents an important finding for several reasons. First, although clinicians and those researching nonsport/hospital-based concussion samples have long believed that preinjury psychiatric symptoms play an important role in symptom recovery after concussion,[14–18] to our knowledge psychological symptoms have not been measured before injury in prior studies. Our studied capitalized on the methodologic advantage of the sports concussion research model that enables preinjury baseline assessment. Second, that this sample was relatively healthy physically and psychologically highlights the relevance of variability on preinjury psychological traits for recovery even in healthy populations (only one participant reported a prior psychiatric diagnosis of anxiety disorder at the baseline evaluation, and only 3.9% of the sample scored at or above a T score of 65 at baseline on the BSI-18 somatization scale). Finally, the predictive value of preinjury psychiatric symptoms was restricted to somatization symptoms, while other self-reported psychiatric variables of potential relevance (e.g., depression, anxiety) were not strongly associated with postconcussive symptom reporting and recovery. This finding rules out the possibility that acquiescence or general distress inflate symptom ratings broadly and instead points to a specific influence of somatization on postconcussive symptoms and subsequent recovery.

Figure 2 **Mediation model depicts hypothesized causal role of preinjury somatization on postconcussive symptom reporting and recovery**



Analyses supported the model that preinjury somatic symptom reporting influences later postconcussive recovery duration largely indirectly, through its effect on athletes' reporting of acute postconcussive symptoms.

That acute postconcussive symptom burden was the most robust overall predictor of symptom recovery is a significant finding, as it demonstrates convergence with prior research conducted largely on athletes presenting to outpatient specialty clinics, where samples are biased toward unusually prolonged recovery.[10,11,33] Thus, to see convergence between findings of clinic samples and this prospective sample highlights the robustness of this predictor. Although it is not surprising that early postconcussive symptoms predict symptom recovery (given the high degree of content overlap between these 2 variables), it is nevertheless important to recognize, as subjective symptom duration is arguably an essential factor in the maintenance of functional impairments and service utilization after injury.

These findings are consistent with recent trends to recognize and identify the key predictors of concussion incidence and recovery across neurobiopsychosocial domains.[34] Despite efforts to pursue a biomarker of concussion that predicts diagnosis and recovery, it is becoming increasingly recognized that the complexity of the injury requires consideration and integration of variables across levels of analysis, including preinjury function (e.g., cognitive, behavioral, and psychosocial function, genotype), injury specifics and context (e.g., severity, frequency, mechanism), immediate postinjury events (e.g., acute characteristics, diagnosis, treatment), and intervening life events (e.g., life stressors). These study findings point to somatization as a target for future research on the mechanisms underlying premorbid factors relevant to concussion recovery. These findings also have directly translational significance, suggesting that clinicians should assess psychological health factors in the management of athletes recovering from SRC.

A key question is what the construct of somatization, as operationalized by the BSI-18 and more generally, reflects from a psychological, behavioral, and neurobiological standpoint. A number of challenges in conceptualizing and studying somatic symptom disorders have slowed progress in this area relative to some other neuropsychiatric conditions. For example, many diagnoses appear to involve the historically central feature of somatization (i.e., preoccupation with or distress about somatic symptoms beyond what can be explained by recognized medical conditions), resulting in sample heterogeneity across studies.[35] Furthermore, many patients presenting with somatization-like symptoms have comorbid medical conditions, rendering diagnosis of somatization symptoms imprecise. Our finding that somatization has predictive value for an important health outcome in a healthy athlete sample suggests that studying somatic symptoms in athletes could yield important findings about these symptoms apart from medical comorbidities and would be consistent with recent efforts to deemphasize the relative contribution of medical vs psychiatric factors to somatic symptoms.[36] In particular, recent changes to formal (*DSM-V*) criteria for somatic symptom disorders were made to deemphasize the mind/body dualism inherent in considering to what degree somatic symptoms are medically unexplained. Instead, the current criteria emphasize consideration of the symptoms themselves and their associated impairment.[35]

Our suggestion to examine the predictive value of psychological traits like somatic symptoms across healthy and psychiatric samples is also consistent with trends in psychopathology research to study core dimensions of psychopathology across the normal and abnormal ranges as opposed to focusing on traditional diagnostic categories.[37] In addition to being more consistent with data supporting the dimensional structure of many psychiatric constructs,[38] this approach facilitates recruitment of samples with adequate statistical power for researching these psychological factors. Incorporating neuroscience research into these efforts may be particularly valuable for understanding the taxonomy of somatic symptom pathology across levels of analysis and identifying the core underlying mechanisms that contribute to somatic symptom pathology and its role in individuals' responses to and recovery following concussive injury or other adverse medical events.[35,39]

Limitations of this study include the limited base rate of psychopathology and reliance on self-reported recovery time. Although we argue that finding a significant role of somatic symptoms in this sample speaks to the robustness of this finding, it is nevertheless possible that additional preinjury psychological factors would predict recovery in samples with greater variance on those factors. This speaks to the importance of accruing large samples of athletes and more systematically examining somatization and other psychiatric constructs in athlete and nonathlete populations. Additionally, although self-reported recovery time could be skewed by athletes' motivations to return to competition, athletes' responses to this interview question were not available to the athletic trainers who made their return-to-play decisions, and self-reported symptom recovery occurred more slowly than recovery by more objective measures (e.g., neurocognitive functioning) in this sample.[21] Further, as stated earlier, the results of formal neurocognitive effort (MSVT) performance suggested adequate cognitive effort, although no symptom validity scales were administered.

This study revealed that preinjury somatization symptoms and acute postconcussive symptoms play significant roles in the duration of recovery from SRC. This sample yielded evidence for the relevance of preinjury psychiatric symptoms in postconcussive recovery, even among healthy athletes free of serious psychopathology. Future work clarifying the psychological, cognitive, and neurobiological underpinnings of somatization is needed to understand the mechanisms by which this construct contributes to enhanced experience or reporting of postconcussive symptoms and subsequent recovery time.

AUTHOR CONTRIBUTIONS

Dr. Nelson contributed to the management of the study, conceptualization of the manuscript, data analysis and interpretation, drafting of the manuscript, and manuscript revisions. Dr. Tarima contributed to statistical analysis, interpretation, and manuscript revisions. A.A. LaRoche contributed to data collection, study management, data analysis, and manuscript preparation. Drs. Hammeke, Barr, Guskiewicz, and Randolph contributed to study conception and design, study management, interpretation of findings, and manuscript revisions. Dr. McCrea contributed to the conception and design of the study, study management, interpretation of findings, and manuscript revisions. Statistical analyses were performed by Dr. Nelson and Dr. Tarima.

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DISCLOSURE

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This Week's *Neurology*® Podcast

Variability in physician prognosis and recommendations after intracerebral hemorrhage **(see p. 1864)**

This podcast begins and closes with Dr. Robert Gross, Editor-in-Chief, briefly discussing highlighted articles from the May 17, 2016, issue of *Neurology*. In the second segment, Dr. Javier Provencio talks with Dr. Darin Zahuranec about his paper on the variability in physician prognosis and recommendations after intracerebral hemorrhage. Dr. Adam Numis reads the e-Pearl of the week about eye and facial findings in psychogenic nonepileptic seizures. In the next part of the podcast, Dr. Ted Burns focuses his interview with Dr. Chris Holstege on a *Neurology Today* story about the lead crisis in Flint exposing the continuing risk to children nationwide and what neurologists should know and what they can do about it.

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