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Longitudinal trajectories of post-concussive and depressive symptoms in adolescents with prolonged recovery from concussion

Elizabeth Stein, B.A.[1], **Waylon Howard, Ph.D.**[1], **Ali Rowhani-Rahbar, M.D., M.P.H., Ph.D.**[2,3], **Frederick P. Rivara, M.D., M.P.H.**[1,3,4], **Douglas Zatzick, M.D.**[3,5], and **Carolyn A. McCarty, Ph.D.**[1,4]

[1]Seattle Children's Research Institute, Center for Child Health, Behavior and Development

[2]University of Washington, Department of Epidemiology

[3]University of Washington, Harborview Injury Prevention and Research Center

[4]University of Washington, Department of Pediatrics

[5]University of Washington, Department of Psychiatry and Behavioral Sciences

Abstract

Primary Objective: To investigate the symptom trajectories of depressive and post-concussive symptoms (PCS) in slow-to-recover adolescents to understand how the two sets of symptoms are related.

Research Design: We used data from a randomized clinical trial of a collaborative care intervention for post-concussive symptoms to better understand how these two sets of symptoms change in parallel over six months.

Methods and Procedure: PCS and depressive symptom scores for 49 adolescents (ages 11–17) were measured at enrollment and after 1, 3, and 6 months. Latent growth curve modeling for parallel processes was used to simultaneously examine change in PCS and depressive symptoms over time and to evaluate the influence of one change process on the other.

Corresponding Author: Elizabeth Stein B.A., Seattle Children's Research Institute, Center for Child Health, Behavior and Development, PO Box 5371, Suite 400, Mailstop CW8-5A, Seattle, WA 98145-5005, lizstein@me.com Phone: (847)309-5289.
Waylon J. Howard Ph.D., Seattle Children's Research Institute, Center for Child Health, Behavior and Development, PO Box 5371, Suite 400, Mailstop CW8-6, 2001 8th Avenue, Suite 400, Seattle, WA 98121, waylon.howard@seattlechildrens.org
Ali Rowhani-Rahbar M.D., M.P.H., Ph.D., University of Washington, Harborview Injury Prevention and Research Center, Pat Steel Building, 401 Broadway, 325 Ninth Avenue, Seattle, WA 98104, rowhani@uw.edu
Frederick P. Rivara M.D., M.P.H., University of Washington, Department of Pediatrics, 6200 NE 74th St, Ste 110, Seattle, WA 98115-8160, fpr@uw.edu
Douglas Zatzick M.D., University of Washington, Department of Psychiatry and Behavioral Sciences, 325 9th Ave #1C18, Seattle, WA 98104, dzatzick@uw.edu
Carolyn A. McCarty Ph.D., Seattle Children's Research Institute, Center for Child Health, Behavior and Development, PO Box 5371, Suite 400, Mailstop CW8-5A, Seattle, WA 98145-5005, cari.mccarty@seattlechildrens.org

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Main Outcomes and Results: On average, patients enrolled 66 days following injury (IQR 43.5, 88.5). PCS and depressive symptoms were significantly associated at enrollment and over time, and both decreased over the course of 6 months. Higher PCS at enrollment predicted a greater decrease in depressive symptoms over time.

Conclusions: Our results suggest that clinicians should screen for and treat depressive symptoms in patients with high post-concussive symptoms one month following injury.

INTRODUCTION

1 in 5 children will experience a traumatic brain injury by the age of 16 years.[1] While symptoms following mild traumatic brain injury present acutely and typically resolve within two weeks of injury, an estimated 13–29% of injured youth experience the burden of persistent somatic, cognitive, sleep, and psychological symptoms 3 months following injury.[2,3] A distinct symptom trajectory for this “miserable minority” of concussed youth is distinguishable from the typical disease course and expected recovery period.[4] Persistent symptoms such as headache, fatigue, dizziness, and inattention confer functional impairment including executive dysfunction and poor academic performance.[5–7] These post-concussive symptoms (PCS) may result in significant reductions in health-related quality of life, preventing youth from returning to school and normal activities.[8] While it is widely accepted that a variety of biological, psychological, and social factors together contribute to increased risk of protracted symptom duration, the etiology and trajectory of persistent post-concussive symptomatology remains poorly understood.[9,10]

Psychological symptoms, whether newly occurring following the injury or exacerbations of pre-injury “trait” symptoms, are factors which can further alter the recovery course and clinical management of persistent post-concussive symptoms. Pre-existing symptoms including those related to depression and anxiety, ADHD, learning disability and migraine are established ‘modifying factors’ that influence both the risk of sustaining a concussion and the duration of symptoms following injury.[11] Moreover, multiple studies indicate that prior psychiatric history is associated with persistent post-concussive symptom burden following concussive injury in youth and collegiate athletes.[12,13] Premorbid psychological status and family history factors play a significant role in predicting post-injury symptoms and must be taken into account in the assessment and management of sports-related concussion.[14] Furthermore, acquired psychological symptoms commonly occur following injury.[15] Particular attention has been paid to the prevalence of depressive symptoms that co-occur with other persistent post-concussive symptoms. A study of previously healthy adults without psychiatric pre-morbidities at time of study enrollment who sustained mild traumatic brain injury indicated that 62.5% of patients with persistent post-concussive symptoms 1 year after injury had a modifiable psychological risk factor (i.e., depression, possible PTSD, and/or low resilience) at 1 month.[16] Given the frequency of co-occurring mood symptoms, depressive symptoms are commonly included amongst the constellation of post-concussive symptoms.[17]

Despite the frequent onset of depressive symptoms following concussion, there is a paucity of research on depressive symptoms in children following concussion, and it remains unclear

how depression is associated with prolonged recovery from concussion. Two prospective studies found incidence rates of 11% - 21.4% for new onset of depression/anxiety in children within 6 months of concussion.[18,19] A recent prospective cohort study of youth with concussion found that 22% self-reported significant depressive symptomatology following their injury.[20] Considering the prevalence and correlation of depressive symptoms in youth experiencing prolonged recovery from concussion, examination of these symptoms independent of other post-concussive symptoms is merited in order to understand their unique role in modifying the post-concussive symptom trajectory. This is particularly warranted given that depression is often treated differently, and sometimes under separate providers, than other post-injury symptoms.

It is well established that the presence of depressive symptoms is associated with persistent somatic symptoms and reduction in health-related quality of life in patients who experience chronic illness, particularly in traumatic brain injury and regardless of injury severity.[21–23] An understanding of the role of psychological symptoms in protracting chronic illnesses prompted the development of collaborative care treatment models of integrated, early intervention amongst patients with chronic illness to manage both physical and psychological symptoms.[24,25] Moreover, our recent clinical trial of collaborative care treatment for adolescents with post-concussive symptoms demonstrated significant reductions in post-concussive symptoms as well as significant depression remission at 6 months in patients who received the collaborative care intervention targeting modifiable psychological symptoms compared with control patients who received care as usual.[26] While it is accepted that a relationship exists between depressive and persistent post-concussive symptoms, it remains poorly understood how depressive symptoms change over time in conjunction with post-concussive symptoms in patients who are slow to recover from concussion, as there is little research in this area, and none focused on youth.[14,15,27–29] To our knowledge, no previous study has prospectively assessed longitudinal trajectories of post-concussive and depressive symptoms in a pediatric population of patients who remain symptomatic from concussion after the acute recovery period.

The Present Study

The principal purpose of this study was to examine the trajectories of depressive and post-concussion symptoms (PCS) over a six month time period following study enrollment for adolescents with prolonged recovery from sports-related concussion. Specifically, we were interested in identifying the shape of the mean trend for depressive and PCS over time and determining whether both trajectories would significantly improve. We also suspected that change over time in post-concussive symptoms would be positively associated with change over time in depressive symptoms. The secondary goal of the study was to examine if initial levels of depressive symptoms predicted the course of post-concussive symptomatology, or whether initial levels of post-concussive symptoms predicted the course of depressive symptoms. We hypothesized that greater depressive symptom severity at the time of sudy enrollment would predict slower rate of change in post-concussive symptoms.

METHODS

Participants

Participants were adolescents who sustained sports or recreational-related concussion diagnosed by sports medicine or rehabilitation medicine specialists trained in concussion diagnosis and management. Patients were eligible to participate if they incurred an onset or increase in severity in at least three post-concussive symptoms post-injury compared to pre-injury based on parent report on the HBI at least one month following concussive injury. Patients and at least one parent/guardian were required to be able to read and speak English, and to live within commuting distance to the regional children's hospital where assessments and treatment took place. Patients were excluded if they had sustained more severe brain injuries as documented by any abnormalities demonstrated on computer tomography or magnetic resonance imaging. Patients who were actively suicidal, abusing substances, or who reported bipolar or psychotic disorders were also excluded.

Design and Procedure

This study used data from a randomized clinical trial of a collaborative care with embedded cognitive-behavioural therapy intervention for patients with persistent post-concussive symptoms. Forty-nine adolescents enrolled in the study. Following enrollment, patients were randomized to receive intervention (n=25) of collaborative care treatment or care as usual (n=24). Overall, participants ranged in age at the time of entry into the study from 11 to 17 years (M = 14.95, SD = 1.63) and the sample was 65.3% female and 34.7% male. The majority (64.6%) of participants identified as white. Participants completed an intial study assessment and then follow-up symptom rating scales at 1, 3, and 6 months following enrollment. Informed consent and assent was obtained prior to data collection and study procedures were approved by the Seattle Children's Institutional Review Board. Recruitment occurred between October 2014 and July 2015.

Measures

Demographic and injury variables—A variety of demographic variables were collected from each participant including gender, age, race, date of injury, and household income. We collected information on whether the concusion was incurred while playing organized sports or in other, non-organized, recreational activities. Pre-injury history of concerns of anxiety or depression based upon chart review were considered instead of a formal diagnosis for this was only for descriptive purposes. This information was not part of the statistical evaluation of the prospectively collected data.

Depressive Symptoms—Depressive symptoms were reported by youth using the Patient Health Questionnaire (PHQ-9), which includes nine questions, based upon DSM-IV major depression criteria, on a 4-point Likert-type scale ranging from 0 (not at all) to 3 (nearly every day) ranging from 0–27.[30] It has been found to have high sensitivity (89.5%) and acceptable specificity (73%) for the diagnosis of major depression in adolescent populations.[31] High depressive symptoms was defined as scores greater than 10.

PCS—Post-concussive symptoms were assessed by youth using the 27-item Health Behaviour Inventory (HBI) which includes somatic, cognitive, sleep, and psychological domains on a 4-point Likert-type scale ranging from 0 (never) to 3 (often) with a range of 0–60.[32] High PCS was measured with scores greater than 22.

Statistical Analyses

The purpose of this study was, first, to examine the nature of change over time in post-concussive symptoms (PCS) and depressive symptoms across four measurement occasions from study enrollment to six months after enrollment. Based on theory, previous empirical findings,[26] and initial examinations of mean and individual trajectories of PCS (see Figure 1) and depressive symptoms (see Figure 2) we hypothesized that both PCS and depressive symptom trajectories would significantly decrease (show improvement) over the course of the study. To evaluate this hypothesis, a series of latent growth models (LGMs) were specified within the structural equation modeling framework using linear, quadratic, and latent basis models to find the best representation of change.[33,34] An a priori Monte Carlo power simulation based on the particulars of the current data indicate this approach is sufficiently powered (b≥.80) for detecting a non-zero mean slope, given the current sample size of 49.

Initially, a linear growth model was specified with basis scores (i.e., factor loadings) fixed to 0, 1, 3, and 6 on the slope growth factor to reflect the unequal time spacing (in months) between measurement occasions. In this model, departures from linearity in the change process are treated as residual error.[34] Then, we tested a quadratic model using orthogonal polynomial contrasts with linear (i.e., −3, −1, 1, 3) and quadratic (i.e., 1, −1, −1, 1) basis scores to avoid collinearity among the estiamted linear and quadratic growth parameters.[33,34] Finally, we allowed the basis weights to optimally estimate the pattern of change using a latent basis model (i.e., unspecified LGM). For this model, basis scores were anchored at 0 at the time of study enrollment (to set the initial status at baseline) and 1 for the 6-month follow-up assessment. The loadings for the second and third measurement occasions were freely estimated to allow the change process to be optimally estimated.[34] Across all LGM models time differences (in days) between planned and actual observations were incorporated as a time-varying covariates to control for unbalanced time-of-observation within each unequal time-structured wave of measurement (i.e., 0, 1, 3, and 6 months).[34,35]

Our second goal was to examine relationships among growth parameters of different processes (i.e., determine if change over time in one variable is related to change over time in another variable). Unlike multilevel modeling (random coefficient models, mixed-effects models), an extention of LGM for parallel processes allowed for a simultaneous examination of change in PCS and depressive symptoms over time and tests of the influence of one change process on the other.[33] This model combined the best fitting LGM for PCS and depressive symptoms into a single model that contained two sets of intercepts and slopes, one for each repeated-measures variable.[34] A conceptual diagram of the parallel LGM with four time points is shown in Figure 3 where repeated measures of the HBI Youth Report (i.e., PCS score) represent one change process, while repeated measures of the PHQ-9 (i.e., depressive symptom score) represent another change process. We hypothesized that (a) the

higher the intial level of PCS (at time of study enrollment) the higher the initial level of depressive symptoms (at enrollment), (b) the greater the decrease in PCS the greater the decrease in depressive symptoms, (c) the higher the initial level of PCS the greater the decline in PCS over time, and similarly the higher the initial levels of depressive symptoms the greater decrease in depressive symptoms, and (d) the higher the intial level of PCS, the greater the predicted decrease in depressive symptoms over time, and higher levels of depressive symptoms will predict a sharper decrease in PCS.

All models were estimated using multiple imputation in the Mplus 7.3 software program to handle missing data with 50 imputed data sets.[36] Model fit was evaluated using the root-mean-square error of approximation (RMSEA) with the associated 90% confidence interval, the Tucker-Lewis index (TLI), and the comparative fit index (CFI). We considered values less than or equal to .08 acceptable for RMSEA and values greater than or equal to .90 acceptable for the TLI and CFI indices.[37] Structural parameters (correlations and predictive effects) were evaluated for significance ($p < .05$) using a chi-square difference test.[33,38]

RESULTS

Descriptive Statistics

49 adolescents were enrolled and followed over six months. One participant dropped out following enrollment, and one participant withdrew from the intervention given spontaneous resolution of symptoms. Patient characteristics at the time of study enrollment are reflected in Table 1. Correlations, means, and standard deviations for the repeatedly measured PCS and depressive symptom variables are contained in Table 2. As shown in Table 2, PCS and depressive symptoms improve over time. Consistent with prior research, declines in PCS were greatest between the first (at study enrollment) and second (1 month after enrollment) measurement occasion.[39] Declines in depressive symptoms were greatest between the second and third (3 months after enrollment) measurement occasions (see Table 2). Initial depressive symptoms were not related to change in post-concussive symptoms over time. Across all four measurement occasions, missing data was minimal at 1.9%. Little's Missing Completely at Random test indicated that missing data are not likely to introduce bias, χ^2 (14) = 15.01, $p = .378$.

Latent Growth Model

Our first research question focused on the understanding the nature of change in PCS and depressive symptoms over 6 months. Model fit comparisons of univariate LGMs for PCS and depressive symptoms are shown in Table 3.

For PCS the latent basis model was preferred due to model fit, χ^2 (14, n = 49) = 11.14, $p > .05$, RMSEA = .000, 90% CI [.000, .111], TLI = .999, CFI = .999 and parsimony for capturing the non-linear pattern of change. We determined this (and subsequent) model fit to be sufficient despite the wide RMSEA confidence interval because RMSEA has been shown to be positively biased in smaller models (i.e., lower degrees of freedom),[40] some research supports a more liberal cut off value of .10 for detecting a poor fitting model,[41] and all other fit indicies suggest the model is acceptable.[42] The initial level of PCS was 32.47 ($p < .001$)

and growth decreased at an average rate of −17.95 (p = .026) from time of enrollment through the 6 month measurement occasion. The R^2 values for the observed PCS measures were .59 or greater, indicating the growth parameters (and time-varying covariates) explain a meaningful amount of variation. The correlation between the intercept and slope was −.59 (p = .012), indicating participants with a higher PCS score at time of enrollment tended to have a sharper decline over time. Both the intercept (95.88, p = .001) and slope (111.27, p = .005) variances were significant, indicating significant individual differences in PCS at time of enrollment and in the nonlinear change growth factor. This is illustrated in Figure 1, which shows nonlinear intraindividual changes in post-concussive symptoms (PCS) based on HBI youth report and interindividual differences in those changes. Additionally, the estimated basis scores (0, .30, .82, and 1) indicated approximately 30%, 52%, and 18% of the total decrease in PCS scores occurred between time of enrollment and 1 month, 1 to 3 months, and 3 to 6 months, respectively.[33] These estimates suggest a rapid decline through the first three measurement occasions followed by a leveling off.

The latent basis model was also chosen for capturing the non-linear pattern of change of depressive symptoms. The model fit the data well, χ^2 (15, n = 49) = 15.63, p > .05, RMSEA = .000, 90% CI [.000, .140], TLI = .988, CFI = .990. The initial level of depressive symptoms was 9.66 (p = .001) and growth decreased by −5.92 (p = .027) across the time period investigated. The model explained 61% or greater of the variance in the observed depressive symptoms scores. The correlation between the intercept and slope was −.57 (p = .024), indicating participants with a higher depressive symptoms at time of enrollment were associated with a sharper decline over time. Both the intercept (25.13, p = .005) and slope (17.49, p = .007) variances were significant, indicating significant variability around the average intercept and slope growth factor (see Figure 2). Similar to PCS, the estimated time scores for the depressive symptoms trajectory (0, .29, .90, and 1) suggest a sharp decline through the first 3-month period after study enrollment followed by a leveling off.

Latent Growth Model with Parallel Processes—Our second research question evaluated the predictive relations among initial status and change over time. Specifically, we examined whether PCS at at time of study enrollment predicted change in depressive symptoms and whether depression sympoms measured at time of enrollment predicted change in PCS.

To evaluate this question, we specified a LGM with parallel processes by combining the latent basis models for PCS and depressive symptoms into a single model.[33] This model is illustrated in Figure 3. The initial unconditional parallel process LGM fit the data well, χ^2 (38, n = 49) = 30.04, p > .05, RMSEA = .004, 90% CI [.000, .100], TLI = .999, CFI = .999. All growth factors were significant, with both growth slope factors decreasing over time. As shown in Table 4, participants at the first study visit with higher levels of PCS tended to have higher levels of depressive symptoms (r = .58, p = .002). Similarly, over the course of the study, we found decreases in PCS to be strongly related to decreases in depressive symptoms (r = .81, p = .002). Also associations between initial status (intercept) and change over time (slope) within each process were large and negative. Specifically, participants with higher levels of PCS at enrollment tended to decline more in PCS over time (r = −.58, p = .023) and

participants with higher initial levels of depressive symptoms tended to decline more in depressive symptoms over time ($r = -.61$, $p = .042$).

Next, to account for the degree of variance related to participant exposure to collaborative care (n = 25) or usual care (n = 24), we fit a conditional LGM with parallel processes by regressing the slope growth factors for each process on a time-invariant covariate denoting group membership using a multiple indicators, multiple causes (MIMIC) model specification.[35,43–45] This allowed subsequent predictive effects of the slope growth parameter residual variance unexplained by group. While group differences related to collaborative care or usual care were beyond the scope of this investigation, results suggest that although collaborative care participants experienced a faster decline (more positive outcome), the effect was small in magnitude.

Predictive effects were then added to the parallel process LGM model by regressing the PCS growth slope factor on the depressive sympom intercept, and correspondingly regressing the depressive sympom growth slope factor on the PCS intercept. Results indicate a significant effect of initial PCS status predicting change in depressive symptoms over time. Specifically, the higher the PCS level at time of study enrollment, the greater the predicted decrease in depressive symptoms over time (standardized $\beta = -.53$, $\Delta\chi^2 (1) = 6.16$, $p = .013$). Said differently, 25% of the variance in change over time in depressive symptoms could be accounted for by initial PCS status.

DISCUSSION

To our knowledge, this is the first study to prospectively assess the parallel trajectories of post-concussive and depressive symptoms as they change over time throughout the recovery course of pediatric concussion patients receiving treatment. Overall, these findings demonstrate that post-concussive symptoms are closely intertwined with symptoms of depression among slow-to-recover youth, and that youth with high levels of post-concussive symptoms are able to experience significant improvements in depression. This study advances current knowledge about depressive symptoms in the context of concussion, suggesting that depressive symptomatology is not only common in patients who experience persistent symptoms but that it changes over time in tandem with other post-concussive symptoms.

With regard to our first aim, our study showed that improvement in PCS was associated with improvement in depressive symptoms across the recovery period. Patients with high levels of PCS frequently also present with high levels of depression symptoms; however, with a course of active treatment, these symptoms can be significantly decreased. The slopes indicating rate of change of post-concussive symptoms and depressive symptoms were similar, suggesting their co-occurrence. The rate of change for both symptoms was most pronounced in the first three months.

Moreover, we found that depressive symptom severity at enrollment did not significantly predict rate of change over time in post-concussive symptoms as initially hypothesized. Instead, in this clinical sample, patients with more post-concussive symptoms at the first

study visit experienced a sharper decline in depressive symptoms over time. This finding might be explained by the fact that patients with higher initial level of symptoms had more room to improve. Given that patients with more post-concussive symptoms at time of study enrollment experienced a sharper decline in depressive symptoms over time, depression remains a tractable area of intervention in treatment of post-concussive symptoms. Our findings suggest that youth with persistent post-concussive symptoms can make appreciable improvements in their depressive symptoms.

As previous studies have determined, patients with elevated post-concussive symptoms more than 1 month in duration following injury may be particularly subject to benefit from treatment that targets co-occurring psychological symptoms given the high prevalence of depression symptoms in this population and our finding that post-concussive and depression symptoms are closely related as they change over time.[16] This study expounds on previous literature that links depression to chronic illness such as with chronic pain and post-traumatic stress disorder.[46]

There were several limitations to our study, including the small sample population drawn from a randomized clinical trial. It is important to consider that this was a treatment-seeking sample that was recruited from Rehabilitation and Sports Medicine clinics, and the improvement in symptoms may have reflected gains that resulted from such treatment. We did not include group-specific results in this study as our study was not sufficiently powered to examine differences in associations by treatment group. Furthermore, the hypotheses evaluated in the current models required moderate to large effect sizes, therefore, non-significant effects may be due to sample size limitations. Time after injury at enrollment was not standardized across participants due to the pragmatic approach that investigators used to simulate real-world clinic conditions. We could not determine causality from our analysis and therefore cannot say whether the presence of depressive symptoms directly causes persistent post-concussive symptoms nor can we determine from our data whether psychological problems are responsible for chronic post-concussive symptoms or if psychological problems result from chronic post-concussive symptomatology. Psychological symptoms, including depression, are commonly reported in youth following concussion, as others have suggested.[47] Our findings suggest that depression and post-concussive symptoms present together and change over time in parallel.

It is worth noting that we used the PHQ-9 to evaluate depressive symptomatology and acknowledge that there is some overlap with the assessment of post-concussive symptoms on 3 questions: 1) trouble falling or staying asleep, or sleeping too much; 2) feeling tired or having little energy; 3) trouble concentrating on things. There were several strengths to our study, including the prospective design that enabled us to longitudinally assess changes in symptom domains over time and the use of sophisticated parallel process linear growth models. Our study provides empirical data at multiple time points for a clinical pediatric patient population for which there is little published data, allowing us to appreciate a more comprehensive understanding of the symptom trajectories across domains.

These data were derived from randomized clincial trial of collaborative care treatment that simultaneously targeted co-occurring psychological symptoms alongside somatic, cognitive,

and sleep complaints with cognitive-behavioural therapy, care management, and psychotropic medication consultation.[29] The findings in our prior report suggest that early intervention targeting both physical and psychological symptoms may be critical to accelerating recovery from persistent post-concussive symptoms, as the collaborative care group demonstrated a more rapid rate of symptom reduction as well as symptom reduction gains that persisted at 6 months. Following the publication of our trial, the evidenced-based guidelines for the care of youth with persistent symptoms were updated to recommend a collaborative care model which can simultaneously address somatic and psychological symptoms following concussion.[29] Targeted approaches to psychological symptoms in pediatric patients with persistent post-concussive symptoms is a promising area of intervention with evidence-based treatment options including cognitive-behavioural therapy and psychotropic medication.[48,49] Our results suggest that clinicians should systematically screen for and treat depressive symptoms in their patients with high post-concussive symptoms one month following injury given that psychological symptoms are modifiable factors that present with and change over time in parallel to other difficult-to-treat somatic symptoms.

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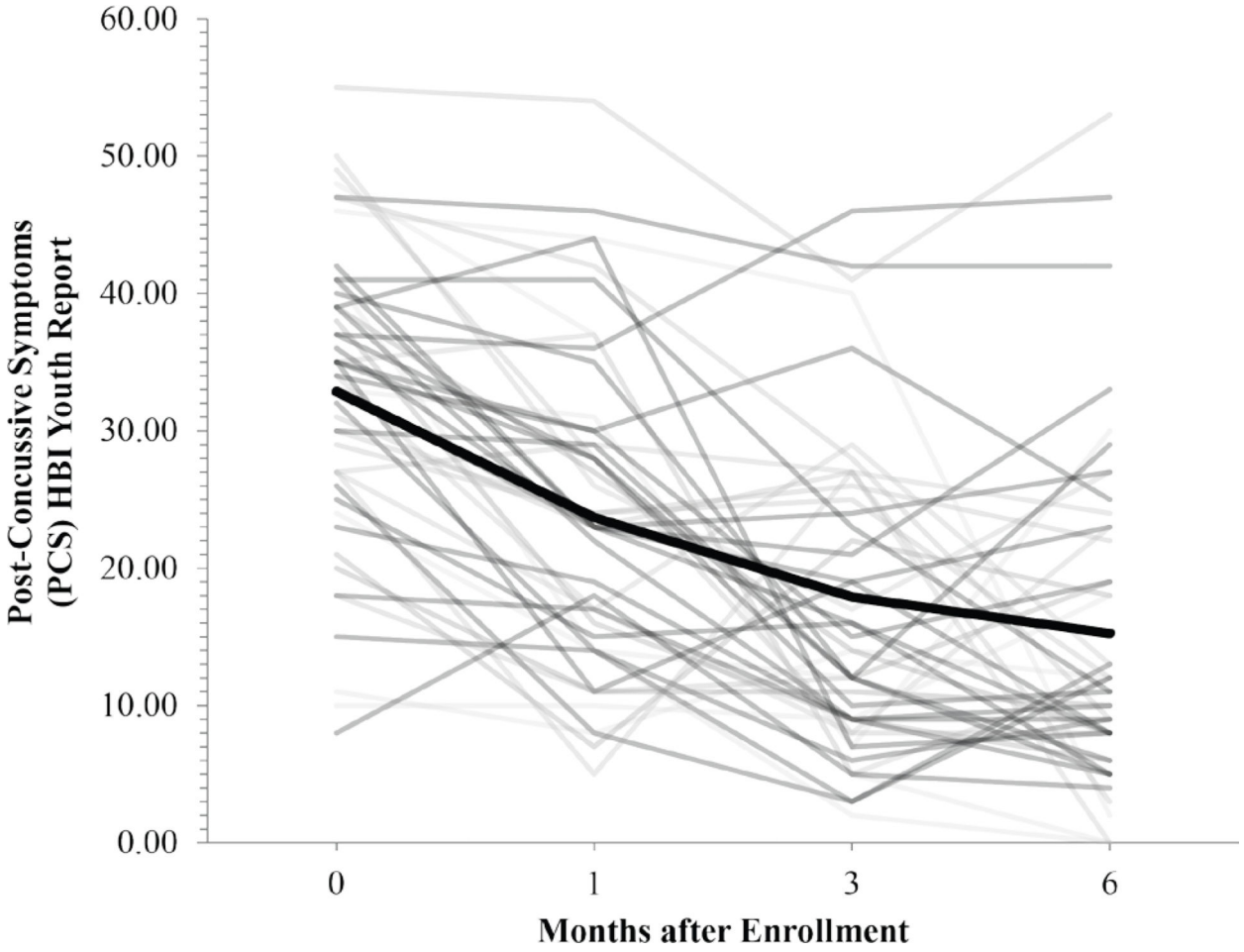

Figure 1.

A plot of growth in post-concussive symptoms (PCS) HBI youth report illustrating nonlinear intraindividual changes and interindividual differences in those changes.

Note: Grey lines represent individual trajectories; black line depicts average trajectory.

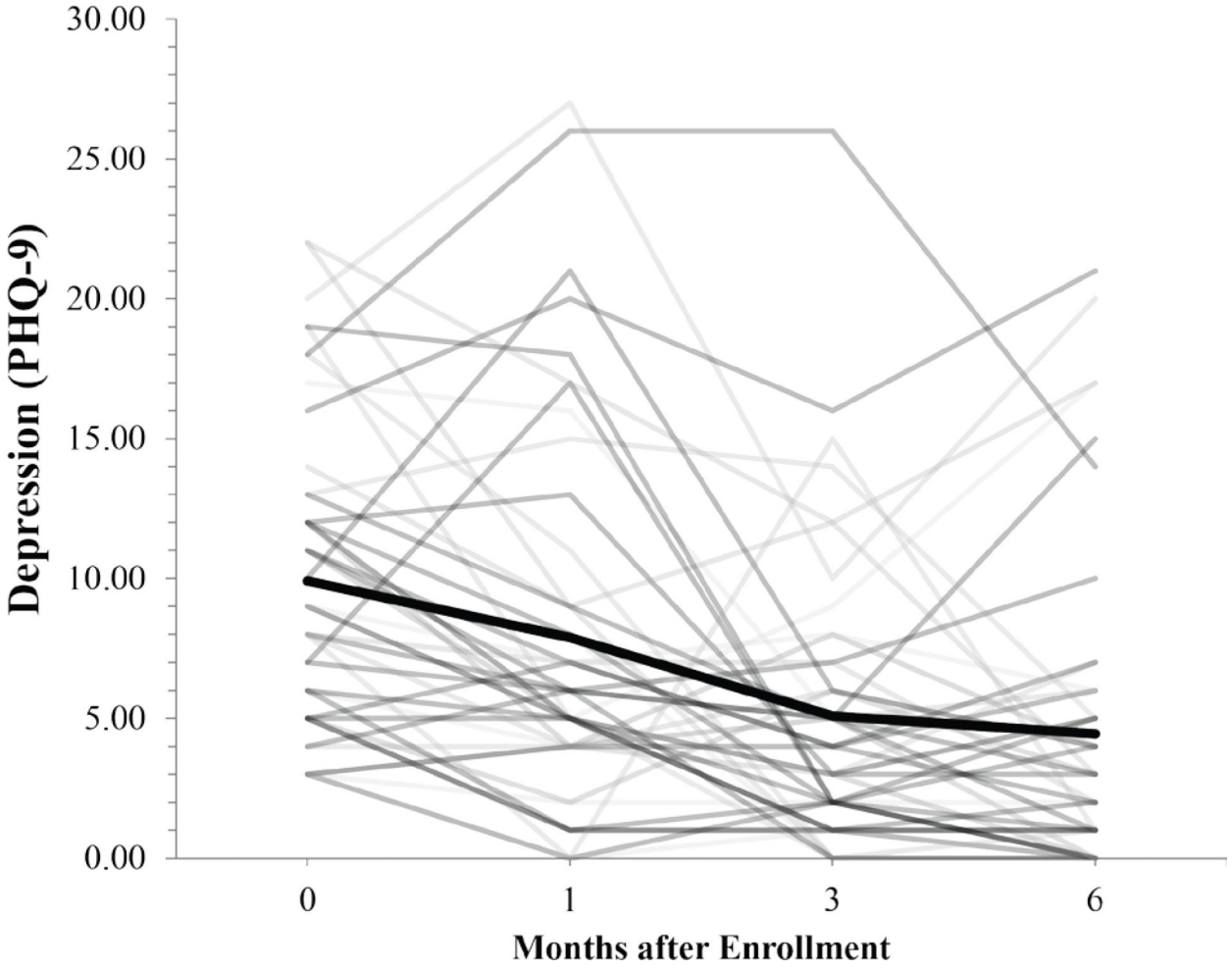

Figure 2.

A plot of growth in depression symptoms (PHQ-9) illustrating nonlinear intraindividual changes and interindividual differences in those changes.

Note: Grey lines represent individual trajectories; black line depicts average trajectory.

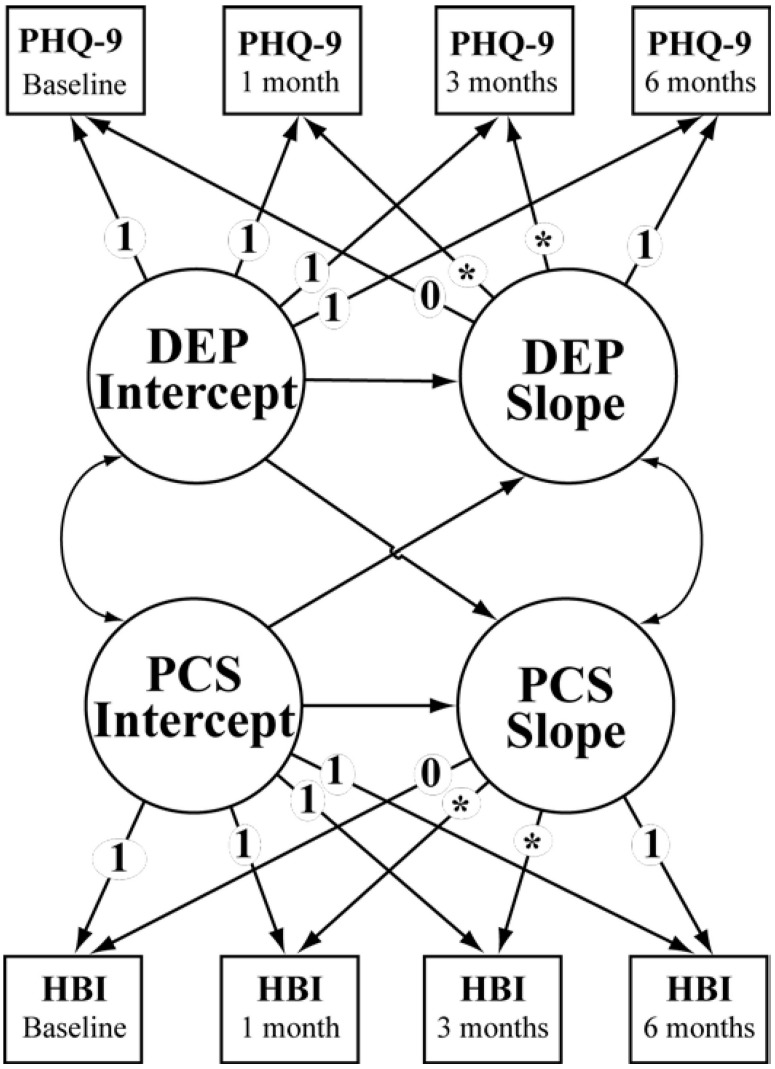

Figure 3.

A conceptual path diagram of a latent growth curve model with parallel processes representing simultaneous growth in PCS and depressive symptoms using the latent basis (freely estimated basis loadings) specification for nonlinearity.

Note: The parallel process model combined each individual LGM into a single model that contained two sets of intercepts and slopes, one for each repeated-measures variable. Circles represent latent variables, squares are measured variables (repeated measures), single headed arrows are path coefficients (regression weights), double headed arrows are variances or covariances. Regression weights related to the intercepts are fixed at a value of 1. Regression weights related to the slopes were fixed at 0 for the first measurement occasion and 1 at the final measurement occasion. The remaining regression weights (i.e., basis weights) denoted by * were freely estimated.

Not all paths are displayed for clarity of presentation.

Table 1.

Descriptive statistics on demographic, pre-injury health concerns, injury type, and clinical profile at time of study enrollment.

Characteristics	Patients %(n = 49)
Demographic	
Age (years), mean (SD	15.0 (1.6)
Female	65.3% (32)
Race	
White	75.5 (37)
Asian/PI	8.2 (4)
Black	2.0 (1)
Other	14.3 (7)
Ethnicity	
Hispanic	8.2 (4)
Non-Hispanic	91.8(55)
Annual household Income	
<=$50K	20.4 (10)
$50–100K	26.5 (13)
>$100K	46.9 (23)
Unknown	6.2 (3)
Pre-Injury Health Concerns	
Prior ADHD/LD Diagnosis	18.8 (9)
History of Anxiety/Depression	37.5 (18)
History of Previous Concussion	52.1(25)
History of Headache	18.8 (9)
Injury Type	
Sports-Related	57.5(29)
Recreational Related	42.5(20)
Clinical Profile at Baseline	
Median days since concussion (IQR)	66.0(45.0)
High Post-concussive Symptoms[a]	83.7
High Depressive Symptoms[b]	40.8

Table 2.

Correlation matrix with means and standard deviations for the indicators of post-Concussive Symptoms (PCS) and depression symptoms (DEP) across time ($N = 49$).

	PCS0	PCS1	PCS3	PCS6	DEP0	DEP1	DEP3	DEP6
PCS0	1.000							
PCS1	0.722	1.000						
PCS3	0.362	0.469	1.000					
PCS6	0.213	0.355	0.518	1.000				
DEP0	0.634	0.570	0.300	0.320	1.000			
DEP1	0.405	0.572	0.317	0.351	0.679	1.000		
DEP3	0.113	0.223	0.615	0.523	0.325	0.502	1.000	
DEP6	0.146	0.291	0.418	0.800	0.363	0.549	0.613	1.000
Mean	32.857	24.351	17.429	15.377	9.796	7.764	5.081	4.425
SD[1]	10.838	11.681	11.594	12.004	5.384	6.505	5.050	5.527

[1] SD represents the standard deviation.

Table 3.

LGM model fit comparisons for PCS and depressive symptoms.

Model	χ^2	df	p	RMSEA	RMSEA 90% CI	TLI	CFI
Post-concussive symptoms (PCS) HBI youth report							
Linear LGM	25.12	14	<.05	0.127	.035-.206	0.724	0.785
Quadratic LGM[1]	11.14	14	>.05	0.000	.000-.111	0.999	0.999
Latent Basis LGM[1]	13.25	15	>.05	0.000	.000-.120	0.999	0.999
Depression symptoms (DEP)							
Linear LGM	24.34	14	<.05	0.123	.024-.202	0.783	0.832
Quadratic LGM[1]	16.93	14	>.05	0.065	.000-.160	0.939	0.952
Latent Basis LGM[1]	15.63	15	>.05	0.029	.000-.140	0.988	0.990

[1]Error variance was equated over time to address convergence issues.[35]

Note: χ^2 = chi-square value; df = degrees of freedom; p = p-value, RMSEA = Root Mean Square Error of Approximation; TLI = Tucker-Lewis Index; CFI = Comparative Fit Index

Table 4.

Parallel process model parameter estimates.

Parameter	Est.	SE	p-value
Depression symptoms (DEP)			
Mean intercept	9.60	.87	.002
Mean slope	−5.27	.94	.028
Intercept variance	25.37	6.83	.004
Slope variance	17.51	7.65	.009
Intercept/slope covariance	−12.25	5.92	.007
Post-concussive symptoms (PCS)			
Mean intercept	32.31	6.83	< .001
Mean slope	−17.68	2.15	.028
Intercept variance	97.71	27.27	.001
Slope variance	115.57	41.80	.005
Intercept/slope covariance	−64.29	28.53	.012
Curve Covariances			
Intercept covariance	34.58	11.57	.002
DEP intercept / PCS slope	−14.10	12.31	.009
PCS intercept / DEP slope	−24.49	11.33	.009
Slope covariance	36.23	15.19	.005
Curve Correlations			
Intercept covariance	0.58		
DEP intercept / PCS slope	−0.26		
PCS intercept / DEP slope	−0.59		
Slope covariance	0.81		