Relationship Between Concussion and Neuropsychological Performance in College Football Players

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Context Despite the high prevalence and potentially serious outcomes associated with concussion in athletes, there is little systematic research examining risk factors and short- and long-term outcomes.

Objectives To assess the relationship between concussion history and learning disability (LD) and the association of these variables with neuropsychological performance and to evaluate postconcussion recovery in a sample of college football players.

Design, Setting, and Participants A total of 393 athletes from 4 university football programs across the United States received preseason baseline evaluations between May 1997 and February 1999. Subjects who had subsequent football-related acute concussions (n = 16) underwent neuropsychological comparison with matched control athletes from within the sample (n = 10).

Main Outcome Measures Clinical interview, 8 neuropsychological measures, and concussion symptom scale ratings at baseline and after concussion.

Results Of the 393 players, 129 (34%) had experienced 1 previous concussion and 79 (20%) had experienced 2 or more concussions. Multivariate analysis of variance yielded significant main effects for both LD ($P<.001$) and concussion history ($P = .009$), resulting in lowered baseline neuropsychological performance. A significant interaction was found between LD and history of multiple concussions and LD on 2 neuropsychological measures (Trail-Making Test, Form B [$P = .007$] and Symbol Digit Modalities Test [$P = .009$]), indicating poorer performance for the group with LD and multiple concussions compared with other groups. A discriminant function analysis using neuropsychological testing of athletes 24 hours after acute in-season concussion compared with controls resulted in an overall 89.5% correct classification rate.

Conclusions Our study suggests that neuropsychological assessment is a useful indicator of cognitive functioning in athletes and that both history of multiple concussions and LD are associated with reduced cognitive performance. These variables may be detrimentally synergistic and should receive further study.

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THE MANAGEMENT OF MILD TRAUmatic brain injury (MTBI; eg, concussion, defined as a traumatically induced alteration in mental status not necessarily resulting in loss of consciousness) in athletics is currently one of the most compelling challenges in sports medicine. Despite the high prevalence[1] and potentially serious outcomes[2,3] associated with concussion, systematic research on this topic is lacking. Many sports medicine practitioners are not satisfied with current return-to-play and treatment options, which do not appear to be evidence based.[4-6] There is also little research examining whether long-term cognitive morbidity is associated with concussion. Past research with nonathletes revealed that repeated concussions appear to impart cumulative damage, resulting in increasing severity and duration with a second MTBI occurring

See also pp 954, 958, 971, 974, and 989.

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within 48 hours.[7] No data were presented which addressed more long-term outcomes.

Although survey data have shown that a prior history of head injury increases the risk for sustaining subsequent MTBI,[8] other potential risk factors associated with sports-related concussion have not been identified. Learning disability (LD), the etiology of which is presumably secondary to central nervous system dysfunction,[9] refers to a heterogeneous group of disorders manifested by difficulties in the acquisition and use of listening, speaking, writing, reading, reasoning, or mathematical abilities and which is traditionally diagnosed in early childhood.[10,11] The incidence of diagnosed LD is 11.8% in the general university population.[12] However, no study to date has addressed whether LD may represent a risk factor (such as that seen with prior head injury) for poor outcome following sports-related MTBI in college athletes.

Previous research has outlined the reliability, validity, and sensitivity of neuropsychological tests in assessing the specific cognitive areas associated with MTBI in the general population.[13-15] To date, 3 published studies have examined the use of neuropsychological testing in US football players.[16-18] The only multicenter study[16] was conducted in the mid-1980s and was designed to address the acute effects of concussion.

The current study was designed to address 2 issues: first, to investigate whether a relationship exists between prior concussion and diagnosed LD among college football players and determine the influence of these variables, in isolation and combination, on baseline neuropsychological performance; and second, to evaluate the use of a neuropsychological test battery in diagnosing concussion and delineating recovery of cognitive function following MTBI in athletes.

METHODS

Subjects

Participants in this study consisted of 393 male college football players from 4 Division IA football programs: Michigan State University, East Lansing (n = 119); the University of Florida, Gainesville (n = 106); the University of Pittsburgh, Pittsburgh, Pa (n = 85); and the University of Utah, Salt Lake City (n = 83).

At the initial preseason baseline session, the following self-reported data were collected: age, playing position, SAT/ACT scores (Scholastic Aptitude Test/American College Testing, ie, college entrance examination scores), history of LD, neurological history (eg, central nervous system neoplasm or epilepsy), history of psychiatric illness (eg, depression and/or mania or anxiety), history of alcohol and/or drug abuse, prior sports played, and history of concussion. Educational records at each institution were used to verify a documented history of diagnosed LD. A standardized concussion history form was administered at baseline to obtain detailed information regarding previous concussions, year of concussion, description of incident, nature and duration of relevant symptoms (eg, confusion and/or disorientation, retrograde and/or anterograde amnesia, and loss of consciousness), neuroimaging results (if any), and days lost from participation in football (if any). Athletes who reported amnesia were asked to provide any known collateral information from the athletic trainer, sports-medicine physician, or other source familiar with the details of the incident. All previous concussions were classified using the practice parameter of the American Academy of Neurology.[19]

Protocol and Outcome Measures

Preseason Baseline Evaluation. Appropriate review for research with human subjects was granted separately from the 4 institutions at which the participants were enrolled. Each participant provided written informed consent for voluntary participation. All data collection was completed by the research team of clinical neuropsychologists (clinicians with PhDs or doctoral-level students) or team physicians or athletic trainers who were thoroughly trained in the use of the measures. Each examiner was required to attend a 2-hour workshop and was supervised during test adminstration (by M.W.C.) to facilitate the appropriate standardized administration of the test battery. All measures were administered and scored in a standardized manner to minimize differences between test administrators and institutions. Project investigators trained in neuropsychological assessment completed all data scoring and interpretation.

Baseline data collection at 3 universities (Michigan State University, University of Pittsburgh, and University of Florida) was completed prior to the 1997/98 and 1998/99 football seasons during the months of May to August. Baseline data collection at the University of Utah occurred during February 1999 for the 1999/2000 season (only baseline data from the University of Utah were used for analyses). Approximately 95% of all roster football players (scholarship and scout team players) voluntarily participated in the project. At these baseline sessions, demographic and player history information was obtained via interview.

Each athlete was then administered a battery of neuropsychological tests (approximately 30 minutes in length) that is used by the National Football League.[17,20] Tests in the battery were the Hopkins Verbal Learning Test (HVLT; verbal learning, delayed memory); Trail-Making Tests, Forms A and B (Trails A and Trails B; visual scanning and executive functioning); Digit Span Test (attention and concentration); Symbol Digit Modalities Test (SDMT; information processing speed); Grooved Pegboard Test, dominant and nondominant hand (bilateral fine motor speed); and the Controlled Oral Word Association Test (COWAT; word fluency). This test battery, described in detail elsewhere,[17] was constructed to evaluate multiple aspects of cognitive functioning. In addition to neuropsychological testing, athletes also completed the Concussion Symptom Scale[17] to assess a baseline level of self-reported symptoms. This Likert scale

consists of 20 symptoms commonly associated with concussion (eg, headache, dizziness, and trouble falling asleep), with symptoms ranging from none (score, 0) to severe (score, 6).

Postconcussion Evaluation. Athletes who sustained a concussion during the course of a season underwent serial neuropsychological evaluations following the incident (within 24 hours of the incident, and at days 3, 5, and 7 postinjury). Concussion was defined according to the American Academy of Neurology practice parameter.[19] Thus, players experiencing a traumatically induced alteration in mental status, not necessarily resulting in a loss of consciousness, were included. Athletic trainers initially identified the majority of suspected concussions, and respective team physicians performed the examinations and made the final decisions. Once the diagnosis was established, neuropsychological testing was administered as soon as possible following injury (within 24 hours in all cases). The neuropsychological tests and self-report inventory used in the postinjury phase were identical to those used at baseline, although alternate and reliable forms of the HVLT and COWAT were administered to minimize learning effects associated with these measures.

Football players from within the sample served as controls. Control athletes were matched with athletes who sustained concussion according to ACT/SAT scores, history of LD, history of previous concussion, institution, and playing position. In addition, to control for exertion, each control athlete was tested within the same time frame as the athletes who experienced concussion (eg, following a game or practice). Within the context of these variables, it was possible for controls to be matched to more than 1 player with concussion. No control athlete experienced a concussion during the course of the study. Controls were excluded from further study.

Data Analysis

Data from the 4 universities were pooled and analyzed using *Statistica* Version 5.1 statistical software for Windows.[23] To explore the relationship between prior history of concussion, diagnosis of LD, and neuropsychological baseline performance, multiple analysis of variance (MANOVA) was performed. Concussion history (no prior concussion vs 1 vs ≥2 concussions) and LD (positive or negative diagnosis) were entered as independent variables, and cognitive and symptom total scores were entered as dependent measures. The MANOVA design was selected to allow an analysis of performance differences between the athletes with different concussion and LD histories, across multiple neuropsychological domains. This design also permitted an analysis of possible interaction effects between concussion and LD histories.

For in-season (postconcussion) data, a discriminant function classification analysis was conducted to determine the accuracy of the neuropsychological test battery in separating athletes with concussions from control athletes within 24 hours of concussion. The 8 tests constituting the neuropsychological test battery were used as predictor variables, and membership in the group with concussions or control group was used as the dependent (grouping) variable.

To provide preliminary information regarding the recovery pattern of athletes with concussions relative to the control group and to their own baseline performance, standard scores were created to convert the selected neuropsychological test scores to a common metric. These standard scores were constructed so that baseline performance for each group would have a mean of 100 and SD of 15.[21] Group differences of one-half SD (7.5 standard score units) are considered to reflect at least a moderate difference between the means.[22] Any deviation from 100 indicates a change in performance relative to baseline for each group. The recovery pattern of players who sustained concussion across different time intervals was evaluated by standardizing all neuropsychological test results and comparing performance of the athletes with concussion with controls' performance within 24 hours, and at 3, 5, and 7 days postinjury.

RESULTS

Demographic Data and Concussion History

The multiuniversity sample included 393 male football players with a mean (SD) age of 20.4 (1.7) years and 2.6 (1.3) mean (SD) years in college. Forty-six percent of the sample was African American, 48% European American, 4% Polynesian American, 1% Asian American, and 1% Hispanic American. Of the 393 players, 6% (n = 25) were quarterbacks; 8% (n = 33), running backs; 13% (n = 52), wide receivers; 16% (n = 64), offensive linemen; 6% (n = 23), tight ends; 17% (n = 67), defensive backs; 16% (n = 61), defensive linemen; 13% (n = 48), linebackers; and 5% (n = 20), kickers.

Of the players completing the ACT examination to qualify for college admission (n = 180), the mean (SD) score was 20.0 (1.7). Of those qualifying with the SAT (n = 200), the mean (SD) score was 952.9 (149.1). College admission scores were missing for 13 individuals. Three players in the sample reported a documented history of diagnosed psychiatric illness (eg, bipolar disorder and major depression). These players completed the baseline evaluation, but were excluded from further study. No player in the sample reported a diagnosis of major neurological disorder or history of abuse of alcohol or other drugs.

Forty-six percent (n = 179) of the sample reported no prior history of concussion, 34% (n = 129) reported experiencing 1 concussion of any grade, and 20% (n = 79) reported a history of 2 or more sustained concussions (range, 2-10) of any grade. A significant relationship was found between total years participating in football and total number of concussions sustained ($r = 0.15$; $P \leq .02$). Quarterbacks (17 of 25) and tight ends (15 of 23) had the the highest rates of prior concussion (68% and 65%, respectively). Running backs–fullbacks (11 of 33) and kickers-punters experienced the lowest rates of prior concussion (33% and 46%, respectively).

The prevalence of LD within the total sample of 393 athletes was 13.5% (n = 53). Of the players with no history of concussion (n = 179), 10.6% (n = 19) had a diagnosed LD; of those who had experienced 1 prior concussion (n = 129), 14.7% (n = 19) had diagnosed LD, and of those who had experienced multiple concussions (n = 79), 19.0% (n = 15) had a diagnosed LD. Although these data suggest a possible trend between history of LD and history of multiple concussions, this relationship was not statistically significant ($\chi^2 = 3.74$; $P = .15$).

Previous Concussions, LD History, and Baseline Neuropsychological Performance

The MANOVA yielded significant main effects for both LD ($F = 4.57$; $P < .001$) and concussion history ($F = 1.91$; $P = .009$) on neuropsychological test results, which indicated that both of these variables were significantly related to overall neuropsychological performance. The interaction of LD and concussion history was not significant ($F = 1.17$; $P = .28$). A follow-up series of univariate F tests was completed to identify the specific neuropsychological measures that accounted for the significant MANOVA. Tests for the LD main effect were Trails B ($F = 15.98$; $P < .001$); SDMT ($F = 22.9$; $P < .001$); COWAT ($F = 11.6$; $P < .001$); and Hopkins delayed memory ($F = 11.8$; $P < .001$). For the history of concussion main effect, significant tests included Trails B ($F = 6.1$; $P = .002$); SDMT ($F = 7.8$; $P < .001$); and total symptoms reported ($F = 4.6$; $P = .01$).

To evaluate concussion group differences on the neuropsychological tests, additional post hoc analyses were conducted using the Tukey Honest Significant Difference test for unequal subjects.[24] The TABLE presents the group means (SDs) for athletes. The group with no history of concussion reported fewer symptoms than both the single concussion group ($P = .04$) and the multiple concussion group ($P < .001$) on the concussion symptom inventory. Baseline symptoms increased as the number of concussions increased. On Trails B, the multiple concussion group performed significantly worse at baseline than the group with no history of concussion ($P = .02$) and the single concussion group ($P < .001$). Baseline data also differed significantly on the SDMT with the multiple concussion group performing worse than both the group with no history of concussion ($P = .008$) and the single concussion group ($P < .001$). These findings are not attributed to preexisting group differences in terms of aptitude as the multiple concussion group had higher SAT and ACT scores than did the group with no history of concussion and the single concussion group. The table presents demographic and neuropsychological test data for the group with LD and the group without LD.

To investigate the interplay between concussion history and LD on baseline neuropsychological test performance, a concussion history and LD

Table. Neuropsychological and Symptom Baseline Results for Concussion and Learning Disability Groupings[a]

	Concussion History, No.			LD History		Total Group Means
	0	1	≥2	No	Yes	
Athletes, No.[b]	179	129	78	332	54	**386**
Mean SAT/ACT scores[c]	928.8/20.2	967.5/21.3	978.9/20.9	964.7/21.1	881.0/18.5	952.9/20.7
Total years in football	8.1 (3.1)	8.6 (2.9)	9.5 (2.4)	8.5 (3.1)	9.2 (2.1)	8.6 (2.8)
Symptom Total[d]	7.9 (10.9)	10.6 (12.1)	15.3 (17.1)[e f]	9.2 (12.3)	12.4 (16.5)	10.3 (12.6)
Hopkins Total[g]	24.6 (3.7)	24.9 (4.0)	24.1 (4.6)	25.0 (4.1)	24.1 (3.4)	24.6 (4.0)
Hopkins Delay[h]	8.0 (1.9)	8.1 (2.1)	7.7 (2.1)	8.4 (2.0)	7.4 (2.0)[i]	7.8 (2.0)
Trail-Making Test, Form, A	21.2 (6.0)	19.9 (5.2)	22.1 (6.6)	21.0 (5.8)	21.0 (6.8)	21.0 (5.9)
Trail-Making Test, Form B	55.3 (15.4)	51.2 (16.4)	62.6 (23.2)[f j]	51.2 (15.5)	60.7 (26.3)[i]	55.4 (17.3)
Digit Span Test	15.3 (3.8)	16.3 (4.1)	16.0 (3.8)	16.3 (3.9)	15.4 (3.9)	15.8 (3.9)
Symbol Digit Modalities Test	56.9 (8.7)	59.2 (8.9)	52.8 (9.2)[f j]	59.4 (8.6)	53.6 (9.1)[i]	56.8 (8.9)
Grooved Pegboard Test, Dominant Hand	65.6 (10.0)	68.0 (10.8)	69.3 (12.4)	66.7 (10.9)	68.2 (10.2)	67.1 (10.7)
Grooved Pegboard Test, Nondominant Hand	72.3 (11.7)	74.2 (11.9)	75.2 (12.4)	73.0 (12.0)	74.8 (11.0)	73.5 (11.9)
Controlled Oral Word Association Test Total	37.0 (9.4)	38.0 (8.6)	37.6 (10.0)	39.8 (9.1)	35.2 (9.8)[i]	37.5 (9.3)

[a] Data are presented as mean (SD) unless otherwise indicated. Higher scores represent better performance for Hopkins Total, Hopkins Delay, Digit Span, Symbol Digit Modalities, and Controlled Oral Word Association Tests. Lower test scores represent better performance for Symptom Total; Trail-Making Tests, Forms A and B; and Grooved Pegboard Test, Dominant Hand and Nondominant Hand. LD indicates Learning Disability.
[b] Of the 393 athletes, 7 were excluded because baseline data were incomplete.
[c] SAT indicates Scholastic Aptitude Test; ACT, American College Testing.
[d] Symptom Total indicates total score from the concussion symptom scale (see Methods section).
[e] *P* value for post hoc comparison (no concussion vs 1 concussion) was <.05 using Tukey Honest Significant Difference (THSD).
[f] *P* value for post hoc comparison (no concussion vs ≥2 concussions) was ≤.01 using THSD.
[g] Hopkins Total is equivalent to trials 1-3 of the Hopkins Verbal Learning Test (HVLT).
[h] Hopkins Delay is equivalent to the total delay score on the HVLT.
[i] *P* value for post hoc comparison between LD and no-LD groups was ≤.01 using THSD.
[j] *P* values for post hoc comparison (1 concussion vs ≥2 concussion) was ≤.05 using THSD.

interaction term was constructed. Univariate F tests for all 10 neuropsychological variables demonstrated statistically significant interactions for Trails B ($F = 4.99$; $P = .007$) and SDMT ($F = 4.74$; $P = .009$). In both cases, athletes with a history of multiple concussions and LD performed significantly worse than did athletes with no history of LD who had experienced multiple concussions (FIGURE 1).

In-Season Concussions

Nineteen players in the study sample were diagnosed by team medical staff as sustaining a concussion during the course of the 1997-1999 seasons. Thirteen individuals sustained a grade 1 concussion (mental status abnormalities resolved within 15 minutes), 4 athletes sustained a grade 2 concussion (mental status abnormalities that lasted longer than 15 minutes, but resolved within 45 minutes), and 2 athletes sustained a grade 3 concussion (brief [approximately 5-10 seconds] loss of consciousness). The time between baseline testing and in-season concussions ranged from 5 weeks to nearly 18 months.

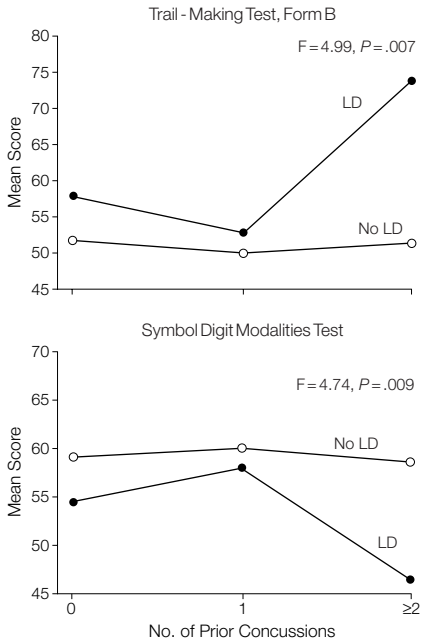
The group with concussions consisted of 16 athletes who had completed all of the neuropsychological measures (3 athletes were excluded because they either missed a testing session or failed to complete at least 1 of the tests at 1 session). The control group consisted of 10 matched control athletes. Given the different numbers of subjects in the 2 groups, a priori probabilities for group membership were set at 62% for the group with concussions and at 38% for the control group. Based on testing conducted 24 hours after injury, the neuropsychological test battery resulted in an overall 89.5% correct classification rate for the 2 groups (87.5% correct classification for players with concussions; 90% correct classification for the control subjects). Current data provide preliminary support regarding the sensitivity of our test battery in classifying athletes with concussions vs controls. Although the number of subjects in each group is small, comparison of the magnitude of change in performance for each group is presented in FIGURE 2. Performance of the athletes with concussions was noticeably worse (approximately 1 SD) than the control group on Hopkins Total (trials 1-3 of the HVLT) and delayed memory (total delay score on the HVLT) within 24 hours after injury. Moderate differences in performance persisted between groups until at least day 5 postconcussion.

COMMENT

The results of this study suggest that history of concussion and LD are independently related to lower baseline cognitive performance within a large, multiuniversity sample of football players. The negative impact of LD on neurocognitive functioning in a general population is well established.[25] Within our sample, the domains of executive functioning (ie, ability to plan and execute a nonverbal behavior), speed of information processing, speeded word fluency, and memory appear to be attenuated in those athletes with LD. We also found that a history of concussion is significantly and independently associated with long-term deficits in the domains of executive functioning and speed of information processing, as well as an increase in self-reported symptoms. Such defects appear to be present in those individuals who have sustained 2 or more prior concussions. In this study, a history of 1 concussion does not appear to result in the long-term cognitive morbidity associated with 2 or more episodes of concussion.

The study findings also suggest an interaction of prior concussion and LD on select neuropsychological measures. Players with a history of 2 or more concussions and LD performed significantly worse on tests of executive functioning (Trails B) and speed of information processing (SDMT) relative to players with concussions who were not diagnosed with LD, suggesting an additive effect of LD and multiple episodes of concussion on lowered functioning. When compared with accepted normative values,[26] athletes with LD and multiple concussions performed in the brain impairment range on these 2 measures.

Figure 1. Interaction Between Concussion and Learning Disability History



For Trail-Making Test, Form B (top panel), lower test scores represent better performance. For Symbol Digit Modalities Test (bottom panel), higher scores represent better performance.

Three potential hypotheses may account for these findings. First, athletes with LD who experienced concussion may have had less brain reserve capacity[27] than athletes without LD. The margin of cognitive reserve may be less in athletes with LD and the threshold for manifesting neurobehavioral morbidity may be lower. A second hypothesis is that LD may have made the initial diagnosis of concussion more complex and confusing. For example, an athlete without LD, on initial postinjury assessment, may have revealed discernable and unequivocal cognitive and neurobehavioral impairment when compared with baseline presentation. Conversely, the athletes with LD may have had difficulty performing in the requisite skill areas, both before and after concussion, which were assessed during the acute phase of injury (eg, se-

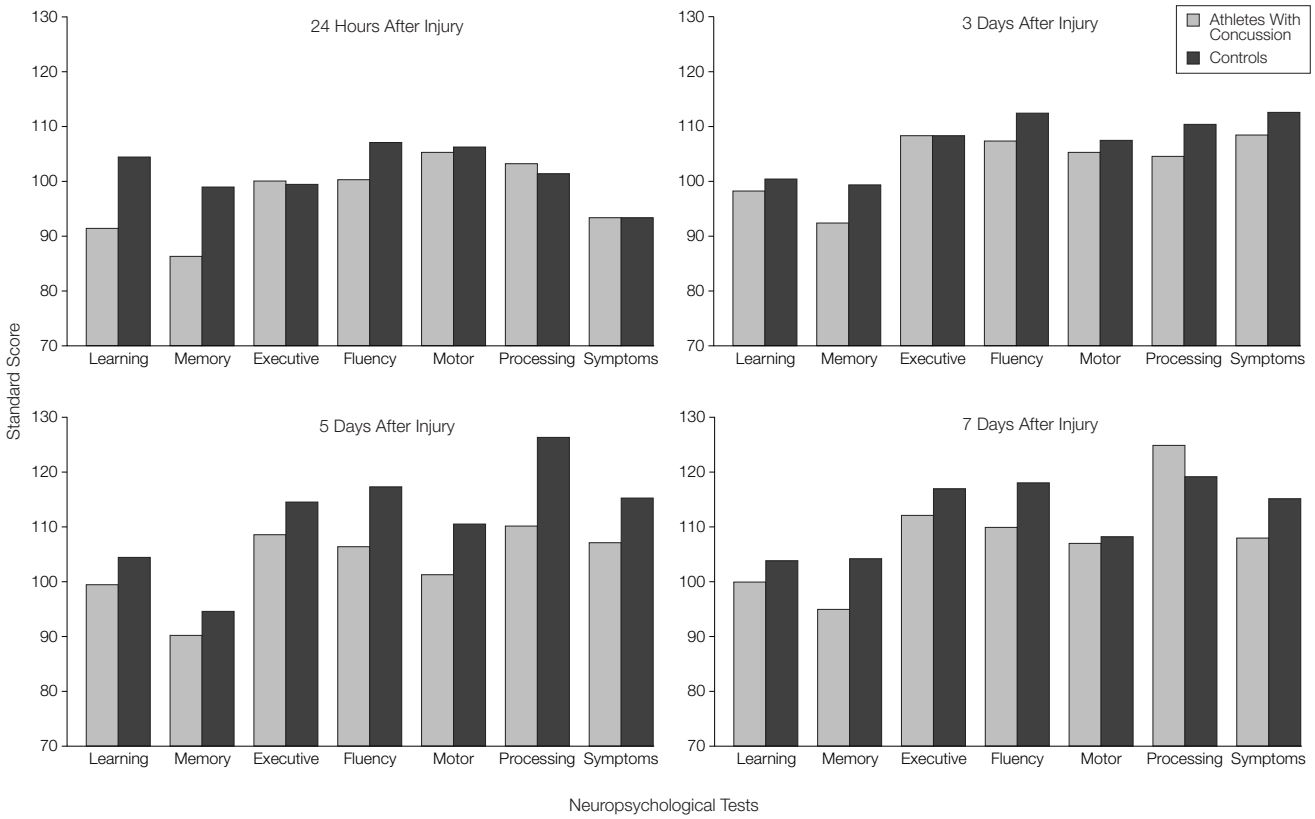
rial 7s, digits backward, months of year in reverse order). Since cursory examination did not reveal overt impairment relative to baseline presentation, these athletes may have returned to play earlier than athletes without LD. Subsequent MTBI may have occurred prior to full neurological recovery. A third hypothesis is that the athletes with LD may have had difficulty learning the proper techniques for safe play or could have neurobehavioral characteristics (eg, impulsivity and attentional impairment) leading to increased risk of injury.

Our data potentially have strong academic implications. Athletes with LD[10] have difficulty in areas of academic achievement, especially within the context of excessive academic and athletic demands. Our data suggest that experiencing 2 or more prior concussions is associated with an attenuation of cognitive skills, which, when combined with the deficits associated with an LD, leads to even further compromised functioning. It is logical to assume that the cognitive domains selectively affected (ie, executive functioning and speed of information processing) are prerequisites for academic success. Thus, academic achievement may become even more difficult for these athletes. This issue is important given that 20% of our sample experienced multiple concussions.

Our study has several limitations. First, although educational records confirmed a diagnosis of LD, concussion history was obtained via structured clinical interview and thus represents nonverifiable self-reported data. However, concussion history is not typically presented in formal medical records and many concussions go unrecognized by trainers or team medical staff.[8] Thus, a self-reported history is essential when examining concussion history and usually represents the only available source of information. Second, although we attempted to gather information regarding alcohol and drug use within our sample, the data obtained were self-reported and may be subject to underreporting.

Data from in-season concussions, although limited by a small sample size, suggest that comprehensive neuropsychological evaluation may prove valuable in the acute and follow-up evaluation of the athlete with a concussion.

Figure 2. Recovery Pattern of Athletes With Concussion and Controls



Scores represent standard scores with mean (SD) of 100 (15). Standard scores for each group were calculated using preinjury baseline means (SDs) for that group to provide an index of change relative to each group's baseline performance. A higher score represents better performance. Learning indicates Hopkins Trials 1-3; Memory, Hopkins Delay; Executive, Trail-Making Test B (Trails B); Fluency, Controlled Oral Word Association Test; Motor, Grooved Pegboard Test, Dominant Hand; Processing, Symbol Digit Modalities Test (SDMT); and Symptoms, Concussion Symptom Scale. A higher raw score indicates better performance for SDMT (speed of information processing).

Any symptoms (cognitive deficit or self-reported) should alert the clinician that full neurological recovery has yet to occur and return to play is contraindicated until these symptoms resolve. Furthermore, mental status screening instruments are variable in nature and may assess only cursory cognitive functioning (eg, no speed of processing or comprehensive memory component), thus missing the subtle deficits associated with injury. In this study, verbal learning and memory appeared to be the most sensitive components in discriminating athletes with concussions from control athletes at the within-24-hours testing session. Less striking differences were found across other cognitive domains. However, careful examination of postconcussion data at days 3, 5, and 7 revealed greater improvement in performance by control subjects relative to athletes with concussion. Specifically, athletes with concussions failed to demonstrate the magnitude of "learning effect" manifested by control subjects, especially across the domains of speed of information processing and executive functioning. This failed learning effect has been observed in previous research.[16] These types of subtle cognitive impairments can be detected through formal neuropsychological testing procedures. With the development and implementation of similar neuropsychological programs, this type of objective information may prove useful in helping athletes, their families, and physicians make more informed decisions about returning to play, thus protecting the athlete from potential adverse effects of concussion.

Finally, identification of other factors associated with risk of concussion may be found through the study of genetic markers such as apolipoprotein E polymorphism. This marker has only recently been linked to poor outcome at 6 months following head injury[28] and has been identified as a risk factor for boxer's dementia.[29] Future studies of sports-related concussion should more systematically investigate the importance of genetic factors in recovery.

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REFERENCES

1. Powell JW. Injury patterns in selected high school sports. In: Bailes JE, Lovell M, Maroon JC, eds. *Sports-Related Concussion*. St Louis, Mo: Quality Medical Publishing Inc; 1999:75-90.
2. Cantu RC, Voy R. Second impact syndrome: a risk in any sport. *Physician Sports Med*. 1995;23:27-36.
3. Kelly JP, Nichols JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-Demasters J. Concussion in sports: guidelines for the prevention of catastrophic outcome. *JAMA*. 1991;266:2867-2869.
4. Torg JS. *Athletic Injuries to the Head, Neck, and Face*. Philadelphia, Pa: Lea & Febiger; 1982.
5. Cantu RC. When to return to contact sports after a cerebral concussion. *Sports Med Dig*. 1988;10:1-2.
6. Jordan BD. Sports injuries. *Mild Brain Injury in Sports Summit: Proceedings of the National Athletic Trainer's Association Research and Education Foundation, Washington, DC, April 16-18, 1994*. Washington, DC: National Athletic Trainers Association; 1994.
7. Gronwall D, Wrightson P. Cumulative effects of concussion. *Lancet*. 1975;2:995-997.
8. Gerberich SG, Priest JD, Boen JR, Staub CP, Maxwell RE. Concussion incidences and severity in secondary school varsity football players. *Am J Public Health*. 1983;73:1370-1375.
9. Nicolson RI, Fawcett AJ, Berry EL, Jenkins IH, Dean P, Brooks DJ. Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet*. 1999;353:1662-1667.
10. Spreen O, Risser AH, Edgell D, eds. *Developmental Neuropsychology*. New York, NY: Oxford University Press; 1995.
11. Donders J, Strom D. The effect of brain injury on children with learning disability. *Pediatr Rehabil*. 1997; 1:179-184.
12. Henderson C. *College Freshmen With Disabilities: A Triennial Statistical Profile*. Washington, DC: Health Resource Center, American Council on Education; 1994.
13. Dikmen S, McLean A, Temkin N. Neuropsychological and psychosocial consequences of minor head injury. *J Neurol Neurosurg Psychiatry*. 1986;49:1227-1232.
14. Hugenholtz H, Stuss DT, Stethem LL, Richard MT. How long does it take to recover from a mild concussion? *Neurosurgery*. 1988;22:853-858.
15. Rimel RW, Giordani B, Barth JT, Boll TJ, Jane JA. Disability caused by minor head injury. *Neurosurgery*. 1981;9:221-228.
16. Barth JT, Alves W, Ryan T, et al. Mild head injury in sports. In: Levin H, Eisenberg H, Benton A, eds. *Mild Head Injury*. New York, NY: Oxford University Press; 1989:257-275.
17. Lovell MR, Collins MW. Neuropsychological assessment of the college football player. *J Head Trauma Rehabil*. 1998;13:9-26.
18. Bream HT. Post-concussion syndrome: a case study. *Athletic Therapy Today*. 1996;1:7-10.
19. Practice parameter: the management of concussion in sports (summary statement). *Neurology*. 1997; 48:581-585.
20. Lovell M. Evaluation of the professional athlete. In: Bailes JE, Lovell M, Maroon JC, eds. *Sports-Related Concussion*. St Louis, Mo: Quality Medical Publishing Inc; 1999.
21. Hurbert RT. *Comprehending Behavioral Statistics*. Pacific Grove, Calif: Brooks/Cole Publishing Co; 1994.
22. Cohen J. A power primer. *Psychol Bull*. 1992; 112:155-159.
23. *Statistica for Windows* [computer program]. Version 5.1. Tulsa, Okla: StatSoft; 1995.
24. Spjotvoll E, Stoline MR. An extension of the T-method of multiple comparison to include the cases with unequal sample sizes. *J Am Stat Assoc*. 1973;68: 976-978.
25. Bears SR, Goldstein G, Katz LJ. Neuropsychological difference between college students with learning disabilities and those with mild head injury. *J Learn Disabil*. 1995;27:315-324.
26. Spreen O, Strauss E. *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary*. New York, NY: Oxford University Press; 1998.
27. Satz P. Brain reserve capacity on symptom onset after brain injury: a formulation and review of evidence for threshold theory. *Neuropsychology*. 1993; 7:273-295.
28. Teasdale G, Nicoli J, Murray G, Fiddes M. Association of apolipoprotein E polymorphism with outcome after head injury. *Lancet*. 1997;350:1069-1071.
29. Jordan BD, Relkin NR, Ravdin LD, Jacobs AR, Bennett A, Gandy S. Apolipoprotein E ε4 associated with chronic traumatic brain injury in boxing. *JAMA*. 1997; 278:136-140.