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# Neuropsychological functioning in athletes with untreated concussion at moderate elevation

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ABSTRACT

*Concussion causes varying degrees of brain damage in athletes, and the neuropsychological consequences of concussion or incomplete recovery can impede skill acquisition. This study examined the neuropsychological recovery from concussion in athletes at moderate elevation, 80% of whom did not seek treatment for their concussion. We collected data on concussions sustained at or around 1966 m among university athletes. American football players at New Mexico Highlands University (N = 13) were administered a 40-minute neuropsychological battery to examine domains affected by concussion such as attention, memory, information processing speed, executive functioning, and depressive symptoms at two time points, before and after the 2016 football season. In total, there were 5 concussed athletes (assessed m = 45.6 days post-injury) and 8 non-concussed control athletes. A repeated-measures ANOVA showed a significant group-by-time interaction for depression, F = 6.335 (p = .029), with concussed athletes showing significant increases in depressive symptoms. Repeated-measures ANCOVAs (controlling for depressive symptoms) of the four athletes who did not seek treatment showed significant group-by-time interactions, with concussed athletes experiencing significant slowing in processing speed F = 26.51 (p = .001) and declines in verbal learning, F = 6.54 (p = .034). Additionally, two athletes (one who sustained a concussion and one who did not) were re-administered the battery mid-season, within 7 days post-injury; the concussed athlete experienced acute deficits in most domains and demonstrated incomplete recovery on measures of depression, verbal learning, and switching. These results indicate that untreated concussions sustained at moderate elevation may not fully recover within the frequently cited 10-day window, and suggest the need for future research into the role of both concussion treatment and elevation in concussion recovery prognosis.*

## 1. Introduction

A concussion is a change in awareness or consciousness caused by a physical impact that can result in cognitive disturbances, physical symptoms, and emotional lability. Up to 3.8 million American university athletes may sustain a concussion each year (Langlois, Rutland-Brown, & Wald, 2006). In New Zealand, a total of 5,556 sport-related concussion medical claims were filed between 2012 and 2016, comprising 28% of all concussion claims and costing NZD $34,421,704 in compensation, or $6,884,341 per year (King et al., 2019). Many athletes choose not to report their injury or seek treatment despite being aware of the symptoms and long-term risks of concussive damage (Leahy, Farrington, Whyte, & O'Connor, 2020; Meier et al., 2015), so these figures may only represent 70% of all sports-related concussions (LaRoche, Nelson, Connelly, Walter, & McCrea, 2016; Meehan, Mannix, O'Brien, & Collins, 2013).

The most common acute symptoms of concussion are headaches, dizziness, fatigue, irritability, and confusion (Daneshvar, Nowinski, McKee, & Cantu, 2011; McCrory et al., 2017). Neurocognitive deficits often include attention, information processing speed, memory, and executive functioning (Collins et al., 1999; Macciocchi, Barth, Alves, Rimel, & Jane, 1996; Macciocchi, Barth, Littlefield, & Cantu, 2001; McCrea et al., 2003). These clinical impairments can translate to functional difficulties with skill acquisition and performance (Van Vleet et al., 2016).

Researchers have often characterized skill acquisition as a process of three stages (DeKeyser, VanPatten, & Williams, 2007). In the first stage (declarative), an individual creates a step-by-step

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understanding of how to do something; in the second stage (procedural), they practice the skill and it becomes less declarative and more performative; when they reach the third stage (automatic), they can perform the skill effortlessly (Anderson et al., 2004; Taatgen, Huss, Dickison, & Anderson, 2008). This progression requires sustained attention to the task; memory for the declarative task instructions; rapid and coordinated visual and motor functioning; and executive control sufficient to monitor task performance, compare task performance to a performative model, and self-correct when errors are detected. Because they are continuously receiving performance feedback and integrating that feedback into their skill development, and as they are also highly trained athletes, university-level athletes likely operate largely within the procedural to automatic stages of skill acquisition.

The neurocognitive deficits commonly found immediately after concussion in university football players are among the most crucial to skill acquisition: problems with sustained auditory attention and visuomotor speed (Macciocchi et al., 1996), verbal learning and delayed recall deficits (Collins et al., 1999), and mild declines in processing speed, verbal fluency, cognitive flexibility (switching), and learning and memory (McCrea et al., 2003). Athletes with multiple concussions show more deficits in executive functioning and processing speed when compared to athletes with no prior history of concussions (Collins et al., 1999). Deficits in verbal learning and memory have been found to persist for at least 5 days (Collins et al., 1999), and to return to baseline after 7 days (McCrea et al., 2003).

Persistent post-concussive symptoms (experienced more than three months post-injury) are often estimated to occur in 10-15% of concussions, but estimates vary from 1.4-29.3%, depending on the diagnostic criteria used (Rabinowitz & Arnett, 2013; Rose, Fischer, & Heyer, 2015; Sterr, Herron, Hayward, & Montaldi, 2006). The most common cognitive deficits observed more than one year post-injury are in attention, memory, and processing speed (Dean & Sterr, 2013; Konrad et al., 2011; Sterr et al., 2006). Depression is also a common persistent post-concussion symptom that affects approximately 50% of concussed people in the first year post-injury (Barker-Collo et al., 2015; Bombardier et al., 2010).

In addition to these cognitive effects, emotional symptoms of concussion can also affect skill acquisition by reducing motivation and by directly decreasing cognitive functioning, potentially seen as lapses of memory or concentration. Depression can be caused or worsened in student athletes by being benched and feeling useless, poor academic performance because of persisting cognitive symptoms, lack of team support, and an uncertain return-to-play timeline (Chen, Johnston, Petrides, & Ptito, 2008; Kuehl, Snyder, Erickson, & McLeod, 2010). Post-concussive mood dysfunction may cause or exacerbate deficits in reaction time, processing speed, and visual memory post-injury (Bailey, Samples, Broshek, Freeman, & Barth, 2010; Kontos, Covassin, Elbin, & Parker, 2012). Furthermore, many other concussion symptoms overlap with symptoms of depression, which can make it hard to detect after an injury (Barker-Collo et al., 2015). As with neurocognitive deficits, depression can impede skill acquisition and performance, and may consequently affect the student athlete's daily functioning, including their academic performance (Hysenbegasi, Hass, & Rowland, 2005). Thus, it is important to assess depression when studying post-concussive neurocognitive deficits.

Risk factors for sport-related concussion include history of concussion (Guskiewicz et al., 2003; Lynall, Mauntel, Padua, & Mihalik, 2015; Macciocchi et al., 2001; McCrory et al., 2017; Nordström, Nordström, & Ekstrand, 2014), fatigue (Finnoff, Jelsing, & Smith, 2011), and pre-existing pathology or psychological distress such as depression or anxiety (Fann et al., 2002; Vassallo, Proctor-Weber, Lebowitz, Curtiss, & Vanderploeg, 2007). Fann and colleagues (2002) examined injury prevalence risks associated with psychiatric illness and found increased rates of concussion in individuals who had been treated in the past year (including medication and mental health services). Vassallo and colleagues (2007) found increased concussion rates in individuals diagnosed with depression, anxiety, and conduct disorders. Additionally, depression that presents or worsens after a concussion is associated with poorer outcome (van der Naalt et al., 2017). In this way, depression could act as both a risk factor and a symptom of concussion. Most research on risk factors of sustaining a concussion has focused on intrinsic pre-injury factors, while research on environmental or post-injury risk factors tends to examine the effects on recovery timelines and not prevalence.

An environmental factor that may affect an athlete's vulnerability to concussion is field elevation (above sea level).[i] Some researchers have found evidence for differences in concussion rates and recovery times among football players by elevation. Lynall and colleagues (2016) determined that the relative risk of concussion was 1.47 times higher for university athletes at the highest quartile of elevation, compared to those at the median elevation of 178 m (584 ft), and was 1.67 times higher than those at the lowest quartile. The athletes' recovery time was also longer when the elevation was higher than the median; 60.3% of athletes concussed below 178 m returned to full activity within six days, as opposed to 37% of athletes concussed above 178 m (Lynall, Kerr, Parr, Hackney, & Mihalik, 2016). Moderate elevation may cause the onset of mental and physical fatigue sooner than at sea level, leading to lapses in concentration and poorer self-protection and increasing the risk of injury. Elevation also could affect injury severity and duration by reducing the body's ability to use available oxygen or by slowing the healing process. Increased oxidative stress can cause cellular damage when unstable free radicals "steal" electrons from proteins and cells, causing DNA breakage and induced apoptosis, or programmed cell death (Askew, 2002; Bakonyi & Radak, 2004). At increased elevation, the oxygen concentration in the air is the same, but the decreased air pressure makes the transport of oxygen more difficult, therefore reducing the usability of the oxygen present (Askew, 2002). These hypoxic conditions interact with factors like reduced antioxidant activity at elevation and increased UV light in a closer and thinner atmosphere, and can cause the formation and increase the activity of free radicals (Askew, 2002; Bakonyi & Radak, 2004). In the presence of one stressor, the body's antioxidant defense system has a reserve of vitamins, enzymes, or other nutrients that can usually "sacrifice" their own electrons to prevent damage to other molecules, but when two or more stressors are present the defense system can become overwhelmed, and oxidative damage can occur (Askew, 2002). When exercising at moderate elevation, hypoxic damage may reduce cellular metabolism and cause or contribute to the aforementioned neurocognitive impairments after concussion.

In contrast, studies done at the high school level (Smith et al., 2013) and at the professional level (Myer et al., 2014) found that athletes sustain fewer concussions at moderate elevations, though they did not study recovery times. The researchers propose a "tight fit" theory to describe physiological mechanisms by which moderate- to high-elevation exposure could swell the brain to protect against concussions (Myer et al., 2014; Smith et al., 2013). Reduced usable oxygen at moderate elevation raises the athlete's blood pressure (because extra force is needed to distribute the oxygen through the bloodstream at higher elevations), causing the cranial blood vessels to swell to fill the space between the brain and the skull. Jugular constriction prevents drainage and holds the blood in the cranium, keeping the brain engorged and firm. This swelling ostensibly limits inertial damage from "slosh," which describes the collision of the semi-liquid brain into the skull due to sudden and rapid changes in acceleration/deceleration forces and the differential rotational speeds between the brain and skull (Gu, Kawoos, McCarron, & Chavko, 2017; Myer et al., 2014; Smith et al., 2013). Though it may seem contradictory, the "tight fit" theory may be compatible with Lynall and colleagues' (2016) findings of more severe concussions at higher elevations. It is possible that the "tight fit" may reduce the overall incidence of concussion at elevation, but those who sustain concussions at higher elevations are still vulnerable to prolonged recoveries because of the effects of mild hypoxia.

### *1.1. Current study*

The current study aims to contribute descriptively to the sparse literature on concussion at elevation by examining the longitudinal course of neuropsychological functioning in a sample of concussed and non-concussed football players over a season at moderate elevation. We hypothesized that concussed athletes would show more pre- to post-season declines in attention, processing speed, memory, and executive functioning, but not in estimated verbal IQ, than athletes who did not sustain concussions. We then posed research questions to 1) examine the relationship between concussion and depressive symptoms pre- and post-season, and 2) examine if depression is a risk factor for sustaining a concussion. Our results also serve as descriptive neuropsychological data of untreated concussions, since 80% of our concussed athletes did not report their injuries until the post-season assessment.

## 2. Methods

### *2.1. Participants*

All participants were male student athletes aged 18 and older who played American football for a Division II university at moderate elevation (1966 m above sea level). Twenty-two athletes consented to participate and were administered a 40-minute neuropsychological test battery before the start of the 2016 college football season. Participants were only included if they completed both the pre-season and post-season assessments; no additional exclusion criteria were applied. Thirteen participants completed both assessments and the end-of-season questionnaire, and were included in analyses. The average participant age ($N$ = 13) was 20.08 years old (SD = 2.40). Participants were almost evenly split between freshmen (first-year students, 30.8%), sophomores (second-year students, 30.8%), and juniors (third-year students, 38.4%). Seven (53.9%) played offensive positions, five (38.4%) played defensive positions, and one (7.7%) played on special teams as a kicker. Participants had been playing football for an average of 9.9 years (SD = 4.73). Eight (61.5%) participants reported having previously sustained concussions, and of those eight, three (37.5%) reported loss of consciousness.

Five of the 13 participants (38.5%) were believed to have sustained concussions during the course of the study, while eight (61.5%) did not. The only injury that was reported mid-season (and tested during the sub-acute phase) was caused by a motorcycle crash and was verified by the athletic trainer. The remaining four concussions were sustained during a game and reported only on the end-of-season self-report questionnaire, so those athletes were not tested during the acute or sub-acute phase. The most frequently cited reasons for not reporting the concussion when it occurred were: not thinking it was serious, not wanting to miss practice, and not wanting to disappoint the team. Three of the athletes played offensive positions (running back, tight end, and offensive lineman), and the fourth played a defensive position (line-backer). Two participants—the participant who sustained his concussion in a motorcycle crash, and another with no evidence of concussion—also completed the neuropsychological battery within a week of the concussion.

### *2.2. Procedure*

We recruited football players from a pre-season team meeting. Players who agreed to participate scheduled a baseline neuropsychological assessment before contact practice began. All participants signed informed consent forms and HIPAA-compliant release forms allowing the athletic trainer to notify us if a participant sustained a concussion. Participants completed an athletic questionnaire that included demographic information and medical history. Neuropsychological assessments were completed individually in sound-proofed rooms and took 40 minutes to administer. If a participant sustained a concussion during the season, efforts were made to have the concussed participant and a non-concussed participant complete the battery again within 48 hours of the concussion, using alternate forms of the neuropsychological tests when possible to reduce the practice effects, as is standard for longitudinal research on neuropsychological functioning (Brandt & Benedict, 2001; Lezak, Howieson, & Loring, 2012). At the end of the season, participants again completed the neuropsychological test battery and an end-of-season questionnaire. Follow-up calls were made to participants who admitted to an unreported concussion to confirm the date and elevation at which it was sustained. Three concussions were sustained at the home elevation (1966 m, including the treated participant), one was sustained at a higher elevation (2076.3 m) and one was sustained at a lower elevation (871.7 m). At the time of the post-season evaluation, one concussed participant was tested six days post-injury, and the remaining four concussed participants were tested at the end of the season, between 26-93 days ($m$ = 45.9) after their injury. One date of injury and one date of end-of-season assessment were estimated and are accurate within a week.

*2.3. Tasks*

The Hopkins Verbal Learning Test, Revised (HVLT-R) measures verbal learning and memory through a list of 12 semantically related words (Brandt, 1991; Brandt & Benedict, 2001). The HVLT-R includes three immediate recall trials (for a composite learning score), a delayed recall trial, and a recognition trial, with six alternate forms to minimize practice effects. Higher scores indicate better performances. Test-retest reliability has been found to be high for the learning and delayed recall trials: learning, r = .74; delayed recall, r = .66; recognition, r = .39 (Benedict, Schretlen, Groninger, & Brandt, 1998; Collins et al., 1999; McCrea et al., 2003; Rabinowitz & Arnett, 2013).

The Grooved Pegboard test (GP) measures fine motor skills and bilateral dexterity, and is reputed to detect neurological injury even when other tests cannot (Collins et al., 1999; Lafayette Instrument, 2014). This test is timed and performed using the dominant and then non-dominant hand, with faster times (lower numbers) indicating better performances. Its test-retest reliability has been found to range from r = .67-.86, and there is a practice effect associated with this test (Bornstein, Baker, & Douglass, 1987).

The Digit Span (DS) is part of the WAIS-IV and measures attention span and working memory by asking participants to repeat increasingly long strings of numbers both forward and backward (Collins et al., 1999; Wechsler, 2008). Higher total scores indicate better attention (DS-forward) and working memory (DS-backward). Since attention span is typically limited to an average of seven items (Miller, 1956), there is no significant practice effect is associated with this test. The digit span has been found to have an internal consistency of r = .93 across the normative sample of people aged 16-90 and an internal consistency of r = .91 for the normative sample of people aged 20-24. It has been found to have a test-retest reliability of r = .74 for the DS-forward and r = .69 for the DS-backward (Wechsler, 2008).

The Trail-Making Test (TMT) is a timed test with two parts that measure visual attention, psychomotor speed, and task switching (Reitan, 1958). Trail-Making Test A (TMTa) primarily measures psychomotor speed and visual scanning (Lovell & Solomon, 2011). Trail-Making Test B (TMTb) additionally measures cognitive flexibility and switching, which are aspects of executive functioning. Used together, these tests examine psychomotor speed, visual attention, and switching. Because the primary score for this test is completion time, lower scores indicate better performance. This test is considered reliable at r = .40-.60 (Ross et al., 2007), but it has been found to be vulnerable to practice effects, with times on the TMTb reduced by as many as four seconds (Lovell & Solomon, 2011).

The Controlled Oral Word Association Test (COWAT) measures phonemic verbal fluency (Benton & Hamsher, 1978). The COWAT is scored by adding the total number of correct words named that start with different letters over three trials (one letter per trial). Higher scores indicate better performances. Many concussion assessment batteries use this test, and it has shown adequate inter-rater reliability between r = .70-.80 (Abwender, Swan, Bowerman, & Connolly, 2001).

The North American Adult Reading Test, Revised (NAART-R) is used to estimate verbal intelligence, which is not thought to be affected by concussion (Blair & Spreen, 1989; Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990; Sterr et al., 2006). The NAART-R is a list of 63 words with silent letters or unconventional pronunciations (debris, quadruped, sidereal), and higher numbers of words pronounced correctly indicates better performance. The NAART-R has been found to have an inter-rater reliability of r = .93 and to be correlated (r = .75) with the WAIS-R Vocabulary subtest (Uttl, 2002).

The Beck Depression Inventory, 2nd Edition (BDI-II) is a 21-item self-report measure of depressive symptoms (Beck, Steer, & Brown, 1996). Each symptom is rated on a 4-point scale that ranges from 0 to 3; total scores can range from 0 to 63, with higher scores indicating more depression symptoms. The BDI-II has shown an internal consistency of $\alpha$ = .92 and a test-retest reliability of r = .93 (Beck et al., 1996). The BDI-II is positively correlated (r = .71) with the Hamilton Psychiatric Rating Scale for Depression (Beck et al., 1996).

At the time of the baseline evaluation, participants completed a demographic and athletic questionnaire to assess their personal histories, including their football playing history, medical history (specifically past concussions with or without loss of consciousness, other brain injuries, and mental health diagnoses) and family history of neurodegenerative disease. At the end of the season, participants completed a questionnaire about concussions sustained that they did not report to their coaches or athletic trainers, which included a definition of a concussion and reasons why the athlete may not have reported the concussion. This questionnaire was adapted from La Roche and colleagues' (2016) study on rates of reported and unreported concussions.

*2.4. Ethical considerations*

This study received IRB approval from New Mexico Highlands University on 6 June 2016. All participants completed an informed consent form, as well as a HIPAA student athlete release form permitting athletic trainers to share information about the athlete's injuries with the researchers. No deception was involved.

*2.5. Statistical approach*

Participants ($N$ = 13) who had completed both the baseline and end-of-season neuropsychological evaluations were included in the data analyses. Out of five concussed athletes, one athlete was diagnosed, removed from play, and treated, so we have excluded his data from group analyses to focus on the remaining four athletes who did not report their concussions and were not treated. We also ran analyses with all five concussed athletes included, and found almost identical results, so only analyses of the untreated athletes are reported here.

We analyzed the data in SPSS, and we primarily used repeated-measures ANOVAs and ANCOVAs (controlling for depressive symptoms, whose effect was determined by a $t$-test). The statistical tests for the hypothesis were one-tailed and the tests for the research questions were two-tailed. The assumption of equality of variance (Levene's test) was met for all subtests, but for the BDI-II it approached a violation at $p$ = .063. The assumption of equality of covariance (Box's test) was met for all subtests. The assumptions of normality (assessed by the Shapiro-Wilk test with residuals) and of sphericity were met for all subtests. All results are reported at a 95% confidence interval.

Table 1: Pre-season to post-season changes in BDI-II scores of concussed participants

| Concussed athlete | Pre-season BDI-II | Range | Days post-injury | BDI-II Increase | Post-season BDI-II | Range |
|---|---|---|---|---|---|---|
| A | 1 | Minimal | 26 | +14 | 15 | Mild |
| B | 3 | Minimal | 92 | +10 | 13 | Mild |
| C | 20 | Moderate | 65 | +1 | 21 | Moderate |
| D | 9 | Minimal | 42 | 0 | 9 | Minimal |
| E (treated) | 12 | Minimal | 60 | +8 | 20 | Moderate |
| Mean | 9 | Minimal | 57 | +6.6 | 15.6 | Mild |

*Note*: BDI-II=Beck Depression Inventory, 2nd Edition.

## 3. Results

### *3.1. Depressive symptoms in concussed & non-concussed participants*

A *t*-test was used to examine whether concussed and non-concussed participants differed in baseline depressive symptoms, to test pre-injury depression as a risk factor for concussion. Participants who sustained a concussion in 2016 reported a higher average BDI-II score before the season ($M$ = 9.0, SD = 7.6) than the participants who did not sustain a concussion ($M$ = 5.7, SD = 2.9), but the between-group differences were not significant, $t(10) = -1.058$, $p = .315$. A repeated-measures ANOVA was then calculated using BDI-II score as the dependent variable and concussion status (concussed vs non-concussed) as the independent variable. A significant main effect was found for time, with an overall trend for increasing depressive symptoms, $F(1, 1) = 5.444$, $p = .040$. Additionally, a significant group (concussed vs non-concussed) by time interaction was found; the non-concussed group showed no change in depressive symptoms over time, whereas the concussed group showed a significant increase in depressive symptoms, $F(1, 1) = 6.335$, $p = .029$ (Figure 1). Because of this significant interaction, changes in BDI-II scores were included as a covariate in repeated measures ANCOVAs to examine the relationship between concussion status and performance on the other neuropsychological measures.

BDI-II scores of 0-13 points are considered indicative of "minimal" depression, scores of 14-19 points "mild" depression, scores of 20-28 points "moderate" depression, and scores over 29 points "severe." Three concussed participants who reported minimal depressive symptoms in the beginning of the season had entered the mild or moderate range post-concussion. The individual changes in BDI-II scores are shown in Table 1.

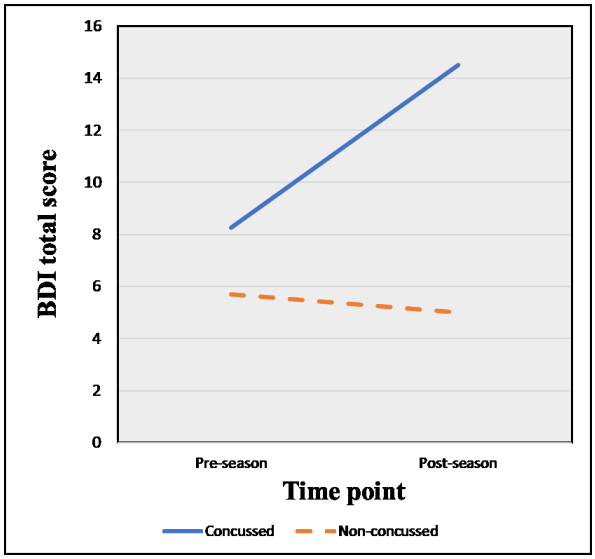


Figure 1: Pre-season to post-season change in BDI-II score by group

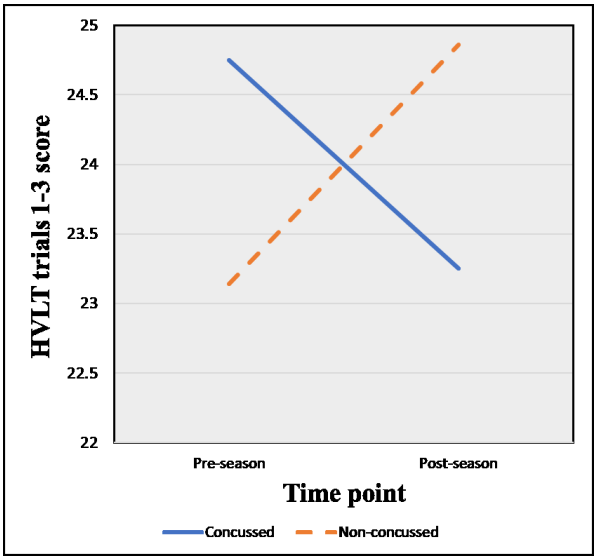


Figure 2: Pre-season to post-season change in HVLT Learning score by group

Table 2: Pre-season ($T_1$) to post-season ($T_2$) repeated-measures ANCOVAs for neuropsychological tests by concussion status, controlling for changes in BDI-II scores

| **Test** | **$T_1$ (conc.)** *M* (*SD*) | **$T_2$ (conc.)** *M* (*SD*) | **$T_1$ (cont.)** *M* (*SD*) | **$T_2$ (cont.)** *M* (*SD*) | ***F (df)*** | ***p*** | **1-ß** |
|---|---|---|---|---|---|---|---|
| HVLT learning | 24.75 (3.59) | 23.25 (3.20) | 23.14 (1.77) | 24.86 (3.81) | 6.54 (1, 8) | **.03** ** | .61 |
| HVLT recall | 9.25 (.50) | 9.75 (1.89) | 9.00 (1.16) | 8.57 (1.99) | .00 (1, 8) | .99 | .05 |
| HVLT recog. | 11.50 (.71) | 12 (0) | 11.80 (.45) | 12 (0) | .64 (1, 8) | .89 | .05 |
| GP dom. | 81.25 (15.31) | 81.75 (9.78) | 69.57 (10.85) | 72.14 (9.35) | .20 (1, 8) | .67 | .07 |
| GP non-dom | 87.00 (12.11) | 96.50 (9.98) | 78.14 (13.46) | 81.71 (20.09) | .88 (1, 8) | .38 | .13 |
| COWAT | 30.50 (6.86) | 37.50 (3.11) | 34.57 (5.47) | 37.43 (7.61) | .29 (1, 8) | .61 | .08 |
| DS forward | 6.75 (2.22) | 7.75 (2.06) | 6.57 (1.72) | 7.00 (1.29) | .23 (1, 8) | .64 | .07 |
| DS back | 7.00 (3.37) | 6.50 (1.29) | 5.33 (1.03) | 6.83 (2.64) | .98 (1, 8) | .36 | .14 |
| TMTa | 24.25 (7.93) | 33.25 (5.38) | 33.71 (8.50) | 24.86 (5.27) | 26.51 (1, 8) | **.001** ** | .99 |
| TMTb | 80.75 (23.17) | 54.75 (6.19) | 85.43 (54.89) | 69.57 (23.84) | .29 (1, 8) | .65 | .07 |
| NAART-R total | 26.75 (3.95) | 26.50 (4.73) | 19.29 (9.27) | 20.71 (9.43) | 1.88 (1, 8) | .21 | .23 |
| NAART-R FSIQ | 101.09 (3.08) | 100.75 (3.78) | 95.26 (7.23) | 96.43 (7.30) | 1.92 (1, 8) | .20 | .23 |

*Note.* $T_1$=pre-season; $T_2$=post-season; conc=concussed participants; cont.=non-concussed participants; 1–ß = power; BDI-II=Beck Depression Inventory, 2nd Edition; HVLT=Hopkins Verbal Learning Test; recog.=recognition; GP=Grooved Pegboard; dom. =dominant; DS=Digit Span; TMT=Trail-Making Test; COWAT=Controlled Oral Word Association Test; NAART=North American Adult Reading Test. ** denotes $p < .05$.

### *3.2. Short-term neuropsychological effects of concussions*

Repeated-measures ANCOVAs (with change in depression as the covariate) were used to compare changes in neuropsychological test performance between the concussed and non-concussed groups. When controlling for depressive symptoms, significant group-by-time interactions were found for the HVLT learning recall trials and TMTa time. Total words recalled in the HVLT learning trials increased for non-concussed participants, but declined for participants who had sustained a concussion, $F$ (1, 8) = 6.543, $p$ = .034 (Figure 2). TMTa times decreased for the non-concussed participants, and increased for the participants who sustained concussions, $F$ (1, 8) = 26.511, $p$ = .001 (Figure 3). There were no significant group-by-time interactions in performance on the NAART-R, $F$ (1, 8) = 1.882, $p$ = .207, or the other neuropsychological tests. Results of all ANCOVAs are reported in Table 2.

### *3.3. Acute neuropsychological functioning comparison of a concussed vs non-concussed athlete*

For illustrative purposes, Figure 4 shows the difference in performance on all neuropsychological tests between the concussed athlete and a non-concussed athlete. Because of the small sample size, these data were not subjected to standard statistical tests, and should instead be interpreted as a descriptive comparison of a post-concussive neuropsychological recovery trajectory to healthy neuropsychological functioning. A closed triangle represents a complete return to baseline after post-concussive impairment.

The athlete who reported his concussion during the season and a control participant without any suspected concussion were administered the neuropsychological battery after the concussion was reported (six days post-concussion). The concussion was sustained at 1966 m during a motorcycle crash, not a game or practice.

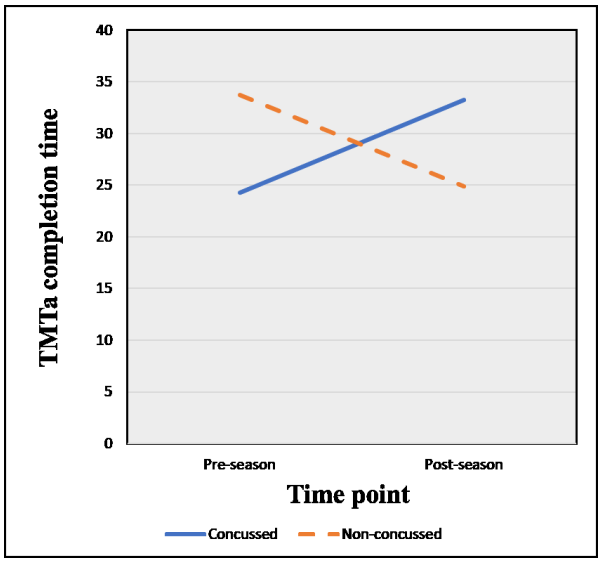


Figure 3: Pre-season to post-season change in TMTa score by group

Declines in functioning post-injury were seen on the HVLT delayed recall, HVLT learning, DS-backward, and DS-forward, with an apparently incomplete recovery by the end of the season. The concussed athlete showed initial impairment after the concussion and then a return to baseline (or better) on the HVLT recognition, COWAT, GP, TMTa, and TMTb by the end of the season. Consistent with the results of the ANCOVAs (comparing neuropsychological functioning of the undiagnosed vs non-concussed athletes), the diagnosed athlete demonstrated an increase in reported BDI-II symptoms and a decline in performance on the HVLT learning trial at the post-season assessment when compared to the control athlete.

## 4. Discussion

This study intended to examine the neuropsychological consequences of concussions in university football players at moderate elevation of 1966 m (6,450 ft), but our results actually appear to reflect the effects of not seeking treatment on the course of recovery. All five football players who sustained concussions[ii] during the season showed significant declines by the end of the season than those without concussion on measures of processing speed and verbal learning (though the individual with the diagnosed concussion was not included in the neurocognitive statistical analyses reported). The specific domains of processing speed and verbal learning are consistent with other researchers' findings of concussed and subconcussed athletes (Collins et al., 1999; Fann, Uomoto, & Katon, 2001; McAllister et al., 2012; McCrea et al., 2003; Talavage et al., 2014), but the duration of injury is not. Because changes in depression symptoms were controlled for, the declines in neuropsychological functioning cannot be explained by depression. Additionally, our data do not support depression as a risk factor for concussion.

### *4.1. Selection & interpretation of data*

The neuropsychological scores of the four athletes who reported a concussion in the end-of-season questionnaire were examined, and in all four cases demonstrated impairments consistent with concussion. We have no reason to believe any of the concussions were falsely reported, as false reports of sports-related concussion are rare because the athletes would have nothing to gain (especially at the end of the season). Very little literature exists regarding false reporting of sport-related concussion by athletes, though some research has studied over-reporting of concussion symptoms in military personnel (Armistead-Jehle et al., 2018; Cooper, Nelson, Armistead-Jehle, & Bowles, 2011; L. Miller, 2001) or those involved in litigation (Silver, 2012; Suhr, Tranel, Wefel, & Barrash, 1997), who have more external motivations to report or exaggerate symptoms (benefits, time off, or compensation) than university athletes. Even if our concussed athletes did not meet the clinical definition of concussion, they still showed more deficits than non-concussed or subconcussed athletes post-season (J. R. Miller, Adamson, Pink, & Sweet, 2007; Moore, Lepine, & Ellemberg, 2017), supporting their self-diagnosis of concussion.

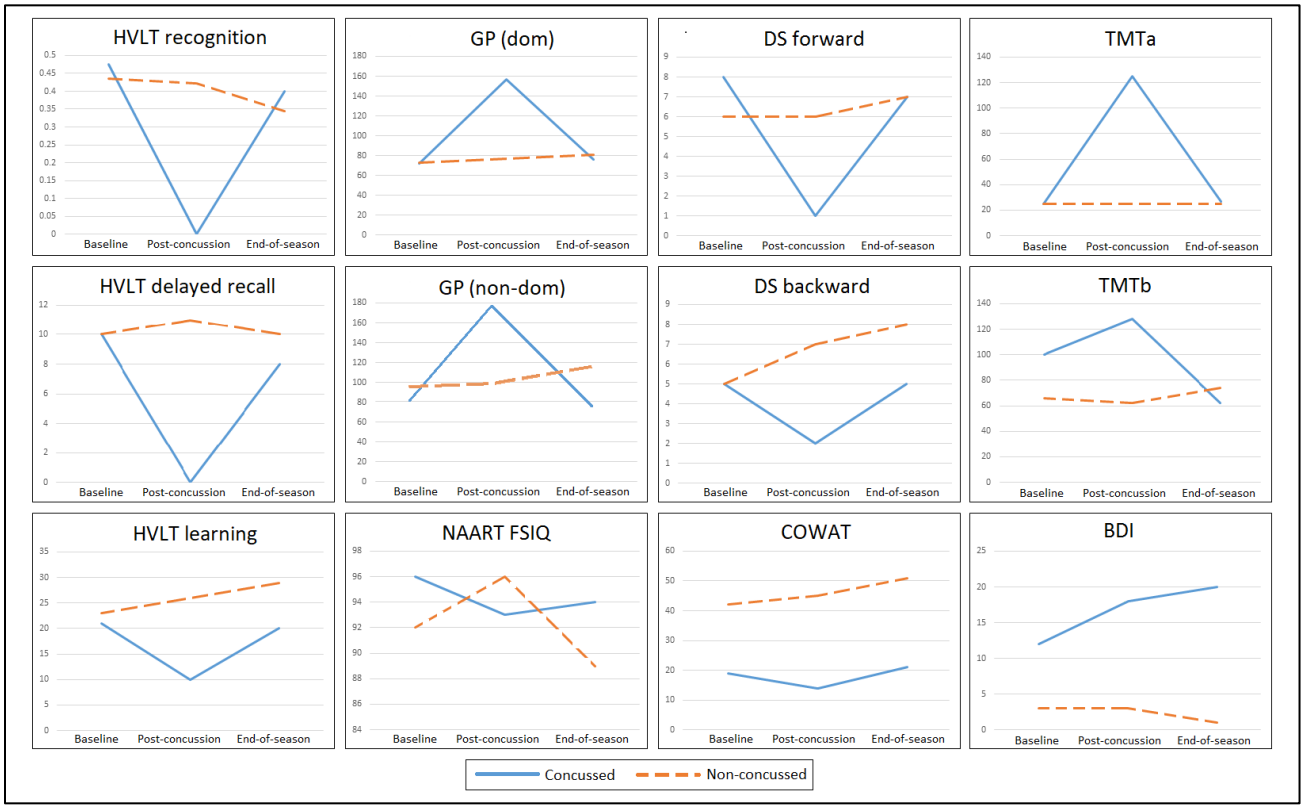


Figure 4: Comparison between concussed participant (in blue) and non-concussed participant (in red) mid-season

Our finding of a higher proportion of initially unreported (than reported) concussions is consistent with Meier and colleagues' (2015) conclusion that athletes are more likely to report symptoms in a confidential setting that is not connected to the athletic program. In a study on Irish amateur athletes, Leahy and colleagues (2020) reported that, of athlete respondents who believed they had sustained a concussion, 27.8% did not seek treatment, and 64% continued to play while symptomatic. Additionally, while 95% of the athletes self-reported understanding the serious nature of concussive damage, 48% of respondents said they would still hide a concussion during an important match (Leahy et al., 2020). La Roche and colleagues (2016) found that 29.4% of their sample of athletes did not report a concussion, and that the most common reasons were not thinking it was serious and not wanting to lose playing time, which is consistent with our results. Meehan and colleagues (2013) similarly found that 30.5% of athletes concussed during the study had previously sustained a untreated concussion. Our non-treatment-seeking finding of 80% is higher than most reports in the literature (rarely estimated over 50%), but we were not methodologically equipped to study reporting rates accurately.

### *4.2. Unreported/untreated concussions & consequences of RHIs*

Current literature suggests that over 80% of concussions should resolve 7-14 days post-concussion (Karr, Areshenkoff, & Garcia-Barrera, 2014; McCrory et al., 2017). The most likely reason that the concussed athletes in this study did not demonstrate a complete recovery is because they did not report their injuries or seek treatment. Time to treatment is a critical determinant of recovery (Eagle, Puligilla, et al., 2020; Kontos et al., 2020), and continued exposure to concussive damage in an already neurologically weakened state is associated with many additional risks, including increased vulnerability from continued head impacts or other injury (Eagle, Kontos, et al., 2020; Stemper et al., 2019; Talavage et al., 2014), prolonged recovery from concussion (Sterr et al., 2006), and increased risk of neurodegenerative disorders like Alzheimer's disease or chronic traumatic encephalopathy later in life (Finkbeiner, Max, Longman, & Debert, 2016; Guo et al., 2000; McAllister & McCrea, 2017; Mouzon et al., 2018), the last of which is especially relevant to individuals with a history of multiple concussions (Guskiewicz et al., 2005). Finally, an athlete who does not remove themself from play is also at increased risk of receiving a second and fatal impact, especially in the first 10 days post-concussion, though this is rare (Bey & Ostick, 2009).

The absence of a diagnosed or suspected concussion (due to observable behavioral or neuropsychological signs) does not mean that no neurological damage has occurred. Subconcussive injury can happen when athletes experience enough repetitive head impacts (RHIs) at forces not severe enough to cause a concussion or gross neurocognitive deficits (J. R. Miller et al., 2007; Moore et al., 2017). Subconcussive damage has mainly been detected as white matter damage (Bazarian et al., 2014; Gu et al., 2017; McAllister et al., 2014; Moore et al., 2017), neurometabolic changes (Bailes, Petraglia, Omalu, Nauman, & Talavage, 2013; Bari et al., 2019; Hunter, Branch, & Lipton, 2019), and alterations to electrophysiological activity or communication networks (Abbas et al., 2015; Moore et al., 2017; Pearce, 2016), though some researchers have found subtle neurocognitive deficits in asymptomatic athletes after a season (McAllister et al., 2012; Talavage et al., 2014).

RHIs may increase the risk of sustaining a concussion by lowering the threshold of force needed to cause injury or induce symptoms (Caccese et al., 2018; Stemper et al., 2019). Stemper and colleagues (2019) found a significant increase in RHIs in 72% of concussed athletes in the days leading up to concussion, which was often sustained from impacts with low concussive probabilities; over half were caused by forces thought to have a <1% risk of injury. Beckwith and colleagues (2013) found that more head impacts were associated with a delayed diagnosis of concussion, while those diagnosed immediately had experienced impacts of higher forces. Because the concussed athletes in this study did not report their injury (and therefore were not removed from play, diagnosed, and treated), they were then exposed to further impacts and subconcussive brain trauma that may have exacerbated their symptoms and prolonged their recovery.[iii]

Though our research focuses on athletes, unreported concussions are common in the general population. Factors linked with not seeking or receiving care include older age, an injury sustained in sport or at home, being a current smoker, and not being an immigrant (Gordon, 2020; Kiefer et al., 2015). While Gordon (2020) did not find any effects for gender, race, or income, he found that 21% of his respondents did not seek treatment in the first 48 hours, though he cites estimates that are higher. Hesitance to report concussion or seek treatment is also prevalent in American military personnel, where almost 50% may not seek care, mainly because they do not think the injury is serious or are worried about how it will affect their career (Escolas, Luton, Ferdosi, Chavez, & Engel, 2020).

### *4.3. Neuropsychological sequelae of untreated concussions & RHIs*

The concussed athletes demonstrated persistent post-concussive neuropsychological effects in domains that are central to all three stages of skill acquisition, including poorer verbal learning and slowed processing speed (as well as increased depression) when compared to the non-concussed athletes at the end of the season. On the HVLT, the untreated athletes learned 1.5 fewer words at the end of the season than they had learned pre-season, whereas non-concussed athletes learned 1.8 more words than they had at baseline, likely due to a practice effect. Time needed to complete the TMTa for the untreated group increased by an average of 9 seconds, while the time of the non-concussed group dropped by an average of 9 seconds. These deficits in processing speed and verbal learning remained even when controlling for increased depression, suggesting that the cognitive declines were not caused solely by depressive symptoms. As predicted, we saw no difference in estimated verbal IQ, which is consistent with vocabulary being a relatively stable domain that is not known to be affected by concussive damage (King & Kirwilliam, 2011; Leininger et al., 1990; Sterr et al., 2006). There is a statistical possibility that, due to the low power, accepting the null hypothesis could be a type II error (false negative), but the probability of this is low, considering the existing research.

We can see from the comparison of the treated athlete to the non-concussed athlete that most neuropsychological domains showed damage immediately post-injury, and while most of these declines did resolve by the end of the season, the isolated deficits that persisted are comparable to those seen in the untreated athletes. Both the treated and untreated athletes in this study showed impairments in verbal learning and memory on the HVLT immediate recall trials. Only the untreated athletes showed post-season processing speed deficits on the TMTa, whereas the treated athlete showed initial impairment followed by a return to baseline. The treated athlete, however, did not show a return to baseline on the TMTb, demonstrating impaired cognitive flexibility not seen in the untreated group.

Though we did not examine head impact exposure, several studies on contact athletes who were and were not formally diagnosed with concussion have found similar short-term neuropsychological deficits (1-3 months post-injury). Verbal learning and memory and switching deficits are common after RHI and sport-related concussion (Collins et al., 1999; McAllister et al., 2012; McCrea et al., 2003; Talavage et al., 2014). Talavage and colleagues (2014) studied functional (not clinical) impairment on verbal and visual memory tasks in RHIs, and estimated that at least 17% of contact athletes who have not been diagnosed with concussion still experience neurocognitive impairments, mainly in visual and verbal working memory. McAllister and colleagues (2012) compared RHIs in contact and non-contact athletes and found poorer post-season performance by the contact athletes on the CVLT (a similar measure of verbal learning and memory), the TMTb, and a measure of reaction time. McCuddy and colleagues (2018) reported that depressive symptoms were correlated with significant changes to functional connectivity at one month post-concussion, specifically between areas involved in attention and default activity in a resting state. Their finding suggests that compensatory neurological alterations associated with attention and depression may persist for a month or more, despite concussion symptoms resolving within 1-2 weeks for a majority of concussed individuals (Karr et al., 2014; McCrory et al., 2017).

Some neuropsychological domains may be especially vulnerable to chronic damage from RHIs and concussive damage, especially if the athlete did not seek treatment. The processing speed deficit seen on the TMTa in the untreated players is somewhat consistent with findings of persistent deficits in information processing speed and working memory in a non-treatment-seeking population at least one year post-concussion, when compared to non-concussed controls (Dean & Sterr, 2013). Moore and colleagues (2017) found that athletes who were concussed at least 11 months before testing remembered significantly fewer words on the HVLT delayed recall trial than the subconcussed athletes, who in turn remembered significantly fewer words than the non-contact athletes. Verbal memory deficits and mood dysfunction were also seen by Lepage and colleagues (2019), who were investigating the long-term effects of RHIs on limbic structure volume in retired professional football players and controls, though the concussion history of the players was not specified.

### *4.4. Concussion and depression*

The group-by-time interaction between depressive symptoms and concussion that we found supports depression as a serious and often persistent symptom of concussion (Chen et al., 2008; Konrad et al., 2011; Kontos et al., 2012; Lavoie et al., 2017). While the non-concussed athletes experienced no significant change in depression symptoms over the season, all five athletes with concussion reported a statistically significant increase of almost seven points, on average, in their BDI-II scores. In addition, three out of the five concussed athletes reported a clinically significant increase in the severity of their depression from their baseline to the end of the season (Table 1). These findings suggest that concussed athletes do experience a greater increase in depressive symptoms over non-concussed athletes, and people with untreated concussion may experience higher rates of post-concussive depression. There could be reasons unrelated to the concussion that the depression scores of these athletes increased (e.g., interpersonal stress, increased academic workload), however, they do not explain why the change in depression scores was more pronounced in concussed participants than in non-concussed participants.

Our data do not support others' findings of pre-existing psychological distress as a risk factor for concussion (Fann et al., 2002; McCauley, Boake, Levin, Contant, & Song, 2001; Vassallo et al., 2007), but our non-significant finding could be a false negative (type II error) due to our small sample size.

Treating an individual's post-concussive depression with medication or therapy can improve their injury outcome. Fann and colleagues (2001) conducted an 8-week pharmacological intervention in patients with depression 3-24 months post-mTBI, after which the patients' neuropsychological performances improved significantly in many domains, including completion times on both the TMTa and the TMTb. They concluded that cognitive deficits (such as verbal memory, psychomotor speed, and cognitive flexibility) and depressive symptoms seen in patients from 3-24 months after mTBI can be improved with depression treatment.

### *4.5. Contributions to elevation research*

Though the likely explanation for our findings of persistent neuropsychological deficits among concussed student athletes is that the concussions were untreated, they should still be considered through the lens of the elevation at which the data were collected. Our study did not directly investigate elevation as a risk factor for concussion incidence or prolonged recovery, but our data of incomplete recovery in all five athletes suggest the need for more methodologically rigorous research into the role of elevation in concussion etiology and management. The three studies on concussion at elevation mentioned above (Lynall et al., 2016; Myer et al., 2014; Smith et al., 2013) were criticized for several methodological flaws, mainly the use of less than 200 m (550-650 ft) as the median between "high" and "low" elevation conditions; physiological changes are not seen below the 2200 m (7,000 ft) threshold accepted as "high elevation" by physiologists (Smoliga & Zavorsky, 2017; Zavorsky, 2016). Furthermore, the

degree of elevation-induced swelling required to cause the "tight fit" only occurs above 4000 m (13,000 ft), and only in fewer than 4% of mountain climbers, an already small subset of the general population (Smoliga & Zavorsky, 2017; Zavorsky, 2014). A meta-analysis of the three studies (Zavorsky & Smoliga, 2016) found the risk of concussion to be equivalent in the low (0-200 m) and high (200-300 m) conditions, but the statistical model used and the selection and interpretation of the data were considered questionable (Bailes & Smith, 2017; D. M. Bailey et al., 2017; Myer, Schneider, & Khoury, 2017; Zavorsky & Smoliga, 2017). In addition, the meta-analysis only examined elevation as a risk factor for sustaining a concussion, and did not address the effect elevation may have on recovery time.

Other researchers have continued to study elevation's influence on concussion prevalence and recovery in athletes. Bogar and Schatz (2019) compared NFL concussion rates of the Denver Broncos (whose "mile high" playing field is the highest in the NFL at 1600 m) to the concussion rates of their three lower-elevation divisional rivals (who train at elevations of -6.4 m, 16 m, and 79 m), but they found no significant differences in the concussion rates. Connolly and colleagues (2018) also studied NFL concussion rates using less than 200 m (644 ft) as the elevation cut-off, and found lower rates of concussion at higher elevations in athletes from teams that train at the higher elevations, but not in athletes from teams who train at lower elevations when they travel to higher elevations. Adams and colleagues (2018) examined if hockey players missed more games (a marker of concussion severity) if their concussion was sustained over 300 m (1000 ft). They similarly determined that athletes who are based at higher elevations sustain fewer concussions when they travel to lower elevations, and that athletes missed fewer games from concussion when they trained at higher elevation, whether they were home or away (Adams et al., 2018).

These studies (Adams et al., 2018; Connolly et al., 2018) attribute this protective effect of elevation to acute and long-term adaptations made by athletes who train at higher elevations, not just an effect of a "theoretically protective physiologic cerebral edema." Adaptation is necessary for skill acquisition because it enables an individual to understand and execute their action capabilities, or the ability to perform common and familiar actions (like sitting down or stepping over something); subtle neurocognitive deficits may interfere with the ability to compensate for small everyday changes to these frequent actions, or with the process of re-integrating an individual's physical senses (such as proprioception or making perceptual judgments) into motor planning (Hirose & Nishio, 2001). Failure to make these dynamic adaptations could result in re-injury. Physiologic adaptation to a moderate elevation supports the "tight fit" theory of fewer concussions at higher elevations (Connolly et al., 2018), but the finding of fewer games missed at higher elevation (Adams et al., 2018) is not consistent with Lynall and colleagues' (2016) findings of longer recovery time for concussions sustained at higher elevation. At this point, it is unclear how or to what degree an adaptation to performing at moderate elevation would influence an athlete's post-concussion recovery.

Hypoxic stress is associated with acute neuropsychological impairment; concussions may produce persistent but subtle deficits in function that only emerge under stress or further injury, even when that individual appears to have recovered. Deficits in memory and vigilance (a type of attention) were seen in concussed participants in a study by Ewing and colleagues (1980), in which ten participants who had recovered from a recent concussion and ten un-injured controls were asked to perform neuropsychological tests in conditions to simulate a hypoxic atmosphere of 3810 m (12,500 ft). Those in the control group scored about 90% correct on the vigilance task at 10 minutes, 20 minutes, and 30 minutes, while the concussion group scored about 85% correct at 10 minutes and 20 minutes, but dropped to 80% correct at 30 minutes. Manderino (2020) also studied the effect of hypoxia as a stressor on cognitive performance (up to 75 minutes exposure) in individuals who had recovered from a concussion and in controls with no concussion history. While control participants experienced improvements in attention scores at the simulated elevation of 4267 m (14,000 ft), the concussed participants did not, though they did exhibit an acute increase in negative affective changes. She attributed this improvement to an adaptive benefit of stress not experienced by those with a history of concussion. This chronic stress-induced vulnerability may be due to the effects of decreased oxygen use (at simulated elevation), but other unmeasured risk factors or stressors (like athletic exertion) might also be involved.

Elevation remains a controversial topic because research done in the 200-300 m "high" elevation condition of these studies is not generalizable to physiologically moderate elevations above 2000 m, and because the physiological explanation of the "tight fit" theory is based on an adaptation that protects woodpeckers and big-horned sheep from repetitive head impacts, and may not be generalizable to humans (Bailey et al., 2017). It is also possible that the effects of mild hypoxia at moderate elevation contributed to lowering the threshold for concussive or subconcussive damage, though it is unclear why one athlete over another would be more vulnerable to chronic damage or neurocognitive symptoms at moderate elevation. The mildly hypoxic effect of moderate elevation could also partially explain why the single treated athlete had still not fully recovered by the end of the season. Our results suggest that return-to-play guidelines at moderate and higher elevations should be more conservative, and athletes should be monitored closely to ensure they fully return to baseline performance before returning to play.

### *4.6. Limitations, strengths, & implications for future research*

This study had several limitations, including a 40% attrition rate, low power due to a small sample size, and reliance on self-report data for many of the concussions. Our information regarding treatment of the athletes post-concussion was incomplete, specifically in concussion management by the athletic training staff and how quickly the concussed athletes returned to play. These factors may have affected our results by limiting our ability to evaluate the athletes immediately after their concussion and potentially causing type II errors in our analysis. In addition, though our initial study design included a sea-level team for comparison, we were unable to establish data collection at a

second location, and therefore were unable to compare prevalence rates and recovery times between elevations.

One strength of this study is the use of an end-of-season questionnaire to identify athletes who likely sustained a concussion but did not report it, resulting in a focus on a different population than intended (though without medical evaluations we cannot say with certainty that they were concussed). This study provided some insight into the field of non-treatment-seeking athletes, which is a difficult group to study intentionally. Another strength is the use of the BDI-II to control for psychological symptoms commonly seen post-concussion that can affect cognitive functioning. Because of the inclusion of the BDI-II, the end-of-season deficits observed in the concussed athletes cannot be attributed to depressive symptoms.

We find elevation a compelling area for future study; future researchers should study larger cohorts of athletes with diagnosed and treated concussions, playing at moderate elevation and sea-level schools concurrently to assess the role elevation plays in concussion risk, severity, and recovery, and to determine the parameters of its physiological effects. Future research can also prospectively focus on comparing athletes with untreated vs treated concussions, based on a similar end-of-season screening.

Our findings also raise the possibility that an early therapeutic or pharmacological intervention in people whose depression followed the concussion may improve the injury outcome, or at least alleviate the depression to treat the other symptoms more effectively (Fann et al., 2001). Objective measures of somatic and psychological symptoms are needed to determine post-concussive damage to quality-of-life factors. A self-report symptom app might encourage reporting of symptoms, and would enable researchers the ability to schedule reminders and increase the number of assessments.

### *4.7. Conclusions*

If an athlete does not remove themself from play after a suspected concussion, the athlete's brain remains exposed to repetitive head impacts and does not have an opportunity to heal, potentially leading to longer recovery times. The concussed athletes in this study showed persistent and clinically relevant changes on neurocognitive and depression symptoms at the end of the season, outside of the expected 7-10 day recovery timeline for treated concussions without complications (Karr et al., 2014; McCrory et al., 2017). These persistent deficits can interfere with skill acquisition and the ability to protect oneself post-injury/from re-injury.

## Conflict of Interest

Neither author has any financial or institutional conflict of interests to declare. This study received no funding and was conducted through New Mexico Highlands University.

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[i] The terms "altitude" and "elevation" are used equivalently in this paper. Although other papers published in this area use the term "altitude" to describe a field's position above sea level, elevation is a slightly more accurate description. "Altitude" refers to the relative height of an object or person suspended above the ground (usually temporarily), whereas "elevation" refers to the height the ground is positioned above sea level.

[ii] Though they should be considered "potential" concussions because of their undiagnosed nature, they will be referred to as concussions in this study.

[iii] When interpreting research on subconcussion, it is important to note that while most athletes who do not report their concussion will experience further subconcussive injury/RHIs, not all athletes who experience subconcussive injury/RHIs have sustained an unreported concussion (although an unknown number have).