THE IMPACT OF CONCUSSION ON PROCESSING SPEED AND INDIVIDUAL REACTION TIME COMPONENTS

A Thesis in Psychology

by

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ABSTRACT

Mild traumatic brain injury (mTBI) is associated with an increased risk for neurocognitive and neurobehavioral deficits. Because these impairments can be difficult to detect using current neuroimaging methods, it is important to identify instruments that are brief and reliable assessments of neuropsychological functioning post injury. Slower reaction times (RTs) have been associated with mTBI. Because RT is comprised of several interactive processes including drift rate (v), boundary separation (a), and Ter, slower RTs may arise due to changes to any of these components. The goal of this study is to determine whether and which RT components are impacted by mTBI, and whether they relate to other neuropsychological variables. Performance on a 25-trial forced choice response task (the Computerized Assessment of Response Bias; CARB) was used to extract Drift Diffusion Model (DDM) estimates of v, a, and Ter. At baseline, 214 college athletes were administered a neuropsychological evaluation and had useable diffusion variables. 166 of these sustained a sports-related concussion. 7 of these participants had useable diffusion variables at a post-concussion visit. Additionally, 39 college athletes had useable diffusion variables at a post-concussion follow-up visit, irrespective of their baseline diffusion data. Baseline analyses indicated associations between v and neuropsychological functioning. However no significant relationships emerged between DDM variables and symptom severity. RT components were generally unaffected by concussion. DDM analyses did not differ among athletes who sustained a concussion versus others. This may be due primarily to the combination of the error rate demanded by the DDM and the typically low error rate of the CARB, as well as possible motivational influences in this particular population.
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Chapter 1
INTRODUCTION

In the US alone, there are one to two million reported cases of mild traumatic brain injury (mTBI; e.g. concussion) annually (Centers for Disease Control, 2007; Jennett & Frankowski, 1990; Thurman & Guerrero, 1997). Approximately 300,000 of these cases are the result of participation in sports (CDCP 1997; Sosin, Sniezek & Thurman, 1996 in Moser et al., 2007). Despite its prevalence, there is still a lack of consensus in the field regarding the definition of concussion. This in turn has had unfortunate implications for diagnosis and subsequent treatment. Currently there are limitations on diagnostic tools, including the wide use of different concussion grading systems that are unsupported by scientific data, and existing neuroimaging technology that lacks the sensitivity necessary to detect many mTBI. In many cases, neuropsychological measures provide a higher level of precision, and consequently, there is a heavy reliance on them when diagnosing sports concussion. However, due to the speed-accuracy trade offs associated with these measures, some lack of specificity remains even in these neuropsychological tests. Therefore, in effort to attain more accurate diagnoses, there is a need for more precise measures of impairment. One way this might be achieved, is by applying the Drift Diffusion Model (DDM) to suitable measures of neuropsychological functioning. By capturing the variance in performance associated in both reaction time (RT) and accuracy, this model allows a more accurate interpretation of the data, moving beyond the speed-accuracy trade off.

The goal of the present work is to (1) review issues relevant to the discussion of sports concussion such as terminology, diagnosis, symptoms, epidemiology and prognosis, (2) address the need for careful review of the psychometrics involved in widely used...
neuropsychological assessments for the diagnosis and treatment of sports concussion (3) argue for the use of more contemporary models of cognitive performance (i.e. DDM) to be used in the process of identifying the neuropsychological sequelae of sports concussion, and (4) test whether there are changes in the DDM parameters (increased $\text{Ter}$, increased/longer drift rate) at post-concussion, that would elucidate variables that might not be visible from the analysis of traditional RT and accuracy data.

**Sports Concussion Defined**

Diverging opinions on diagnostic criteria are responsible for at least 27 suggested gradation systems and new definitions continue to develop along with scientific progress (Lovell, Echemendia, Barth, & Collins, 2004). The National Academy of Neuropsychology (NAN) defines concussion as brain injury resulting from an acceleration/deceleration force(s) to the skull or within the skull, leading to linear and/or rotational movement of the brain, and the subsequent brain tissue moving against itself, increasing risk for neurocognitive and neurobehavioral deficits. (Moser, et al., 2007). The Committee of Head Injury Nomenclature of the Congress of Neurological Surgeons defines concussion as “a clinical syndrome characterized by immediate and transient post-traumatic impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc. due to brainstem involvement.” (Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons, 1996). The former definition stresses the etiological, physical aspects of concussion as the core construct, whereas the latter emphasizes the psychological impact of the injury.

Since the Traumatic Brain injury Act of 1996 became federal law, the terms *concussion* and mild traumatic brain injury (*mTBI*) have been used interchangeably (Maroon, Lovell, Norwig, Podell, Powell, Hartl, 2000). That is, traumatic brain injury (TBI), the leading cause of
death and disability in young people (Conroy & Kraus, 1998; Tepas, DiScala, Ramenofsky, & Barlow, 1990), is generally defined as evidence of brain pathology such as cerebral dysfunction, or alteration in brain function, caused by an external force (Menon, Schwab, Wright, Maas, 2010). The phrase “alteration in brain function” in the latter definition refers to any loss or decrease in consciousness, or any retrograde amnesia or post traumatic amnesia (PTA) (Menon, et al., 2010). A generally accepted definition of mTBI is a traumatically induced change in mental status with or without loss of consciousness (Collins et al., 1999a; Maroon, et al., 2000). The physical effects of TBI can be directly observable, and neuroimaging may be used to diagnose the injury. However the same is not true for mTBI; confirmation of mTBI is dependent on neuropsychological and behavioral assessments.

Though frequently used interchangeably, a consensus statement on concussion in sport, put forth by a panel of experts in the field (McCrary, Meeuwisse, Johnston, Dvorak, Aubry, Molloy & Cantu, 2009), drew a clear distinction between the terms concussion and mTBI and advised against using the two terms interchangeably. Although the panelists recognized that mTBI and concussion may indicate different injuries, they chose not to further define mTBI at that time. However, when referred to in sports, the term mTBI still typically refers to concussion (Maroon, et al., 2000; Moser, et al. 2007) and the terms mTBI, concussion, and sports concussion will be used synonymously herein referring to both the physical and the psychological effects of impact to the head that results in neuronal damage. However, the lack of consensus with respect to the term concussion negatively impacts diagnosis and course of treatment, as well as return to play (RTP) decisions that may have further consequences regarding the cumulative effects of concussion (Cantu, 1998; Maroon, et al., 2000).
Diagnosis

Symptoms most commonly found in sports concussion include dizziness, headache, nausea, confusion, memory deficits, difficulties with balance, mental fogginess, fatigue, sleep disturbances and attention problems (Erlanger, Kaushik, et al., 2003; Iverson, Gaetz, Lovell, & Collins, 2004; McCrory & Johnston, 2002; Merrit, Rabinowitz, & Arnett, 2015). Sports concussion specialist and expert in the field, Dr. Robert Cantu advises assessing the following symptoms when making a diagnosis of mild sports concussion: impairment of cortical function (e.g. recent memory), the ability to assimilate and interpret new information, and retrograde amnesia (Cantu, 1998).

According to the Zurich Consensus Statement, the following criteria should be used when a concussion is suspected (McCrory et al., 2009 p):

a.) symptoms: somatic (e.g. headache), cognitive, (e.g. feeling like in a fog), and/or emotional symptoms (e.g. lability)

b.) physical signs (e.g. loss of consciousness, amnesia)

c.) behavioral changes (e.g. irritability)

d.) cognitive impairment (e.g. slowed reaction times)

e.) sleep disturbance (e.g. drowsiness)

However, any change in an athlete’s behavior should be taken seriously, and these generic standards are not reliable tools for making RTP decisions (Collins et al., 1999). Instead, regular and repeated neuropsychological examinations within 24 hours following mTBI to detect the subtle neurocognitive effects of sports concussion is necessary (Collins, 1999). Diagnosis and RTP decisions therefore depend largely on the use of neuropsychological evaluations (McCrory
et al., 2009; Mosser, 2007). However, an athlete’s motivation at baseline and post-concussion testing can often complicate diagnosis and RTP decisions. Athletes may present what seems to be improvement on some neuropsychological assessments post injury. However, this may simply be an effect of change in motivation, as a player may wish to RTP and consequently exhibit increased motivation as compared to baseline testing (Bailey, Echemendia, & Arnett, 2006).

Mental health problems, particularly depression, are also associated with concussions (McCrorry, 2011; Teasdale, 1997). Specifically, Guskiewicz et al (2007) found that retired athletes who had a history of at least 3 concussions were three times as likely to meet criteria for a diagnosis of depression. Retired players reporting one or two previous concussions were found to be 1.5 times more likely to be depressed (Guskiewicz, 2007). There is data that suggests an increased risk of suicide for patients with a history of mTBI, however it seems that this risk might be the result of the comorbidity of other psychological issues, as well as certain psychosocial disadvantages (Teasdale, 2001). Vargas, Rabinowitz, Meyer, & Arnett (2015) found that athletes who sustained a concussion were more likely to consistently show an increase in the symptoms of depression, as well as clinically important depression. These authors also found baseline depression symptoms, baseline post-concussion symptoms, estimated premorbid intelligence, and athletes’ age at initial involvement in an organized sport, to be predictive of post-concussion depressive symptoms.

There are several gradation systems used to index concussion severity. Though LOC is included as criteria for concussion severity by some (Guskiewicz et al., 2004), most sports concussions (90%) occur without LOC, and there are differing opinions on whether or not LOC can be used as a measure of concussion severity (McCrorry, et al. 2009; Lovell et al. b, 1999).
However, there is consensus that prolonged LOC (>1 minute in duration) does influence outcome and course of treatment (Guskiewicz et al., 2003; Macciocchi et al., 1996; McCrea et al., 2003; reviewed in McCrory, et al., 2009).

Two of the most common severity grading systems are the Cantu and American Academy of Neurology (AAN; see Table 1). Since the AAN system is not based on scientific data with regard to recovery and no relationship was found between the concussion grade and level of cognitive impairment in the athlete (Hinton-Bayre & Geffen, 2002), some researchers in the field, as well as many practitioners of sports medicine, do not believe these guidelines are sufficient for making return to play decisions (Collins, et al. 1999; Lovell, 2004). In contrast, the Cantu guidelines were formulated based on empirical data that found, in opposition to the conventional assumption, the presence of LOC is not reflective of the severity of concussion injury (Maroon, et al., 2000). Despite these data, some remain uncomfortable ignoring LOC in their RTP decisions, arguing that athletes should not RTP immediately after an LOC while even minimally symptomatic (Collins, et al., 1999; Maroon, et al., 2000), (See table 1).

**Epidemiology**

In the United States, there are one to two million reported cases of mTBI annually. Approximately 300,000 of these cases are the result of participation in sports. In high school football teams, 20% of all players have at least one concussion per year (Lovell, 2004), which means high school football games alone may be responsible for approximately 200,000 of those concussions (Gerberich, Priest, Boen, Staub, & Maxwell 1983). In 2015, Voss, Connolly, Schwab, & Scher found that 50% of all university athletes sustain a concussion, and nearly 50% of all college football players reported sustaining at least one concussion before attending university (see Barth et al., 1989). Rates of concussion in all sports have been reported to be
higher at the university level rather than at the high school level (Gessel, Fields, Collins, Dick, & Comstock, 2007).

Current research is beginning to show evidence for sex difference in presenting symptoms and recovery, which may have vast implications for today’s diverse playing fields. According to the Stanford Center on Ethics, 2.9 million girls nationwide participate in high school sports (Rhode, 2007), and researchers at the Women’s Sports Foundation found that female participation in college sports has increased by 25,000 from 1995-1996 to 2004-2005 (Acosta & Carpenter, 2010). In a sample of 895 four-year colleges, 41.7% of athletes were women (Cheslock, 2007). Incidence of concussion has been found to be higher for high school girls than it is for high school boys (Gessel et al., 2007). Concussed female athletes have been shown to have longer recovery times (Covassin, Elbin, Harris, Parker & Kontos, 2012), perform significantly worse on verbal memory tasks (Covassin, 2007), have poorer reaction time, be more cognitively impaired compared to their concussed male counterparts (Broshek, 2005). These verbal memory scores are especially interesting since women have been found to outperform their male counterparts at baseline neuropsychological testing on such tasks (Covassin, et al. 2006). However, post injury, concussed men have been reported to be more symptomatic of certain effects of concussion like vomiting and sadness (Covassan, 2007).

Type of sport also impacts the prevalence of concussion among athletes. In a systematic review of the literature, Koh, Cassidy, and Watkinson (2003) evaluated the incidence of concussion among four commonly offered sports at the high school, college and amateur levels (Koh, et al. 2003). Ice hockey was shown to have the highest incidence of concussion when compared to American football, rugby and soccer (Koh, et al. 2003). However, the authors noted that the number of studies involving the risk of concussion for women in contact sports were
insufficient. Because soccer is the most commonly offered sport to women (Carpenter & Acosta, 2010), it is important to note that it has been found to carry the highest risk for sports concussion in high school girls (Powell & Barber-Foss, 1999) and girls have been shown to have a higher incidence of concussion in the sport (Covassin, Swanik, & Sachs, 2003).

**Risk Factors**

One of the strongest risk factors for a concussion is the presence of a prior concussion. In fact, the likelihood of sustaining a second concussion may be up to four times greater than that of an athlete who has not suffered a concussion (Cantu, 1998), with a greater risk of neuropsychological impairment (Iverson Gaetz, Lovell, & Collins, 2004) and neurophysiological changes occurring after three or more concussions (Gaetz, Goodman, & Weinberg, 2000).

Once a concussion is sustained there is a higher possibility of another, and the symptoms may be up to four times more likely to occur, may be more severe, and slower to resolve than initial concussions (Gerberich, 1983; Guskiewicz, 2004; Guskiewicz, 2003). This may be because the impact of a concussion triggers a neurochemical cascade that increases cerebral vulnerability and cell dysfunction (Giza and Hovda, 2004). However, these neuropathological changes may not be detected with standard structural neuroimaging (McCrorry et al., 2009). Vulnerable cells that die following a second concussion, result in increased risk for a phenomenon known as Second Impact Syndrome (Maroon, et al. 2000). Although not fully understood or well defined as a diagnosis, SIS is generally described as the incidence of a second concussion before the resolution of the signs and symptoms of the first, and as an injury to a brain already made susceptible by a previous concussion and subsequent increased intracranial pressure (ICP) and subclinical edema (Bender, Barth, & Irby, 2004). Decreased blood flow and
further ICP from the second impact results in severe tissue damage and may be associated with vascular congestion (Alves & Polin, 1996).

The effects of SIS are cumulative in nature, making even seemingly minor impacts dangerous (Cantu, 1998; Guskiewicz, et al., 2003). Since an individual still recovering from a concussion is at greater risk for injury, the striking blow required for SIS to develop does not have to be directly to the head; any force that will jolt the head is sufficient to SIS (Cantu, 1998).

Initially, SIS may present similarly to a grade one concussion. That is, the athlete will likely be able to finish the play, while seemingly in a mental fog (Cantu, 1998). However, unlike a single concussion, in 15 seconds to several minutes the athlete will collapse into a semicomatose state, displaying other physical symptoms such as loss of eye movement, rapidly dilating pupils, and respiratory failure. (Cantu, 1998)

Pre-existing cognitive dysfunction (Collins, 1999) and psychiatric conditions, such as anxiety, depression, and conduct disorder (Vassallo, 2007), may also increase the risk of concussion. The perceived risk is approximately 1.57 times greater in the presence of such pre-existing conditions, and is thought to be greater for young adults than it is for adolescents (Teasdale, 1997). Since mood and conduct disorders have been found to increase the risk of head injury, earlier detection may aid in prevention of mTBI (Vassallo, 2007). A relationship between pre-existing learning disability (LD) and cognitive decline post-concussion has been established in a study of a college football population by administering computerized neuropsychological evaluations at baseline and post-concussion (Collins, 1999).

There is also scientific evidence pointing toward the possibility of a genetic predisposition to the effects of concussion (Jordan, 2004). There are at least two studies suggesting a relationship between the Apolipoprotein E (APOE) genotype and the neurological
consequence of sports concussion (Jordan, Relkin, & Ravdin, 1997; Kutner, Ehrlanger & Tsai, 2000). APOE is a cholesterol carrying molecule in the central nervous system that is associated with the metabolic changes that occur after concussion. Jordan et al. (1997) studied 30 boxers, both active and retired, and found that those with the APOE 4 allele showed more cognitive deficits, such as memory, attention, and concentration, than those without the genotype (Maroon, 2000). A review of the clinical literature shows several studies suggesting the presence of APOE predisposes individuals to the neurocognitive effects of TBI (Jordan, 2004). Further supporting this idea of genetic risk, Merrit & Arnett (2016) recently found increased reporting of concussion symptoms in APOE 4 positive athletes. Genetic testing is available, but is not without controversy (Jordan, 2004) and therefore may be underutilized.

Prognosis and Prevention:

80-90% of sports concussion symptoms resolve within 7-10 days (McCrory et al., 2009). The neuropsychological issues associated with mTBI are typically resolved within 1-3 months after the event, (Dikmen, Machamer, & Temkin, 2001), with most athletes recovering within one month (Mosser, 2007). Several meta-analyses support this view (Rohling, Binder, Demakis, Larrabee, Ploetz, & Langhinrichsen-Rohling, 2011; Belanger et al., 2005; Schretlen & Shapiro, 2003) and a recent meta-analysis of these data advanced this perspective by concluding that the neuropsychological effects of mTBI are initially small and resolve quickly (Rohling, et al., 2011). While many earlier findings have associated LOC with cognitive deficits (McCrory, et al. 2009), one study found that athletes with LOC did not seem to be more likely to exhibit cognitive impairment (Hinton-Bayre & Geffen, 2002).

However, reports of the long-term effects of concussion are becoming increasingly more prevalent in the literature, revealing somewhat chronic pan symptomatology involving
emotional, physical and cognitive domains (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005). Persistent neurobehavioral problems have been found in mTBI patients (Hartlage, Durant-Wilson, Patch, 2001). A recent study by Konrad et al. (2011) yielded compelling findings with regard to the long-term psychological effects of mTBI. In this study, subjects were found to have maintained depressive symptoms more than half a decade after the injury. A total of 66 subjects (33 healthy controls), were assessed for psychiatric symptoms using the Beck Depression Inventory (BDI) and the Structured Clinical Interview for DSM IV for Axis I Disorders (SCID-I). The patient group scored higher on the BDI and three of these subjects were found to be clinically symptomatic of major depression, suggesting that the psychological effects of mTBI may be longer lasting than once thought. Further, Post-Concussive Syndrome (PCS) can lead to other long-term negative consequences in neuropsychological functioning. For example, executive function (i.e. inhibition) was found to be impaired in PCS patients at three months post-concussion on the interference measure of the Stroop Color-Word test (Bohnen, Twijnstra, & Jolles, 1992).

Because the effects of concussion are quite heterogeneous, because the physical changes due to a concussion are not identifiable using current imaging technology, and because return-to-play decisions (RTP) are critical for athletes, it is imperative that research improves both the ability to identify the presence of a concussion, as well as the ability to monitor recovery more effectively. In pursuit of this goal, developing effective neuropsychological indices that can be administered at brief test-retest intervals, and can reliability predict current functioning, is critical (Iverson, Lovell, & Collins, 2003).
Executive Function

Executive function (EF) refers to a broad and abstract set of processes such as selective attention, behavioral inhibition, working memory, and rule-based or goal-directed behavior (Barkley 1997; Miller & Cohen 2001; Miyake, Friedman, Emerson, Witzki, Howerton, & Wager, 2000). They are internally guided, dependent upon the healthy functioning of the prefrontal cortices, and allow individuals to respond flexibly to novel and rapidly changing situations (Miller & Cohen, 2001).

Although there are many different models of EF, most agree that there are three major processes: inhibitory control, working memory, and mental set shifting (Miyake et al. 2000). Although these processes are moderately correlated, they are not unitary and are clearly distinguishable from one another (Miyake et al., 2000). Whereas Miyake et al. (2000) conceptualizes EF processes as non-hierarchical, Barkley (1997) has proposed a different model. Barkley asserts behavioral inhibitory control (i.e. the inhibition, delay, or stopping of an ongoing response to a stimulus, and the protection of response process from competing stimuli) is required for the healthy execution of four other executive processes. These processes include working memory, self-regulation, internalization of speech, and reconstitution (Miller & Cohen, 2001). Together, these functions have the ability to generate novel complex solutions in the service of goal directed behavior (Miller & Cohen, 2001).

EF Impairment post mTBI

Regardless of the cognitive architecture of EF, deficits in this area are often, but not always, found in mTBI (Barth, Macciocchi, Giordani, Rimel, Jane, & Boll, 1983; Cantu 1998; Collins, Lovell, McKeag 1999; Collins, et al., 1999, McCrory et al. 2009). Following concussions, studies have documented impairments in working memory (Erlanger 1999;
Guskiewicz, 2001; Iverson 2005), inhibitory control (Bohnen, Twijnstra, Jolles, 1992; Echemendia, Putukian, Mackin, Julian, & Shoss, 2001), and processing speed (Bleiberg, Halpern, Reeves, & Daniel, 1998; Collins 1999; Cremona, et al. 1994; Echemendia, et al. 2001; Iverson, et al. 2003; McDowell, Whyte, & D'Esposito, 1997; Stuss 1985). However, when such deficits are found, they are not always long lasting (Belanger, 2005; Shretlen & Shapiro, 2003), present in a significant proportion of injured patients (Barth et al., 1983; Binder, 1997), or consistently present across different measures of EF or processing speed (Brooks, Fos, Greve, & Hammond., 1999; Erlanger et al., 2001; Maddocks & Saling, 1996; Bleiberg et al., 1998; McDowell et al., 1997). This apparent discrepancy suggests that traditional EF or traditional processing speed tasks might not be the most sensitive indices of impairment. In particular, paper-and-pencil measures (e.g. TMT) that are traditionally used to measure processing speed require heavy fine motor and literacy skills which can confound interpretations of poor performance.

To address these confounds, a number of forced choice reaction time tasks have been developed to better index global processing speed (Bleiberg et al., 1998). This advance has been illustrated by studies which have used both traditional paper and pencil as well as reaction time tasks on similar populations that have found group differences in the latter but not the former (Barth et al., 1983; Bleiberg et al., 1998; Brooks, Fos, Greve, et al., 1999; Cremona et al., 1994; Erlanger et al., 2001; Maddocks & Saling, 1996; McDowell et al., 1997).

Research in developmental and individual differences in working memory, a prototypical EF, is predicted by developmental and individual differences in global processing speed, typically measured by forced choice reaction time tasks (Kail & Salthouse, 1994; Karalunas & Huang-Pollock, 2013). Because of this robust association, brief measures of processing speed
(including simple FCRT), may eventually prove to be a more efficient way of assessing post mTBI impairment as opposed to a lengthy battery of general EF or traditional speeded tasks that can be confounded by literacy and significant fine motor skills. If such evaluations could be validated, then they would have important clinical implications for immediate RTP decisions made on the field. Additionally, this sort of measure can be helpful more generally in making decisions that could result in earlier intervention of mTBI.

**Drift Diffusion Model**

While computerized assessments can be used to index RT, they still may not be the most sensitive measures for that purpose. Traditionally psychological science has separately examined RT from error rates/accuracy. However, when these variables are analyzed independently, the presence of speed-accuracy trade-offs confound interpretation of RT. For example: Amy has very fast RTs, but also but commits lots of errors. Does this mean that her processing speed is faster than Rich’s, who responds more slowly, but more accurately? Or could it be that Amy’s processing speed only appears faster because she is more impulsive than Rich (Table 2)? Therefore, what is needed is a variable or set of variables that is able to simultaneously capture the variance in performance associated in both RT and accuracy.

To be able to accurately describe how fast or efficiently a person can process information, it is also important to separate the speed and efficiency of an individual’s central cognitive processing speed from the time it takes to encode a stimulus and to prepare a motor response (Wagenmaker, et al., 2007). Ratcliff’s diffusion model is a computational modeling procedure that is able to both account for speed-accuracy trade-offs, as well as separate the variance in performance due to encoding and motor speed from that of central cognitive processing efficiency. The diffusion model assumes that simple forced choice decisions are made
after a noisy information accumulation process. As the information accumulates, it moves towards one of two boundaries, and when the process reaches a boundary, the corresponding response is initiated (Fig. 1). This model uses the reaction time distributions for correct and incorrect forced choice response tasks into these separable components. Within this rubric, drift rate ($v$) refers to the speed of information processing, and is associated with task-difficulty as well as individual differences in processing speed. Boundary separation ($a$) indicates the quality or amount of information used to make the decision, and is related to the confidence in the choice. $Ter$ refers to non-decision time, comprised of encoding processes and motor preparation (Karalunas & Huang-Pollock, 2013; Voss et al., 2013).

Thus, the diffusion variables, drift rate, boundary separation, and $Ter$, can lead to a more thorough understanding of what is influencing an individual’s performance on speeded reaction time tasks, and elucidate differences between groups that may otherwise look perfectly comparable. For example, what could appear as no difference between groups could actually be important differences in process, if one group is more impulsive than the other. Using the above example of Amy and Rich, Amy, as the more impulsive individual might have a smaller boundary separation, but slower drift rates, than Rich. Thus, even though her overall RT is faster (or equivalent to) Rich’s, and might in a standard situation be considered an index of normal or healthy functioning, the speed of her central cognitive processing ability as indexed by drift rate, would in fact reveal possible deficits in functioning. The diffusion model holds potential for future research that can inform clinical practice, treatment (White, Ratcliff, Vasey, McKoon 2010), and RTP decision-making.

One of the limitations of the full diffusion model, however, is that as a model fitting procedure, it requires a substantial number of trial by trial data and a moderate error rate, both of
which are often not available (Wagenmaker et al., 2007). This makes the Ratcliff model impractical in many laboratory settings. Recently, attempts have been made to develop simplified methods to estimate the diffusion parameters in order to popularize its use. The EZ Diffusion model is one such method. Rather than requiring the full RT distribution of error and correct trials, the EZ diffusion model simply requires the mean reaction time (MRT), reaction time variance (RTV), and response accuracy (Pc) (Wagenmakers et al., 2007). The EZ diffusion model is a simplified version of the diffusion model that does not require parameter fitting and can be applied to common experimental setups (Wagenmakers, Mass, & Grasman, 2007), making it a good candidate for an experiment with fewer trials or when trial-by-trial data is not available.

Slower reaction times often observed among individuals following mTBI (Guskiewicz et al., 2007) might arise as a function of changes to any of the components that contribute to RT. The degree to which these RT components are related to other neuropsychological processes is also not clear. In the proposed study, I plan to evaluate whether and which reaction time components may be impacted by mTBI, and whether any components are related to other neuropsychological variables. Hypotheses are as follows:

**Hypotheses**

**Hypothesis 1:** It’s important to evaluate premorbid group differences in neuropsychological functioning that would confound interpretation of post-concussion group differences. It is predicted that there will be no baseline group differences among athletes who have never had a concussion, and athletes who later had a concussion on any measure of neuropsychological, cognitive, or socioemotional functioning. This will be addressed with an ANOVA with the group
(never concussed, future concussed) as the independent variable, and measures of neuropsychological, cognitive, or socioemotional functioning, as the dependent variables. If hypothesis 1 supported, no significant group differences will be found on any dependent variable.

**Hypothesis 2:** Consistent with previous literature, declines in neuropsychological functioning are expected post-concussion. To test this, a paired-sample t-test comparing performance on standard measures of neuropsychological performance (Vigil, CARB, and ImPACT) pre and post-concussion will be conducted. If Hypothesis 2 is supported, then performance post-concussion will be worse than baseline.

**Hypothesis 3:** Following concussion there will be changes in the DDM parameters (increased $Ter$, slower drift rate) that may not be visible from the analysis of traditional RT and accuracy data. No directional hypotheses are proposed for boundary separation, but any pre-post concussion changes will be evaluated. A paired sample t-test will be used to compare within participant changes in the DDM variables ($Ter$, drift rate, boundary separation) at baseline and post-concussion. In addition, an ANOVA will also be used to compare independent groups of participants who have useable DDM variables only at baseline vs. those who have useable DDM variables only at post-concussion to evaluate whether changes in the DDM variables are observed post-concussion in a cross sectional sample.

**Hypothesis 4:** There will be an association of drift rate and $Ter$ during the first post-concussion assessment with the concussion severity at time of injury as indexed by the ImPACT Total Symptom Score (ImPACT TSS). Because the distribution of ImPACT TSS scores is anticipated to be dichotomous, this hypothesis will be evaluated using a between groups ANOVA with a
median split. If hypothesis 4 is supported, participants reporting a greater number of concussion symptoms on the ImPACT will have slower drift rates and slower Ter.

**Hypothesis 5:** If drift rate is a valid index of neuropsychological functioning, then it should be correlated with other measures of neuropsychological functioning. To evaluate this, a Pearson’s correlation coefficient analysis of neuropsychological performance and baseline drift rate will be conducted. If Hypothesis 5 is supported, then there will be a positive association between baseline drift rate and performance on other neuropsychological tasks.
Chapter 2

METHODS

Participants

Current and past students of Penn State University (n=1346; male, 69.9%, female 30.1%; mean age, 18.73, SD 1.25) underwent neuropsychological baseline testing on campus. Participants include student athletes from football, ice hockey, soccer, wrestling, basketball and lacrosse, as well as individuals from the general university participant pool. All testing was conducted at the University Park campus of Penn State University by a graduate, or undergraduate, student trained in the administration of the assessment battery.

Measures

Drift Diffusion Variables.

Continuous Assessment of Response Bias (CARB). The CARB is a 25-trial, computer-administered, forced-choice recognition task that was used to estimate the RT components of speed and accuracy from the following variables: percent correct (Pc), mean RT (MRT), and RT variance (VRT). During this task, participants are shown a series of paired stimuli (i.e. digit spans), and are later required to indicate which stimulus was presented earlier. Performance on the CARB was used to extract estimates of drift rate, boundary separation, and Ter.

The calculation of DDM variables requires the presence of errors. Of the total sample, 214 (16% of the sample) college athletes (151 male; 63 female) had useable diffusion variables at baseline. Of these 214, 166 had sustained a concussion. Of these 166 who had usable diffusion variables at baseline and later had concussions, 7 committed at least one error on the CARB and
useable diffusion variables at a post-concussion visit. Consequently, of the 214 participants who at baseline had useable DDM variables, only 7 had useable diffusion variables at post-concussion. Additionally, of the total sample, 39 (3% of the sample; 26 male: 13 female) had useable diffusion variables at a post-concussion follow-up visit, regardless of the presence or utility of baseline diffusion data.

Neuropsychological and other measures of well being

**Vigil (Cegalis & Cegalis, 1994).** The Vigil is a computerized, continuous performance task designed to measure reaction time, sustained attention and concentration (Cegalis & Cegalis, 1994), and is used in concussion assessment (Bailey et al., 2006; Echemendia & Julian, 2001; Echemendia et al., 2001). During this task, different letters flash across the computer screen one at a time and participants are instructed to respond to a target letter (“K”) as quickly as possible by pressing the space bar.

**Immediate Post-concussion Assessment and Cognitive Testing (ImPACT: Lovell, Collins, Podell, Powell, & Maroon, 2000).** The ImPACT is a 25-minute computerized assessment battery that includes neuropsychological measures of attention, memory, processing speed, and reaction time, as well as the Post-Concussion Symptom Scale. The test has been shown to be reliable and valid, and resistant to practice effects (Iverson, Lovell, & Collins, 2003; Iverson, Gaetz, Lovell, & Collins, 2005).

**Wechsler Test of Adult Reading (WTAR: Holdnack, 2001).** Participants were asked to read aloud from a list consisting of 50 words of increasing difficulty (e.g. fierce, conscientious, vertiginous, etc.). Each correctly pronounced word is worth one point. The WAIS-III Full scale IQ (FSIQ) is estimated by using normative tables (age, gender, education, and education corrected) to convert the raw scores.
Beck Depression Inventory-Fast Screen (BDI-FS: Beck et al., 2000). The BDI-FS is a brief self-report measure of depression consisting of seven items. Using a 3-point scale, participants rate depressive symptoms experienced during the past two weeks. Mild symptoms are indicated by low ratings, and symptom severity by higher ratings. This abbreviated measure of depression exhibits good internal consistency (α = .84) and concurrent validity with the BDI-II (r = .85) and Center for Epidemiologic Studies Depression scale (r = .86).
Chapter 3

RESULTS

Baseline differences in functioning. At baseline, there were no significant group differences between participants who later sustained a concussion and those who did not in estimated full scale IQ, as measured by the WTAR (F(1,1206) = 1.53, p = .22, η² = 0.01) or in the ratio of males to females (X²(1, N = 1276) = 2.51, p = .11). While most baseline measures in neuropsychological, cognitive, or socioemotional functioning between these groups were not significant, (all p > .17, n² < .002), athletes who later sustained a concussion committed more errors on the CARB at baseline (baseline CARB summary % correct, F(1,1131), p = .005, η² = 0.07) and performed worse with respect to visual-motor response speed, (including visual processing, learning and memory) compared to athletes who did not later sustain injury (ImPACT Visual Motor Speed Composite: F(1,1225), p = .03, η² = .004). There was also a significant difference in age (F(1, 936) = 14.74, p < .001, η² = .015), although the practical difference was so small, there was no meaningful age difference (both groups had a mean age of 18 years).

These results indicate that, in general, participants who were later injured and those who were never injured had similar premorbid cognitive and socioemotional functioning.

Comparing neuropsychological functioning at pre- and post-concussion time points. Compared to baseline, there were significant changes to performance post-concussion for four of the variables examined. Average reaction time on the VIGIL, visual recall and word memory on the ImPACT declined post-concussion (Vigil: Average Delay, t(87) = 3.12, p = .002; ImPACT design Memory: hits delay, t(83) = 2.98, p = .004; ImPACT word memory: total % correct, t
(86)=2.150, p=.034; ImPACT word memory: delayed memory % correct, t (86)=2.040, p=.044). These findings are consistent with previous literature, as declines in neuropsychological functioning are expected post-concussion. However, no other changes were found on other examined measures of neuropsychological functioning. There were no significant changes in overall accuracy on the CARB or Vigil, nor was significant change found for inattention, impulsivity, superfluous responding (perseverations) on the Vigil (Vigil Omissions, Vigil Commissions, Vigil perseverations, Hit Rate), or overall neuropsychological functioning on the ImPACT (Memory Composites –Verbal and Visual, Visual motor speed composite, Impulse control composite, Reaction time composite) (All, p = >.08). Similarly, neither self-reported ratings of depression (BDI), nor Full Scale IQ (WTAR estimated FSIQ) changed significantly (All, p = >.08).

These results indicate that, in general, there were no significant changes in neuropsychological functioning post-concussion.

Comparing Drift Diffusion variables at pre- and post-concussion time points. Compared to baseline, there were no significant changes in the DDM parameters at post-concussion, all p>.22 (drift rate t (6)= -1.36, p=.22; boundary separation t (6)= -1.31, p=.24; Ter (6)= -1.23, p=.26). However, of the sample, only N = 7 had DDM parameters that could be calculated at both pre and post-concussion time points. Thus, in addition to this paired sample t-test, a between groups ANOVA was also run to compare participants at baseline whose DDM parameters could be calculated (n = 204), with participants at a post-concussion time point whose DDM parameters could be calculated (n = 39). Results did not change (All, p > .34, all $\eta^2 < .004$).
Thus, similar to the lack of findings for neuropsychological measures, there were no changes in the DDM parameters derived from the CARB post-concussion.

**Assessing relationship between the number of concussion symptoms and Drift diffusion variables.** Expected associations between examined DDM variables and ImPACT Total Symptom Score (ImPACT TSS) were not found. There were no significant correlations between drift rate ($r=-.250, p=.12$), boundary separation ($r=.02, p=.92$), and $Ter$ ($r=-.07, p=.69$) during the first post-concussion assessment with the concussion severity at time of injury, as indexed by the ImPACT TSS. When a median split was used to examine concussion severity dichotomously, there were no group differences among participants who reported a greater number of concussion symptoms vs. those who reported fewer symptoms (all $p > .55$, $n^2 = .009$).

**Assessing relationship between the baseline drift rate and performance on other neuropsychological tasks.** Expected positive associations between baseline drift rate performance and two measures of neuropsychological functioning were found (BL CARB summary percent correct, $r=.44, p = .00$; BL ImPACT symbol match: total correct symbols, $r=.15, p = .03$), indicating that faster information processing speed is associated with better neuropsychological performance. One additional variable (ImPACT design memory hits delay, $r=.13, p=.064$) approached significance in the same direction, suggesting further possible support for this idea. Expected negative associations were also found on the CARB Summary Total Correct ($r= -.20, p= .003$) and several indices of the Vigil (BL Vigil omissions, $r= -.16, p= .02$; BL Vigil commissions, $r= -.15, p= .03$; BL vigil perseverations, $r= -.15, p= .03$; BL Vigil False Alarm Rate, $r= -.17, p= .01$), further indicating a relationship between speeded information processing and better neuropsychological performance.
Additionally, BL ImPACT Impulse control composite approached significance (r = -0.13, p = .066), lending possible support for this relationship.

While such expected associations were found, unexpected negative correlations were also found between baseline drift rate and performance on the BL ImPACT Color match: total correct (r = -0.14, p = 0.04). These findings indicate that slower information processing speed was associated with better neuropsychological functioning. There were no significant associations found between drift rate and other indices of neuropsychological performance; nor were such associations found for measures of neuropsychological functioning such as estimated full scale IQ and depression.
Chapter 4
DISCUSSION

Limitations of diagnostic tools, such as concussion grading systems and neuroimaging technology, have resulted in a heavy reliance on computerized neuropsychological measures that can provide a higher level of sensitivity. However, even these measures are limited because they analyze RT and accuracy independently, increasing susceptibility to speed-accuracy trade offs; that is, faster response rates can lead to more errors and confound RT interpretation. Therefore the present work sought to increase this level of diagnostic insight by applying a procedure that can simultaneously capture the variance in performance associated in both RT and accuracy. The Drift Diffusion Model achieves this by including the RT variance in addition to the mean RT and error rate. This computational model of RTs parses out variance associated with drift rate, boundary separation, and ter. Briefly defined, drift rate ($v$) is associated with processing speed, boundary separation ($a$) relates to conservativeness in responding, and Ter is non-decision time (encoding and motor prep). This more contemporary model of cognitive performance could elucidate RT variables otherwise not visible from traditional analysis, and could more precisely identify the neuropsychological sequelae of sports concussion.

At baseline, there were no indications of premorbid differences in neuropsychological, cognitive, or socioemotional functioning. The premorbid statistical difference found in age between the injured and non-injured group, was not clinically meaningful (as both groups had a mean age of 18) and was likely an effect of the very large total sample size used at baseline. This means any difference in neuropsychological performance between students who later sustain a concussion can be reasonably attributed to injury and not to any pre-existing differences.
Post-concussion differences in neuropsychological functioning between injured and non-injured participants were examined using traditional outcome measures and were compared with a paired sample t-test. While significant differences were found on four indices of neuropsychological functioning (Vigil Average Delay, ImPACT Design Memory: hits delay, ImPACT word memory: total % correct, and ImPACT word memory: delayed memory % correct), overall, the pattern of data does not indicate significant changes in neuropsychological functioning post-concussion. In contrast to this finding, a worsening in neuropsychological performance at post-concussion has been found in other studies (Echemendia, et al., 2001; Guskiewicz, 2001; Iverson, 2005; Iverson, et al., 2003), and the ImPACT is known to be sensitive to the effects of mTBI (Iverson, Lovell, Collins 2005; Iverson, et al. 2003; Schatz, Pardini, Lovell, Collins & Podell, 2006). Possible reasons for this disparity include: (1) some student athletes might have underreported symptoms in effort to be cleared for RTP, reflecting current literature that finds increased motivation at post-concussion testing among athletes for this reason (Bailey, Echemendia, & Arnett, 2006); (2) it is possible that the sample examined in the present work is skewed toward athletes with fewer, or no prior history of, concussions (multiple concussions have been associated with an increased drop in memory and RT; as much as 7.7 more likely in the former [Covassin, Moran, & Wilhelm, 2013; Iverson, Echemendia, LaMarre, Brooks & Gaetz, 2012; Iverson, et al. 2004]); (3) symptoms could have been shorter in duration for a significant portion of the population (Belanger, 2005; Shretlen & Shapiro, 2003; Echemendia, et al., 2001), in which case, post-concussion assessment could have taken place after symptoms resolved and/or; (4) this finding could be due to too much variation in timing of the post injury assessment. That is, amount of time between concussion and post-concussion
evaluation varied so that acute symptoms of concussion could have resolved by the time of testing.

The drift rate ($v$), boundary separation ($a$), and non-decision time ($Ter$) of both groups at baseline and post-concussion were compared using a paired sample t-test. Despite support in the literature for increasing RT post-concussion, we did not find evidence of such slowing; diffusion parameters did not differ among athletes who had concussions versus those who had not. There are two likely reasons for this: First, the CARB may not sensitive to the effects of mTBI, as it was designed to detect malingering and not as a measure of concussion. However, the CARB contains both the RT and variance required for extracting the three DDM variables (MRT, variance and percent correct) and therefore was appropriate to that purpose. Second, a small sample size resulted from the combination of the error rate demanded by the drift diffusion model, and the high level of accuracy associated with performance on the CARB. For this reason, the data were also analyzed with a univariate ANOVA; this increased the sample size, but results did not change.

The expected positive relationship between DDM variables and post-concussion symptom severity was also not found. Athletes have been shown to report a greater number of symptoms two days post-injury, with full resolution by one week (Covassin, Elbin & Nakayama, 2010). It is possible that our finding is due to the same potential confounding issues of variation in assessment timing, and motivation, mentioned above.

Validating the interpretation of drift rate, drift rate from the CARB, Vigil, and ImPACT was associated with performance on other neuropsychological tasks. As expected, increased drift rate, or faster information processing, was significantly associated with better
neuropsychological functioning (BL CARB: summary percent correct, summary total correct; BL Vigil: omissions, commissions, perseverations, False Alarm Rate; and BL ImPACT symbol match: total correct symbols), and other results approached significance (BL ImPACT Impulse control composite; BL ImPACT design memory hits delay). However, an unexpected association was also observed, indicating a relationship between slower information processing speed and better neuropsychological functioning (BL ImPACT color match: total correct,), which may simply be the result of Type I error given multiple analyses.

Limitations & Future Directions

More research is needed in this area. Because the drift diffusion model requires a moderate error rate, further scientific study could benefit from extracting these parameters from a task of moderate or high difficulty (as opposed to the CARB). This would increase the likelihood of the model yielding more meaningful data. With respect to the relationship between DDM variables and concussion symptoms, future research could benefit from the examination of samples that underwent neuropsychological testing and reporting within one week from injury and who had similar concussion histories. This would increase the likelihood that pooled participants would have more common symptomatology, thereby clarifying any relationship between symptoms and the components of RT.
Conclusion

Overall, we present the diffusion model as having the potential to expound traditional interpretation of RT data, thereby elucidating the decision-making processes associated with the neuropsychological evaluation of sports concussion. Using drift diffusion variables as indices of speed of central cognitive processing ability, strategy, and non-decision time, the diffusion model can reveal possible deficits in functioning that would otherwise be unobserved. In this way, the diffusion model can move past current testing limitations related to traditional data analysis (i.e. speed-accuracy trade offs), and be used as a valuable research tool with clinical applications that inform diagnosis, treatment options, and RTP decisions (White, Ratcliff, Vasey, McKoon 2010). Future work applying the DDM model to concussion data in this manner could help clarify the confounding issues inherent in traditional analysis of RT data, and lead to greater specificity in diagnosis, better treatment, and improved RTP decisions.
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APPENDIX

Table 1. Cantu concussion grading system

<table>
<thead>
<tr>
<th>Author</th>
<th>Grade</th>
<th>Definitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cantu</td>
<td>Grade 1</td>
<td>No LOC; PTA &lt; 30 min</td>
</tr>
<tr>
<td>Cantu</td>
<td>Grade 2</td>
<td>LOC &lt; 5 min; PTA &gt; 30 min &amp; &lt; 24h</td>
</tr>
<tr>
<td>Cantu</td>
<td>Grade 3</td>
<td>LOC &gt; 5 min or PTA &gt; 24h</td>
</tr>
<tr>
<td>AAN</td>
<td>Grade 1</td>
<td>Transient confusion, no LOC, symptoms resolve in &lt; 15 min</td>
</tr>
<tr>
<td>AAN</td>
<td>Grade 2</td>
<td>Transient confusion, no LOC, symptoms last &gt; 15 min</td>
</tr>
<tr>
<td>AAN</td>
<td>Grade 3</td>
<td>LOC of any duration</td>
</tr>
</tbody>
</table>

PTA = post-traumatic amnesia

Adapted from Lovell, Echemendia, Barth, & Collins (2004)

Table 2. Example: Drift Diffusion

<table>
<thead>
<tr>
<th>Participant</th>
<th>MRT</th>
<th>RTV</th>
<th>Pc</th>
</tr>
</thead>
<tbody>
<tr>
<td>George</td>
<td>0.517</td>
<td>0.024</td>
<td>0.953</td>
</tr>
<tr>
<td>Rich</td>
<td>0.467</td>
<td>0.024</td>
<td>0.953</td>
</tr>
<tr>
<td>Amy</td>
<td>0.422</td>
<td>0.009</td>
<td>0.881</td>
</tr>
<tr>
<td>Mark</td>
<td>0.372</td>
<td>0.009</td>
<td>0.881</td>
</tr>
</tbody>
</table>

Adapted from Wagenmakers, Mass, & Grasman, (2007)
Table 3A. Sample demographics. Means and standard deviations provided in parentheses.

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Never injured</th>
<th>Injured at some point</th>
<th>F-test ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>976</td>
<td>18.53 (1.09)</td>
<td>18.93 (1.19)</td>
<td>F (1, 974)= 17.01, p&lt;.00, η² =.012</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=814</td>
<td>n=162</td>
<td></td>
</tr>
<tr>
<td>Est. FSIQ (WTAR)</td>
<td>1208</td>
<td>103.98 (7.56)</td>
<td>102.97 (5.92)</td>
<td>F (1,1206)=1.526, p=.217, η² =.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=1120</td>
<td>n=88</td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>1236</td>
<td>1.20 (1.81)</td>
<td>1.39 (2.23)</td>
<td>F (1,1234)=.872, p=.351, η² =.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=1144</td>
<td>n=92</td>
<td></td>
</tr>
<tr>
<td>CARB summary % correct</td>
<td>1133</td>
<td>99.14 (2.46)</td>
<td>98.30 (4.65)</td>
<td>F (1,1131)=7.84, p=.005, η² =.007</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=1046</td>
<td>n=87</td>
<td></td>
</tr>
<tr>
<td>ImPACT memory composite-verbal</td>
<td>1228</td>
<td>86.22 (29.13)</td>
<td>83.63 (11.85)</td>
<td>F (1,1226)=.778, p=.378, η² =.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=1128</td>
<td>n=100</td>
<td></td>
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<tr>
<td>ImPACT memory composite-visual</td>
<td>1216</td>
<td>77.46 (22.03)</td>
<td>75.43 (13.59)</td>
<td>F (1,1214)=.738, p=.390, η² =.001</td>
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<td></td>
<td></td>
<td>n=1127</td>
<td>n=89</td>
<td></td>
</tr>
<tr>
<td>ImPACT Visual Motor Speed Composite</td>
<td>1227</td>
<td>37.95 (14.00)</td>
<td>34.81 (8.09)</td>
<td>F (1,1225)=4.90, p=.027, η² =.004</td>
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<td></td>
<td></td>
<td>n=1127</td>
<td>n=100</td>
<td></td>
</tr>
<tr>
<td>ImPACT Impulse control Composite</td>
<td>1227</td>
<td>9.39 (14.78)</td>
<td>11.56 (19.92)</td>
<td>F (1,1225)=1.85, p=.174, η² =.002</td>
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<tr>
<td></td>
<td></td>
<td>n=1127</td>
<td>n=100</td>
<td></td>
</tr>
<tr>
<td>ImPACT Reaction time composite</td>
<td>1227</td>
<td>.80 (3.45)</td>
<td>.60 (.08)</td>
<td>F (1,1225)=.325, p=.569, η² =.000</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n=1127</td>
<td>n=100</td>
<td></td>
</tr>
</tbody>
</table>

Table 3B. Sample demographics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Never injured</th>
<th>Injured at some point</th>
<th>X²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:female</td>
<td>574:202</td>
<td>22:41</td>
<td>X²=.05, p=.82</td>
</tr>
</tbody>
</table>
Table 4.

<table>
<thead>
<tr>
<th>H2 Variable (NP function)</th>
<th>n</th>
<th>Pre-concussion</th>
<th>Post-concussion</th>
<th>Paired Sample T-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARB summary % correct</td>
<td>84</td>
<td>98.24 (4.72)</td>
<td>98.43 (3.52)</td>
<td>t(83) = -.304, p = .762</td>
</tr>
<tr>
<td>Vigil Omissions</td>
<td>88</td>
<td>1.91 (3.65)</td>
<td>2.48 (4.46)</td>
<td>t(87) = -.961, p = .339</td>
</tr>
<tr>
<td>Vigil Commissions</td>
<td>88</td>
<td>2.52 (2.11)</td>
<td>2.92 (3.96)</td>
<td>t(87) = -.931, p = .354</td>
</tr>
<tr>
<td>Vigil Perseverations</td>
<td>88</td>
<td>.44 (1.73)</td>
<td>.11 (.49)</td>
<td>t(87) = 1.777, p = .079</td>
</tr>
<tr>
<td>Vigil hit Rate</td>
<td>88</td>
<td>.97 (.10)</td>
<td>.98 (.04)</td>
<td>t(87) = 5.80, p = .564</td>
</tr>
<tr>
<td>Vigil False alarm rate</td>
<td>88</td>
<td>.009 (.01)</td>
<td>.01 (.02)</td>
<td>t(87) = -.921, p = .360</td>
</tr>
<tr>
<td>Vigil Average Delay</td>
<td>88</td>
<td>413.31 (39.74)</td>
<td>428.00 (49.62)</td>
<td>t(87) = -3.115, p = .002</td>
</tr>
<tr>
<td>FSIQ (WTAR)</td>
<td>64</td>
<td>102.42 (5.62)</td>
<td>101.67 (14.98)</td>
<td>t(63) = .472, p = .638</td>
</tr>
<tr>
<td>BDI fastscreen total</td>
<td>86</td>
<td>1.35 (2.21)</td>
<td>1.85 (2.25)</td>
<td>t(85) = 1.782, p = .078</td>
</tr>
<tr>
<td>ImPACT Memory Composite-Verbal</td>
<td>84</td>
<td>83.59 (12.24)</td>
<td>81.48 (11.91)</td>
<td>t(83) = 1.548, p = .125</td>
</tr>
<tr>
<td>ImPACT Memory Composite-Visual</td>
<td>84</td>
<td>74.98 (13.59)</td>
<td>74.18 (14.31)</td>
<td>t(83) = .493, p = .623</td>
</tr>
<tr>
<td>ImPACT Visual motor speed Composite</td>
<td>87</td>
<td>34.28 (8.06)</td>
<td>35.44 (8.32)</td>
<td>t(86) = 1.086, p = .281</td>
</tr>
<tr>
<td>ImPACT Impulse control Composite</td>
<td>87</td>
<td>11.01 (16.49)</td>
<td>9.02 (13.05)</td>
<td>t(86) = .881, p = .381</td>
</tr>
<tr>
<td>ImPACT Reaction time Composite</td>
<td>87</td>
<td>.61 (.08)</td>
<td>.60 (.10)</td>
<td>t(86) = .170, p = .865</td>
</tr>
<tr>
<td>ImPACT X &amp; O: total correct memory</td>
<td>87</td>
<td>8.41 (2.36)</td>
<td>9.95 (11.21)</td>
<td>t(86) = 1.280, p = .204</td>
</tr>
<tr>
<td>ImPACT X &amp; O: avg. correct RT interference</td>
<td>86</td>
<td>.46 (.15)</td>
<td>.49 (.29)</td>
<td>t(85) = -.708, p = .481</td>
</tr>
<tr>
<td>Test Type</td>
<td>Group 1</td>
<td>Group 2</td>
<td>t (df)</td>
<td>p</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>----------</td>
<td>----------</td>
<td>--------------</td>
<td>---------</td>
</tr>
<tr>
<td>ImPACT Design Memory: hits delay</td>
<td>9.88 (2.03)</td>
<td>9.19 (1.90)</td>
<td>t(83)=2.981, p=.004</td>
<td></td>
</tr>
<tr>
<td>ImPACT Symbol match: total correct symbols</td>
<td>28.86 (.35)</td>
<td>26.73 (2.95)</td>
<td>t(86)=.409, p=.683</td>
<td></td>
</tr>
<tr>
<td>ImPACT color match: total correct</td>
<td>8.38 (1.37)</td>
<td>9.14 (9.97)</td>
<td>t(86)=-.710, p=.480</td>
<td></td>
</tr>
<tr>
<td>ImPACT Color match: avg correct RT</td>
<td>.84 (.15)</td>
<td>.82 (.24)</td>
<td>t(86)=.674, p=.502</td>
<td></td>
</tr>
<tr>
<td>ImPACT Color match: total correct commissions</td>
<td>.99 (2.24)</td>
<td>1.10 (3.15)</td>
<td>t(86)=-.276, p=.783</td>
<td></td>
</tr>
<tr>
<td>ImPACT Color match: avg commissions RT</td>
<td>.50 (.69)</td>
<td>.40 (.75)</td>
<td>t(85)=.883, p=.379</td>
<td></td>
</tr>
<tr>
<td>ImPACT Word memory: delayed memory % correct</td>
<td>87.80 (11.98)</td>
<td>84.31 (13.01)</td>
<td>t(86)=2.040, p=.044</td>
<td></td>
</tr>
<tr>
<td>ImPACT Word memory: total % correct</td>
<td>91.53 (8.64)</td>
<td>88.09 (13.20)</td>
<td>t(86)=2.150, p=.034</td>
<td></td>
</tr>
<tr>
<td>ImPACT Design memory: delayed memory % correct</td>
<td>76.77 (16.49)</td>
<td>73.92 (15.02)</td>
<td>t(83)=1.464, p=.147</td>
<td></td>
</tr>
<tr>
<td>ImPACT Design memory: total % correct</td>
<td>78.36 (14.18)</td>
<td>75.68 (15.32)</td>
<td>t(83)=1.562, p=.122</td>
<td></td>
</tr>
<tr>
<td>ImPACT X &amp; O: total correct interference</td>
<td>118.35 (20.44)</td>
<td>117.16 (21.04)</td>
<td>t(86)=.436, p=.664</td>
<td></td>
</tr>
</tbody>
</table>
### Table 5A (H3 Paired $t$ test)

<table>
<thead>
<tr>
<th>H2 Variable (DDM performance)</th>
<th>Pre-concussion (n=7)</th>
<th>Post-concussion (n=7)</th>
<th>Paired Sample $T$-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drift rate ($v$)</td>
<td>.11 (.05)</td>
<td>.13 (.05)</td>
<td>$t(6)=-1.355, p=.224$</td>
</tr>
<tr>
<td>Boundary Separation ($a$)</td>
<td>.30 (.14)</td>
<td>.24 (.06)</td>
<td>$t(6)=1.313, p=.237$</td>
</tr>
<tr>
<td>$Ter$</td>
<td>-.09 (1.05)</td>
<td>.39 (.26)</td>
<td>$t(6)=-1.232, p=.264$</td>
</tr>
</tbody>
</table>

### Table 5B. (H3 ANOVA)

<table>
<thead>
<tr>
<th>DDM Variable</th>
<th>Pre-concussion (n=204)</th>
<th>Post-concussion (n=39)</th>
<th>F-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drift rate ($v$)</td>
<td>.13 (.05)</td>
<td>.12 (.06)</td>
<td>$F(1,241)=.921, p=.338, η^2 =.004$</td>
</tr>
<tr>
<td>Boundary Separation ($a$)</td>
<td>.28 (.12)</td>
<td>.29 (.14)</td>
<td>$F(1,241)=.076, p=.783, η^2 =.000$</td>
</tr>
<tr>
<td>$Ter$</td>
<td>.84 (2.5)</td>
<td>.68 (1.8)</td>
<td>$F(1,241)=1.52, p=.697, η^2 =.001$</td>
</tr>
</tbody>
</table>

Note. Larger absolute values of drift rate ($v$) indicate faster drift. Larger values of boundary separation ($a$) indicate more conservative responding. $Ter$ = non-decision time.
Table 6. Correlations (Baseline neuropsychological variables with drift rate)

<table>
<thead>
<tr>
<th></th>
<th>Drift rate (v)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Drift rate (v)</td>
</tr>
<tr>
<td>2.</td>
<td>WTAR</td>
</tr>
<tr>
<td>3.</td>
<td>BDI - FS</td>
</tr>
<tr>
<td>4.</td>
<td>CARB sum tot corr</td>
</tr>
<tr>
<td>5.</td>
<td>CARB sum % corr</td>
</tr>
<tr>
<td>6.</td>
<td>Vigil Omissions</td>
</tr>
<tr>
<td>7.</td>
<td>Vigil Commissions</td>
</tr>
<tr>
<td>8.</td>
<td>Verbal Vigil Perseverations</td>
</tr>
<tr>
<td>9.</td>
<td>Vigil Hit Rate</td>
</tr>
<tr>
<td>10.</td>
<td>False Alarm Rate</td>
</tr>
<tr>
<td>11.</td>
<td>Vigil Avg Delay</td>
</tr>
<tr>
<td>12.</td>
<td>ImPACT Memory Comp-Verbal</td>
</tr>
<tr>
<td>13.</td>
<td>ImPACT Memory Comp-Visual</td>
</tr>
<tr>
<td>14.</td>
<td>ImPACT Visual motor speed Comp</td>
</tr>
<tr>
<td>15.</td>
<td>ImPACT Impulse control Comp</td>
</tr>
<tr>
<td>16.</td>
<td>ImPACT RT comp</td>
</tr>
<tr>
<td>17.</td>
<td>ImPACT X&amp;O: total corr memory</td>
</tr>
<tr>
<td>18.</td>
<td>ImPACT X&amp;O: avg corr RT interference</td>
</tr>
<tr>
<td>19.</td>
<td>ImPACT X&amp;O: tot corr interference</td>
</tr>
<tr>
<td>20.</td>
<td>ImPACT Design Memory; hits delay</td>
</tr>
<tr>
<td>21.</td>
<td>ImPACT Design memory: tot % corr</td>
</tr>
<tr>
<td>22.</td>
<td>ImPACT color Match: tot corr</td>
</tr>
<tr>
<td>23.</td>
<td>ImPACT Color Match: avg corr RT</td>
</tr>
<tr>
<td>24.</td>
<td>ImPACT Color Match: tot commissions</td>
</tr>
<tr>
<td>25.</td>
<td>ImPACT Color Match: avg commissions RT</td>
</tr>
<tr>
<td>26.</td>
<td>ImPACT Word memory; delayed mem % corr</td>
</tr>
<tr>
<td>27.</td>
<td>ImPACT Word memory: tot % corr</td>
</tr>
<tr>
<td>28.</td>
<td>ImPACT Symbol Match: tot corr sym hidden</td>
</tr>
<tr>
<td>29.</td>
<td>ImPACT Symbol Match: avg corr RT sym</td>
</tr>
<tr>
<td>30.</td>
<td>ImPACT Symbol Match: tot corr sym</td>
</tr>
<tr>
<td>31.</td>
<td>ImPACT Design memory: delayed mem % corr</td>
</tr>
</tbody>
</table>

**p<0.01, *p<0.05
Figure 1. Drift Diffusion Model