

Copyright  
by  
Karon E. Wynne  
2016

**The Capstone Committee for Karon E. Wynne Certifies that this is the approved  
version of the following capstone:**

**A Review of Associations Between Traumatic Brain Injury & Addiction  
and Therapeutic Interventions for Addiction in the Brain  
Injured Population**

**Committee:**

---

Christine Arcari, PhD, MPH,  
Supervisor

---

Gary Seale, PhD,  
Mentor

---

Jacques Baillargeon, PhD

---

Dean, Graduate School

**A Review of Associations Between Traumatic Brain Injury & Addiction  
and Therapeutic Interventions for Addiction in the Brain  
Injured Population**

**by**

**Karon E. Wynne, PhD**

**Capstone**

Presented to the Faculty of the Graduate School of  
The University of Texas Medical Branch  
in Partial Fulfillment  
of the Requirements  
for the Degree of

**Masters of Public Health**

**The University of Texas Medical Branch  
April, 2016**

## **Acknowledgements**

I would like to thank my mentor, Dr. Gary Seale for his support and guidance with my capstone, as well as my practicum experience at TLC. I would also like to thank the rest of my committee, Dr. Christine Arcari and Dr. Jacques Baillargeon for their invaluable expertise and encouragement for this capstone. I would also like to thank my family and friends for all of their support during my time in graduate school.

**A Review of Associations Between Traumatic Brain Injury & Addiction  
and Therapeutic Interventions for Addiction in the Brain  
Injured Population**

Publication No. 3

Karon E. Wynne, PhD, MPH

The University of Texas Medical Branch, 2016

Supervisor: Christine Arcari

The research linking drug and alcohol use with increased risk of sustaining a traumatic brain injury (TBI) has been well established. There is some evidence suggesting that the inverse relationship exists; that sustaining a TBI increases the likelihood for developing a drug or alcohol abuse problem. However much less is known about the impact of TBI on substance use disorders (SUD) and many barriers exist in determining these links. Research also suggests that for individuals with previously existing drug or alcohol problems, sustaining a TBI increases the likelihood of relapse. SUD greatly impact recovery after injury and because this association between TBI and SUD is not well understood there is a need to address how to treat addiction in this population. This work reviews the body of literature about the links between TBI and addiction and strategies for improving SUD treatment in individuals with cognitive impairments. Using the existing body of literature we identified strategies for how SUD treatment can be modified to address the specific needs of individuals with a TBI.

## Table of Contents

<b>List of Tables.....</b>	<b>viii</b>
<b>List of Abbreviations.....</b>	<b>ix</b>
<b>Chapter 1 Introduction.....</b>	<b>1</b>
<b>Substance Use Definition and Statistics.....</b>	<b>1</b>
<b>Traumatic Brain Injury: Definition and Statistics.....</b>	<b>3</b>
<b>Chapter 2 Methods.....</b>	<b>6</b>
<b>Chapter 3 Results.....</b>	<b>8</b>
<b>Relationship Between Substance Abuse and Traumatic Brain Injury.....</b>	<b>8</b>
<b>Substance Abuse Disorders Affect Recovery After TBI.....</b>	<b>9</b>
<b>Relationship Between Traumatic Brain Injury and Substance Use.....</b>	<b>10</b>
<b>Biochemical and Structural Evidence For Traumatic Brain Injury     Increasing the Risk of Substance Use Disorder.....</b>	<b>12</b>
<b>Challenges in Studying Traumatic Brain Injury’s effects on Substance Use     Disorder.....</b>	<b>12</b>
<b>Treating SUD in the TBI Population.....</b>	<b>13</b>
<b>Reducing Treatment Barriers.....</b>	<b>14</b>
<b>Public Health Interventions Prior to Injury.....</b>	<b>14</b>
<b>Treatment Strategies for Substance Use Disorders.....</b>	<b>15</b>
<b>Addressing Patient Retention in Treatment Programs.....</b>	<b>15</b>
<b>Chapter 4 Discussion.....</b>	<b>18</b>
<b>Approaches for Improving Program Effectiveness.....</b>	<b>18</b>
<b>Peer Mentorship Programs.....</b>	<b>18</b>
<b>Community Based Treatment Programs.....</b>	<b>19</b>

<b>Chapter 4 Discussion (cont.)</b> .....	<b>19</b>
<b>Adapting to Accommodate Patient Comorbidities</b> .....	<b>19</b>
<b>Motivational Interviewing</b> .....	<b>20</b>
<b>Cognitive-behavioral Therapy</b> .....	<b>20</b>
<b>Best Practices for SUD Treatment in the Brain Injured Population</b> .....	<b>21</b>
<b>Lessons From Other Patient Populations in Substance Abuse Treatment Programs</b> .....	<b>22</b>
<b>Anxiety Therapy in TBI Patients</b> .....	<b>22</b>
<b>The Association between Posttraumatic Stress Disorder, TBI and SUD</b> ...	<b>24</b>
<b>Conclusion</b> .....	<b>26</b>
<b>Literature Cited</b> .....	<b>28</b>
<b>Vita</b> .....	<b>32</b>

## List of Tables

Table 1.....	5
Table 2.....	25

## **List of Abbreviations**

<b>BAL</b>	<b>Blood Alcohol Level</b>
<b>CBT</b>	<b>Cognitive Behavioral Therapy</b>
<b>DMS-V</b>	<b>Diagnostic and Statistical Manual of Mental Disorders</b>
<b>MAST-AD</b>	<b>Modified Michigan Alcohol-Drug Screening Test</b>
<b>MN-SAPS</b>	<b>Substance Abuse Problem Scale</b>
<b>PHRED</b>	<b>Public Health and Education Development</b>
<b>PTSD</b>	<b>Post Traumatic Stress Disorder</b>
<b>SUD</b>	<b>Substance Use Disorder</b>
<b>TBI</b>	<b>Traumatic Brain Injury</b>

## **Chapter 1: Introduction**

There is a well-established understanding that the use and abuse of drugs and alcohol increases the likelihood that an individual will sustain a traumatic brain injury (TBI) (Corrigan 2008, Graham 1995). Additionally, a small body of literature suggests the converse relationship exists; that sustaining a TBI increases the likelihood of drug use and abuse (Graham 2008). However, this relationship is not as well understood. Many confounding factors make studying this association more difficult. Through an extensive review of the existing literature this paper will explore the relationships between TBI and substance abuse as well as how TBI effects drug use and abuse. Additionally the literature on existing treatment of substance abuse in brain-injured patients will be discussed. Finally, we will examine adaptive substance abuse treatment options that have been used in other populations with cognitive impairments to provide insight on how the treatment for TBI patients might be adapted.

### **Substance Use Definition and Statistics**

We will first begin by reviewing the definitions and statistics on substance use and abuse and TBI. Substance use disorders (SUD) are classified as abuse or dependence on alcohol and/or drugs that is considerably higher than that of the general population (West 2011). More specifically, for an individual to abuse a substance they must experience one of the follow four domains. These domains are “recurrent failure to meet responsibilities due to use, recurrent use when physically dangerous, recurrent legal problems related to use or continued use despite such problems” (West 2011). One of these domains must have occurred in the last year in the absence of dependence (West 2011). Another important

definition comes from the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders 5<sup>th</sup> edition (DSM-V). SUD in the DSM-V is defined as a pattern of behavior of impaired control, social impairment, risky use and pharmacological effects due to use SUDs are also characterized by continued despite adverse consequences (American Psychiatric Association 2013). For the purpose of this paper SUD will apply to the abuse of both drugs and alcohol. Substance dependence is defined as experiencing three of the following symptoms within a year period, "tolerance, withdrawal, use in durations longer than intended, a desire or inability to cut down, extended time obtaining the substance or using it, using in lieu of other important activities, or continued use despite knowing that such use causes or exacerbates problems" (West 2011).

The DSM-V reports that the lifetime prevalence of SUD is 14.6% (Kessler 2005). This use has enormous public health implications, with drug and alcohol addiction causing millions of deaths and accounting for billions in associated costs annually (Birnbaum 2011, Shield 2014). In 2012, 23.1 million Americans were dependent on drugs or alcohol and were in need of treatment for substance abuse (National Institute for Drug Abuse 2014). Alcohol abuse is one of the most common psychiatric disorders and substance abuse disorders are one of the most common classes of psychiatric disorders (Kessler 2005). In 1990, the lifetime incidence rate for alcohol dependence was 13.5% and the incidence of drug dependence was 6.1% (Regier 1990), these numbers have risen in the last 2 decades with SUD now over 14% (Kessler 2005).

The average onset for substance abuse disorders is 20 and they are most common in individuals between 18 and 27 (Kessler 2005). However SUDs can occur in people of

all ages. SUD are also more common in men than in women (Kessler 2005). Substance abuse is less common in non-Hispanics whites (Kessler 2005). Those who are married are less like to experience SUDs than those who were previously married or never married (Kessler 2005). Over 3 million people globally died in alcohol related deaths, which accounts for 5.9% of the total global deaths for the year (World Health Organization 2014).

### **Traumatic Brain Injury Definition and Statistics**

TBI is damage to the brain that results in “an alteration in brain function, or other evidence of brain pathology, caused by an external force (Menon 2010).” The committee on traumatic brain injury defines TBI as “a brain injury caused by a blow or jolt to the head or a penetrating injury that disrupts the function of the brain (Eden 2006).” It is one of the most common, disabling, neurological conditions worldwide (Zasler 2013). Falls, auto accidents and violent physical attacks are the most common accidents that result in TBI (West 2011). Gun violence is also a significant contributor to brain injury. Of these falls is the single greatest contributor at 28% of injuries (Eden 2006). Annually, 2.5 million brain injuries occur in the United States, which amounts to approximately 7,000 per day (Center for Disease Control 2014). 230,000 of TBI lead to hospitalization (Thurman 1999) and 50,000 deaths occur (Cecil 2010, Ling 2010). TBI is a leading cause of morbidity and mortality in people under 45 worldwide (Werner 2007). TBI, like SUDs, is a major public health concern with medical costs from injuries totaling over 60 billion dollars (Kurland 2012).

TBI is considered an under diagnosed condition in the US and worldwide (Eden 2006). Individuals with mild TBI might not be aware of their condition or seek medical

attention (Eden 2006). It is estimated that at least 5.3 million people are suffering from a disability due to a TBI in the US alone (Eden 2006). Traumatic brain injury is more prevalent in children, adolescents and elderly populations (Eden 2006). Children under 5 are most susceptible with 1,120.7 hospitalizations, ER visits and deaths due to TBIs per 100,000 (Eden 2006). For adolescents (ages 15-19) the rate is 814.4 per 100,000. For people over 75 there are 659.1 brain injuries per 100,000 (Eden 2006). The rates for children between 5 and 14 are also over 600 per 100,000. Table 1 illustrates the rates of sustaining a TBI across all age groups (Eden 2006). Males are more likely to sustain a TBI than females. American Indians, Alaskan natives and military personal are all groups at higher risk for sustaining a TBI as well (Eden 2006).

<b><u>Age Group</u></b>	<b><u>Rate per 100,000</u></b>
0-4	1120.7
5-9	659.3
10-14	628.6
15-19	814.4
20-24	555.5
25-34	450.2
35-44	374.7
45-54	285.2
55-64	229.5
65-74	267.4
Over 74	659.1
All Ages	506.4

Table 1: Rates of TBI-related hospitalizations, emergency room visits and deaths by age in the U.S. between 1995 and 2001 (Eden 2006)

TBI, substance use and SUD are significant public health problems facing the United States. SUD affected 23.1 million Americans in 2014 (National Institute for Drug Abuse 2014), while that same year, 2.5 million brain injuries occur in the United States (Center for Disease Control 2014). Through our research we will examine the associations between SUD and TBI in order to understand how each condition can further exacerbate its comorbidity. We will also identify unique SUD treatment approaches for TBI patients by examining other SUD programs for patients with cognitive deficits.

## **Chapter 2: Methods**

A systematic literature review to examine the links between TBI and addiction was conducted. The primary databases utilized were Medline (Ovid) and Medline (PubMed). Search terms in each database included ‘brain injury’ in combination with the terms ‘addiction’, ‘alcoholism’, ‘opioid related disorders’ and ‘cocaine related disorders’ using the Boolean operator ‘AND’. This search resulted in 453 papers. Paper titles and abstracts were reviewed to identify the papers demonstrating applicable associations between drug abuse and TBI. Only papers that examined associations between the two factors were further reviewed for this section.

To research current psychological and pharmacological therapies a second search was conducted uniting the terms ‘intervention’, ‘treatment’, ‘therapy’ with the Boolean operator ‘OR’, in combination with the terms ‘addiction’, ‘alcoholism’, ‘opioid related disorders’ and ‘cocaine related disorders’ with the Boolean operator ‘OR’. Paper titles and abstracts were reviewed to narrow papers applicable to SUD treatment in patients with cognitive deficits.

Research to be excluded from our systematic literature review included work that was not yet published in a peer-reviewed journal, work not published in English and work published before 1985. Research was also limited to papers on Human brain injury and substance abuse disorders. The quality of sources was assessed using the Public Health, Research and Education Development (PHRED) program (Comper 2005). The PHRED program evaluates a paper by examining selection bias, study design, confounders, blinding, data collection, withdrawals, dropouts, intervention integrity and analyses

(Comper 2005). Each section was classified as strong, moderate or weak and then an overall score given to the paper (Comper 2005). Upon reviewing titles and abstracts, as mentioned above, papers were given a PHRED score. Only strong and moderate papers were further reviewed for inclusion in this work.

## **Chapter 3: Results**

### **Relationship Between Substance Abuse and Traumatic Brain Injury**

There is a well-established link that being under the influence of drugs or alcohol increases the risk of sustaining a TBI (Bjork 2009, Corrigan 2013). Studies on this association have been conducted for several decades and research has reported that the percent of individuals who were intoxicated at the time of injury as high as 51% (Kreutzer 1996, Parry-Jones 2006, West 2011). Individuals who consume alcohol are four times more likely to sustain a TBI than those who do not drink at all (Olson-Madden 2012). TBI patients have higher rates of alcohol and drug abuse than the general population. Studies have reported alcohol abuse rates between 44% and 79% and drug abuse rates between 21% and 40% (West 2011). Substance use prior to injury in certain demographics, such as young males, rates were reported as high as 87%. Intoxication was defined as having a blood alcohol level (BAL) above 100 mg/dL (Parry-Jones 2006). Another study found that 78% percent of TBI patients had some alcohol in their system, with 58% of those having a BAL classified as intoxicated (Kreutzer 1996). Additionally, studies report SUD for TBI patients prior to injury ranging from one third to as high two thirds of patients (Walker 2003, West 2011). The lifetime occurrence of TBI for alcoholics is 41% in men and 22% in women (Felde 2006). In the general population unaffected by alcoholism, these rates are 15% for men and 6% for women (Felde 2006). Studies in military personal have found that substance misuse increases the risk of sustaining a mild TBI (Corrigan 2013).

## **Substance Abuse Disorders Affect Recovery After TBI**

Substance use has a significant impact on both injury severity and recovery. Immediately after injury, TBI patients who are under the influence of drugs or alcohol typically have more severe injuries as indicated by coma ratings and neuropsychological measures (Walker 2003, West 2011). Drug use causes permanent changes to cerebral blood flow that can alone cause neural damage, and compounds the effects of TBI (Walker 2003). It has been found that these patients have more brain tissue atrophy than those not under the influence, which is important to cognitive outcome (West 2011). Alcohol and drug use has also been found to significantly increase the length of hospital stay (West 2011). In addition to injury severity this is in part due to the increased risk of sepsis, pneumonia, and organ failure (West 2011). Another study of veterans found that those with a history of SUD have higher rates of hospitalization after TBI when those without SUD (Corrigan 1995, Parry-Jones 2006). Comorbid occurrences of TBI and SUD are associated with poorer outcomes in both cases where the alcohol use is pre-injury or post-injury (Corrigan 1995, Olson-Madden 2012, Parry-Jones 2006).

Substance abuse slows recovery from injury and increases the chance of continuing to abuse drugs or alcohol after injury (Bjork 2009). SUD in combination with TBI is associated with a greater risk of reinjury, psychosocial dysfunction and psychiatric disorders (Olson-Madden 2012). A study by Felde, et al., showed that TBI patients with the comorbid occurrence of SUD had higher rates of depressive symptoms and were more likely to attempt suicide (Felde 2006). TBI patients that use drugs and alcohol are less likely to participate in rehabilitation after they are released from the hospital (West 2011).

Substance abuse in TBI patients is associated with increased risk of death (Fazel 2014).

It is difficult to detect substance use in TBI patients because of the overlap of symptoms and impact on cognitive function immediately after injury (West 2011). Long term, TBI and substance use have similar effects on cognitive function, mood and disinhibition, as well (West 2011). Little work has been done on how to differentiate symptoms due to TBI versus symptoms due to substance use (West 2011). Some methods have been developed to diagnosis substance use in patients with TBI (West 2011). Several scales have been evaluated with little success in the TBI population (West 2011). Tested scales include the Brief Michigan Alcoholism Screening Test (BMAST), the Substance Abuse Subtle Screening Inventory (SASSI), CAGE, and structured psychiatric interviews; few have been evaluated for their effectiveness with this population (West 2011).

### **Relationship Between Traumatic Brain Injury and Substance Abuse**

Some research has suggested that sustaining a brain injury increases the likelihood of developing addiction or relapsing compared to the general population but this relationship is not as well understood or established (Bjork 2009, Fazel 2014, West 2011). Little information is available about whether an injury increases the risk for substance abuse in those with no previous history (Bjork 2012). Research indicates that drug and alcohol use typically decreases immediately after injury (Corrigan 1998, West 2011). This is likely due to hospitalization or physical barriers that restrict access to alcohol or drugs (Corrigan 1998, West 2011). Drug and alcohol use begins to increase over time as the patient recovers and physical barriers are eliminated or decreased (Corrigan 1998). One year after injury, as many as 50% of TBI patients participate in substance abuse (West

2011). A study in prison inmates who needed treatment for substance abuse, 68% of them reported having sustained a TBI (Walker 2001). Another large study (n=7,000) of patients being treated for SUD found that 31% of individuals had a head injury that resulted in loss of consciousness (Walker 2007).

Research indicates that brain injuries increase an individuals' propensity for drug and alcohol abuse (Kreutzer 1996, Walker 2003). In a study that compared individuals with no brain injuries to those with one or more they found that the TBI group had higher rates of both alcohol and marijuana use (Walker 2003). Marijuana has been reported as the most commonly abused drug by TBI patients (Taylor 2003). In a study of military personnel, they found that the danger of alcohol dependence was increased for as long as 6 months after sustaining a mild TBI (Staff 2013). It has also been demonstrated that those who suffered a mild TBI as a child were significant more likely to show some signs of SUD as adolescents (Corrigan 2013).

Several studies found that alcohol use increased post-injury (Bogner 2001, Kreutzer 1996). In one survey, more than half of individuals who were classified as moderate to heavy drinkers after injury returned to pre-injury drinking habits within one year after their accident. (Kreutzer 1996). Other studies also report that drinking increases over time after injury and typically returns to pre-injury drinking patterns (Sparadeo 1989). Some work suggests that after TBI the severity of substance abuse increases. One study in an adult population used the clinical rated substance abuse problem scale (MN-SAPS) and self-rated modified Michigan alcohol-drug screening (MAST-AD). They found an increase in the severity of substance abuse (Felde 2006).

## **Biochemical and Structural Evidence For Traumatic Brain Injury Increasing the Risk of Substance Abuse Disorder**

There are some structural and chemical changes in the brain that occur after TBI that account for increased risk for substance abuse (Bjork 2009). One such change is damage to the orbitofrontal cortex that can result in an organic personality disorder which is associated with a higher risk of substance abuse (Bjork 2009). Frontal lobe dysfunction has been linked to increased risk of SUD (Walker 2003). Damage to this area of the brain is also associated with apathy, impulse control issues, depression and reduced problem solving abilities (Walker 2003). All of these are characteristics that are associated with an increased risk for drug and alcohol use (Walker 2003). While the direct link of brain injuries effect on SUD is not clear, evidence suggests that TBI results in damage to the brain that increase risk factors for SUD.

## **Challenges in Studying Traumatic Brain Injury's effects on Substance Abuse Disorder**

It has been noted that there is a deficit in our understanding of post injury substance use. There are few studies that report drug and alcohol use in TBI patients in the first two years after injury and even fewer studies examine use in TBI patients after 3 years (Kreutzer 1996, Ponsford 2007). The studies that do exist often only examine small, localized populations (Kreutzer 1996). It is difficult to determine if drug and alcohol risk increases because there is such a high proportion of the TBI population that had histories of SUD prior to injury (Graham 2008).

Another difficulty in determining if TBI increases the likelihood for SUD is the way TBI is classified. For many patients, the physician determines the severity of the

injury, and sometimes the injury itself subjectively (Bjork 2009). Also substance abuse rates prior to illness are so high that it can be hard for the physician to determine what aspects of the neurological symptoms are due to the injury and which are due to the SUD (Bjork 2009). Substance abuse and TBI present clinically in very similar ways. They both affect cognitive function, motor function and disinhibition (West 2011).

Very little work has been published about how to address addiction problems in the TBI populations that have co-occurring physical and cognitive deficits that might impair the effectiveness of traditional drug treatment. However, treatment options developed for other cognitively impaired groups may provide guidance for developing better therapies for TBI patients who also suffer from SUD.

### **Treating SUD in the TBI Population**

Treating substance abuse disorders has important implications for reducing the risk of secondary injury, decreasing mortality and reducing health care cost for patients with SUD (West 2009, West 2011). It also helps to improve the patients overall health and quality of life (Walker 2003). Successful treatment for SUD also has a significant impact on crime rates and costs related to crime (West 2009, West 2011). However, barriers exist for TBI patients to obtain treatment for SUD (West 2009, West 2011). The number of TBI patients in SUD treatment is relatively low compared to the proportion of individuals with TBI that are suffering from SUD (West 2009, West 2011). Personal physical barriers, as well as barriers in program accessibility are the most common causes for TBI patients not receiving treatment for their SUD (West 2009, West 2011). Service denials to SUD

treatment programs for individuals with TBI that try to gain admittance typically range from 42% to 68% of requests (West 2009, West 2011).

### **Reducing Treatment Barriers**

The first step to improving the treatment of SUD in TBI patients is actually getting them to treatment in the first place. Physical barriers are one consideration that prevents those with physical impairments resulting from their brain injury from participating in programs (West 2011). Patients are sometimes denied access to a program when they apply because of physical and institutional barriers, rather than working around those barriers (West 2011). Treatment providers should keep this in mind when planning programs and take steps to reduce physical barriers whenever possible. Speaking to patients about perceived barriers might also be helpful.

### **Public Health Interventions Prior to Injury**

Due to the link between substance use and the risk of sustaining a TBI (Bjork 2009, Corrigan 2013), it is important to address substance use in all individuals before they sustain a brain injury. Additionally, because TBI can increase the risk of developing a SUD it is advantageous to work to prevent TBI. Currently there are no pharmacological treatments for TBI (Agoston 2012). The primary tool for treating TBI is primary prevention (Binder 2005). Educating people in order to prevent injury is the best method available (Binder 2005). Wearing helmets while riding a bike or motorcycle is one example of a preventative measure for TBI. Educating patients about other behaviors that can lead to

TBIs, such as violence, falls and car accidents, and what they can do to mitigate the risk of sustaining an injury is key in the defense against TBI (Binder 2005).

### **Treatment Strategies for Substance Use Disorders**

Many treatment strategies have been proposed for the addressing substance use disorders in brain-injured individuals. However, little work has been done to address the efficacy of specific treatments for TBI patients. Of the work that has been done, evidence suggests that traditional treatment strategies may be less effective in TBI patients. The research suggest that substance use treatment should be adapted for this unique population that often demonstrates impairments in multiple domains, including cognitive, emotional and behavioral deficits following injury (Graham 2008). Using community based, multifaceted treatment options may also be beneficial (Graham 2008).

### **Addressing Patient Retention in Treatment Programs**

One aspect of treating SUD in the brain-injured population is addressing patient retention, which is a major barrier for successful treatment. TBI patients have significantly higher dropout rates compared to patients that do not have a brain injury (Graham 2008). These patients are also more likely to miss appointments (Graham 2008). Several studies have looked at addressing this issue for TBI patients in SUD programs (Graham 2008). A study by Corrigan et al., compared several methods to help improve retention in TBI populations (Corrigan 2007). Patients were either provided with a financial incentive, or barrier reduction and these methods were compared to a control group (Corrigan 2007). Financial incentive was a gift card given for perfect attendance during the first month

(Corrigan 2007). Barrier reduction including providing patients with extra attention and help to promote their attendance (Corrigan 2007). Techniques such as calling patients to remind them of appointments, helping with transportation or childcare were used in this group (Corrigan 2007). For the attention control group patients were only given the time and location of the next session (Corrigan 2007). There was no follow up or incentives with this group (Corrigan 2007). It was determined that the financial incentive was most effective to increase patient attendance at sessions during the first month (Corrigan 2007). However, only 50 percent of patients in this group and in the barrier reduction finished the entire treatment program (Corrigan 2007). These groups were more likely to finish treatment than those in the attention control group, who were 3 times more likely to not complete the entire duration of treatment (Corrigan 2007). While patients receiving financial incentives early in treatment still dropped out of treatment at a high rate, they were significantly more likely to complete the program than those that were not provided an incentive (Corrigan 2007). Working to reduce treatment barriers also provided an increase in successful completion of a treatment program (Corrigan 2007).

In a separate study by Corrigan, financial incentive was again compared to barrier reduction and a control condition (Corrigan, 2005). However, they also compared these methods to motivational interviewing (Corrigan, 2005). The percentage of patients that signed treatment plans within 30 days was the outcome measure (Corrigan, 2005). Financial incentive was again found to be most effective, with 83% of patients signing treatment plans (Corrigan, 2005). Barrier reduction was the second most effective with 74% of patients signing treatment plans (Corrigan, 2005). Financial incentive and barrier reduction were also the most effective in improving patient attendance (Corrigan, 2005). For those

provided a financial incentive only 40% missed visits (Corrigan, 2005). In the barrier reduction group 42% missed one or more visits (Corrigan, 2005). Motivational interviewing improved patient attendance as well when compared to the control group (Corrigan, 2005). 57% of those in the motivational interviewing group missed an appointment versus 64% on the control group (Corrigan, 2005).

An additional approach to improving treatment program retention involved providing TBI patients in SUD treatment with a case manager (Heinemann 2004). In outpatient treatment programs, case managers are an important aspect of a successful treatment program (Sparadeo, 1992). They have commonly been employed in substance abuse treatment programs (Heinemann 2004). Literature suggests that they improve program retention (Heinemann, 2004). A study by Heinemann et al. that examined the use of case management in SUD treatment for individuals with TBI in two different case management SUD programs (Heinemann, 2004). One program using case management found 59% percent retention and in the other case management program 43% remained in the program (Heinemann 2004). This compared unfavorably to the control program that had 79% retention (Heinemann 2004). The study sited differences in demographics between the groups as a possible cause for these results (Heinemann, 2004). Other programs have demonstrated more success with case management (Corrigan, 1995; Ford, 1995). More research is needed to determine the effectiveness of the case management approach (Heinemann, 2004).

## **Chapter 4: Discussion**

### **Approaches for Improving Program Effectiveness**

Now that we have reviewed the existing body of literature examining associations between TBI and SUD, we will build upon this knowledge to determine literature-based recommendations for the treatment of SUD in patients with TBI. These recommendations incorporate suggestions from the small body of treatment literature previously examined. We also use research from other groups demonstrating cognitive impairments to identify ways to improve SUD treatment for TBI patients.

### **Peer Mentorship Programs**

Program retention is not the only aspect of SUD treatment that should be addressed to improve the quality of SUD treatment for TBI patients. A small body of research has examined how to individualize treatment for brain-injured patients. One approach involved pairing patients with a history of TBI with individuals who recently sustained a brain injury. The mentor TBI patient was to share resource information, provide emotional support and advocacy skills for the newly injured individual. Participants were then surveyed to determine their sense of empowerment, degree of knowledge, degree of family support, quality of life, and coping mechanisms for sadness, anger and anxiety. In many of the above-referenced categories, patients reported benefits from participation in the program. 82% reported an improved sense of empowerment, 53% reported improved coping with sadness, 82% reported improved knowledge of TBI and 54% reported improved quality of life. In other categories the program was less successful. Only 18% of

participants reported an improvement in family support and 36% reported improved coping with anxiety (Hibbard 2002).

### **Community Based Treatment Programs**

Another treatment approach that was evaluated for addressing substance use in TBI patients focused on community inclusion. Hensold, et al., illustrates an example of this approach. This study combined group and individual counseling sessions. The purpose of these sessions was to educate patients and promote self-monitoring. Patients were then rewarded for successful execution of self-monitoring of drug and alcohol use. The outcomes measured were job status, housing status, awareness, independence and productive activities. Improvements were seen in all areas except job status. Independence was the area of greatest improvement for participants. A major weakness of this study failure to measure rates of drug and alcohol consumption before and after treatment (Hensold, 1997).

### **Adapting to Accommodate Patient Comorbidities**

Another interesting study utilizing a community-based approach to treating substance use compared the difference between TBI patients and patients without TBIs. The study found that TBI patients had higher comorbidity rates for certain disorders such as depression, schizophrenia, anxiety and psychosis. In this study 25% of the patients also suffered from psychological disorders, while none of the patients without a TBI had a personality disorder. Comorbidities increased with the number of past TBIs sustained. The younger the age of patient when the TBI occurred, the greater the risk of sustaining a TBI.

These comorbidities required extra intervention of pharmacological treatments in order to improve addiction treatment. We will further discuss comorbidities later in this paper (Corrigan, 2008).

### **Motivational Interviewing**

Motivational interviewing and motivational counseling are techniques that have been shown to promote abstinence after TBI (Olson-Madden, 2012). In motivational interviewing the patient is counseled to understand the risks and consequence of continued substance use, with the ultimate goal of discouraging further use (Miller, 1991). Motivational interviewing has been shown to be a good short-term intervention for alcohol abuse, but should be combined with other long-term techniques for sustained abstinence (Morgenstern, 2007). Even a single session of motivational interviewing has been shown to be beneficial (Miller, 1991). Studies have shown a decrease in drug and alcohol consumption, as well as improving a desire to change behavior with a single session of motivational interviewing (Miller, 1991). In general, motivational interviewing is most effective when used in combination with other tools (such as cognitive-behavior therapy, pharmacological interventions, etc.), early in treatment (Bombardier, 1999; Hettema, 2005). Some of these additional tools will be discussed below.

### **Cognitive-behavioral Therapy**

One approach that can be used in combination with motivational interviewing is cognitive behavioral therapy. Cognitive-behavioral therapy (CBT) focuses on changing thought patterns in order to change behavior. This approach is centered on individual goals,

so it is specifically tailored to individual needs. This is ideal for the unique problems each TBI patient faces (Corrigan, 2013). However it is also important that a patient has adequate cognitive functioning to participate in and fully benefit from CBT (Aharonovich 2003). Therefore, proper evaluation of a patient's cognitive function before beginning cognitive-behavioral therapy is important to the success of this therapy approach. Combining medication with cognitive behavioral therapy is another strategy to improve the effectiveness of this type of treatment. It has been shown that medications can help improve the effects of therapy compared to therapy alone for substance use (Corrigan, 2013). Naltrexone, Acamprostate and Disulfiram have been approved by the FDA as being affect for use in treating SUD (Corrigan 2013).

### **Best Practices for SUD Treatment in the Brain Injured Population**

Of the small body of literature that does exist some conclusions have been drawn regarding how to improve the treatment of substance use for the brain injured population. Due to the cost and availability, community based programs are usually most accessible and affordable. Motivational interviewing can be an effective intervention when combined with other treatment options, and skill-based interventions appear to be more successful than motivational interviewing. Additionally, financial incentives have proven successful in improving program attendance and retention, and reducing barriers for patients (i.e., providing transportation, child care, etc.) is also helpful, but to a lesser extension than incentives. The use of peer support and mentoring has been shown to be helpful in aiding patients recover from SUD. Finally, group and individual counseling can both be important components of successful therapy, even in an outpatient setting (Graham 2008). Using

these best practices in various combinations would allow for treatment providers to tailor a program to meet individual needs. For example, providing outpatient group and individual therapy and adding a mentoring aspect between the participants could be easily implemented. Adding a financial incentive to promote patient attendance to at these sessions, as well as interventions to reduce barriers, could also be incorporated.

### **Lessons From Other Patient Populations in Substance Abuse Treatment Programs**

In addition to using the knowledge gained from the small body of literature for treatment of SUD in TBI patients, it might be useful to examine how other psychological therapies have been adapted for TBI patients and borrow best practices from these strategies. General recommendations for cognitive behavioral therapy for treating brain-injured patients include using various forms of reinforcement during therapy sessions. Verbal reinforcements in therapy sessions improved behavior change (Coetzer, 2014). In TBI patients, it was also observed that monetary reinforcement improved attendance. Simplicity, consistency, and repetition are also recommended adaptations for cognitive behavioral therapy (CBT) for TBI patients.

### **Anxiety Therapy in TBI Patients**

Some recommendations have been made for improving anxiety therapy for TBI patients that may also be applied to treatment of SUD in the TBI population. Again, increasing repetition of information or increased practice of strategies help compensate for cognitive deficits. Increasing the duration of treatment, along with increased follow up after treatment ends may improve response to treatment and maintenance of outcomes. It is also

suggested that shorter but more frequent therapy sessions might improve the quality of treatment (Hsieh, 2012). Employing these strategies for SUD therapy might be worthwhile, but it is important to keep in mind that program retention is a particular problem in treatment with this population. Examining retention in any study that increase the length and frequency of treatment would be an important variable. In anxiety therapy for TBI patients, limiting the scope of treatment and focusing on a few key skills is also recommended (Hsieh, 2012). Providers should give thought to limiting the number of skills and the information provided in each session. Changing the pace and dissemination of information may benefit knowledge retention and prevent patients from becoming overwhelmed. Research also indicates that early intervention was beneficial for the TBI population (Bryant, 2003). Due to the high rates of substance use prior to injury, it would be relatively easy to identify individuals who are at high risk for substance abuse following TBI. Preventative therapy could be implemented in these individuals, as well as others with risk factors for developing SUD. Because TBI may increase the risk of developing a SUD it might be beneficial to provide all patients with knowledge of available resources, as well as coping mechanisms to prevent stress and depression that might lead to substance use.

### **The Association between Posttraumatic Stress Disorder, TBI and SUD**

Another consideration when treating SUD in TBI patients is that patients with SUD often suffer from accompanying posttraumatic stress disorder (PTSD). PTSD is a common anxiety disorder in people with SUD. TBI and PTSD also often coexist, especially among veterans (Brady, 2009). Table 2 illustrates the rates of TBI and PTSD comorbidities in a veteran population. Almost 20% suffer from both TBI and PTSD. Over half are currently

consuming alcohol, though the study did not look at the rates of alcohol abuse. Of note is that some of the veterans surveyed not only suffered from TBI and PTSD, but were also suffering from panic or depressive disorders. Rates of depressive disorders were very high in the veterans surveyed. Over 20% of the populations with PTSD and TBI had a panic disorder as well (Graham, 2008). It seems that patients should not only be tested for PTSD, but also screened for depression and panic disorders as well. Identifying and treating a patient's PTSD might help to improve their SUD treatment. Some treatment programs address both PTSD and SUD individually, while other programs treat these co-occurring conditions concurrently. Substance dependence posttraumatic stress disorder therapy and concurrent treatment with prolonged exposure are two examples of methods that attempt to address both PTSD and SUD. More research is needed to determine which options are the most effective combinations (Brady, 2009).

<b>Variable</b>	<b>+PTSD,+TBI</b>	<b>+PTSD, -TBI</b>	<b>-PTSD, +TBI</b>	<b>-PTSD, -TBI</b>
<b>% of Sample</b>	19.2%	16.4%	10.8%	53.6%
<b>Mean Age</b>	27.4	30.2	29.7	30.4
<b>% Male</b>	95.1	88.6%	82.6%	83.3%
<b>% White</b>	58.5%	54.3%	56.5%	46.5%
<b>% Married</b>	36.6%	48.6%	52.2%	40.4%
<b>% With at least a high school education</b>	39.0%	40.0%	30.4%	31.6%
<b>Past alcohol use</b>	65.9%	62.9%	73.9%	68.4%
<b>Current alcohol use</b>	53.7%	45.7%	52.2%	60.5%
<b>Past Cannabis Use</b>	17.1%	28.6%	21.7%	18.4%
<b>Current Cannabis Use</b>	2.4%	5.7%	4.3%	5.3%
<b>Past Cocaine Use</b>	2.4%	11.4%	0.0%	5.3%
<b>Current Cocaine Use</b>	2.4%	0.0%	0.0%	0.9%
<b>% Depressive Disorder</b>	75.6%	62.9%	43.5%	36.3%
<b>% Panic Disorder</b>	22.0%	11.4%	8.7%	4.4%

Table 2: Shows data on mental disorders and drug use, as well as other demographic information from 213 Iraq and Afghanistan veterans (Graham 2008)

## **Conclusions**

Diagnosing and treating SUD in the brain-injured population remains a significant challenge to health care providers. It seems the first step to improving the care for these patients is to improve diagnostic tools for detecting and discerning those effects due to injury versus those due to drug and alcohol use or abuse. Proper diagnosis can aid in providing proper treatment to patients. For example, if symptoms are attributed to use of drugs or alcohol that are in fact due brain injury, then cognitive issues might not be addressed that potentially impact the effectiveness of a substance abuse treatment program.

In conjunction with better diagnostic tools is the need for more research on how TBI impacts drug and alcohol use and abuse after injury. Anxiety and CBT research suggest that increased repetition is helpful to TBI patients. These patients can also benefit from shorter duration, but more frequent sessions, focusing on core skills development so as not to overwhelm the patient. Research has indicated that the damage that occurs after TBI results in alteration to the brain that increase risk factors for TBI such as impulsivity and depression, among others.

Another area that needs more attention is how to best treat SUD in TBI patients. Treating SUD in the TBI population is a complicated issue. Research has indicated the need for improve access to programs, as well as improved program retention. These are both major problems that hinder proper SUD treatment for TBI patients. The research that has been conducted has made several conclusions about how to best treat TBI patient's substance abuse problems. Community based programs have been found to be the most practical for patients, and have shown success in treating SUD. Motivational interviewing

is also recommended for these patients. Research has indicated that motivational interviewing is more of a short-term therapy that should be used in conjunction with other treatments.

While there is certainly a need for more research, there has been work in treating other psychiatric disorders in TBI patients that might provide lessons that can be borrowed for TBI patients. There are also comorbid conditions like PTSD, depression and panic disorders that may exist in TBI patients that hinder their success in SUD program. Research indicates that addressing these other conditions leads to improved SUD treatment.

The key take away from our review is that more research is needed. TBI and SUD are each individually billion-dollar public health issues (Birnbaum 2011, Kurland 2012, Shield 2014) that affect millions of people (Eden 2006, National Institute For Drug Abuse 2014). This clearly is an important, but relatively unexplored area. We advocate for more studies on the associations between TBI and SUD, as well as more in-depth studies on the techniques that have proven effective for SUD treatment in TBI patients. Larger more robust studies are needed in order to develop a standard of care for this population. Additionally we advocate for new research on SUD treatment that builds upon work done for other psychiatric conditions in TBI patients. Drawing from work done in treating SUD in individuals with other cognitive disorders might prove a good starting point for expanding research.

## Literature Cited

Agoston, D.V., Rislind, M., Bellander, B. (2012). Bench-to-bedside and bedside to the bench; coordinating clinical and experimental traumatic brain injury studies. *Frontiers in Neurology*, 3(3), 1-5.

American Psychiatric Association (2013). Diagnostic and statistical manual of mental disorders (5<sup>th</sup> Ed.). Arlington, VA: American Psychiatric Publishing.

Binder, S., Corrigan, J.D., Langlois, J.A. (2005). The public health approach to traumatic brain injury: an overview of CDC's research and programs. *The Journal of head trauma rehabilitation*. 20(3): 189-195.

Birnbaum, H.G., White, A.G., Schiller, M., Waldman, T., Cleveland, J.M., Roland, C.L. (2011). Societal Costs of Prescription Opioid Abuse, Dependence, and Misuse in the United States. *Pain Medicine* 12:657-667.

Bjork, J.M., Grant, S.J. (2009). Does Traumatic Brain Injury Increase Risk for Substance Abuse? *Journal of Neurotrauma* 26:1077-1082.

Bogner, J.A., Corrigan, J.D., Mysiw, J., Clinchot, D. Fugate, L. (2001). A Comparison of Substance Abuse and Violence in the Prediction of Long-Term Rehabilitation Outcomes After Traumatic Brain Injury. *Arch Phys Med Rehabil*. 82: 571-577.

Bombardier, C.H., Rimmel, C.T. (1999). Motivational interviewing to prevent alcohol abuse after traumatic brain injury: A case series. *Rehabilitation Psychology*, 44: 52-67.

Brady, K.T., Tuerk P., Back, S.E., Saladin, M.E., Waldrop, A.E., Myrick, H. (2009). Combat posttraumatic stress disorder, substance use disorders and traumatic brain injury. *J. Addict Med*. 3(4):179-188.

Bryant, B.A., Moulds, M., Guthrie, R., Nixon, R.D.V. (2003). Treating acute stress disorder following mild traumatic brain injury. *Am. J. Psychiatry*. 160: 585-587.

Cecil, Sandy, Chen, P.M., Callaway, S.E., Rowland, S.M., Adler, D.E., Chen, J.W. (2010). Traumatic Brain Injury Advanced Multimodal Neuromonitoring From Theory to Clinical Practice. *Crit Care Nurse* 31: 25-37.

Coetzer, R. (2014) Psychotherapy after acquired brain injury: Is less more? *Rev. Chil. Neuropsicol*. 9(1E):8-13.

Comper, P., Bisschop, S.M., Carnide, N., Tricco, A. (2005) A systematic review of treatments for mild traumatic brain injury. *Brain Injury*. 19(11): 863-880.

Corrigan, J.D., Lamb-Hart, G.L., Rust, E. (1995). A programme of intervention for substance abuse following traumatic brain injury. *Brain Injury* 9:221-236.

Corrigan, J.D., Smith-Knapp, K., Granger, C.V. (1998). Outcomes in the first 5 years after traumatic brain injury. *Archives of Physical Medicine Rehabilitation* 79: 298-305.

- Corrigan, J.D., Bogner, J., Lamb-Hart, G. (2005). Increasing substance abuse treatment compliance for persons with traumatic brain injury. *Psycholo. Addict. Behav.* 19:131-139.
- Corrigan, J.D., Bogner, J. (2007). Interventions to promote retention in substance abuse treatment. *Brain Inj.* 21:343-356.
- Corrigan, J.D., Deuschle J. (2008). The presence and impact of traumatic brain injury among clients in treatment for co-occurring mental illness and substance abuse. *Brain Inj.* 22: 223–231.
- Corrigan, J.D., Adams, R.S., Larson, M.J. (2013). When Addiction Co-Occurs With Traumatic Brain Injury. *Am. J. Psychiatry* 170(4):351- 354.
- Drug Facts: Nationwide Trends. National Institute on Drug Abuse (2014). Date Accessed on November 18, 2014.
- Eden, J., & Stevens, R. (2006). Evaluating the HRSA traumatic brain injury program. National Academy Press.
- Fazel, S., Wolf, A., Pillas, D., Lichtenstein, P., Langstrom, N. (2014). Suicide, Fatal Injuries and Other Causes of Premature Mortality in Patients with Traumatic Brain Injury. *JAMA Psychiatry* 71(3): 326-333.
- Felde, A.B., Westermeyer, J., Thuras, P. (2006). Co-morbid traumatic brain injury and substance use disorder: Childhood predictors and adult correlates. *Brain Injury* 20(1): 41-49.
- Ford, J., Moore, D. (1995). Making treatment work. *Magazine* 3: 19-23.
- Graham, D.P., Cardon, A.L. (2008). An update on substance use and treatment following traumatic brain injury. *New York Academy of Sciences*, 1141: 148-162.
- Heinemann, A.W., Corrigan, J.D., Moore, D. (2004). Case management for traumatic brain injury survivors with alcohol problems. *Rehabilitation Psychology.* 49(2): 156-166.
- Hettema, J., Steele, J., Miller, W. R. (2005). Motivational interviewing. *Annual Review of Clinical Psychology*, 1, 91-111.
- Hsieh, M., Ponsford, J., Wong, D., McKay, A. (2012). Exploring variable associated with change in cognitive behaviour therapy (CBT) for anxiety following traumatic brain injury. *Disability & Rehabilitation* 34(5): 408-415.
- Kessler, R.C., Berglund, P., Demler, O., Jin, R., Merikangas, K.R., Walters, E.E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replications. *Arch Gen Psychiatry.* 62: 593- 603.
- Kreutzer, J.S., Witol, A.D., Marwitz, J.H. (1996). Alcohol and drug use among young persons with traumatic brain injury, *Journal of Learning Disabilities* 29(6): 642-651.

- Kurland, D., Hong, C., Aarabi, B., Gerzanich, V., Simard, J.M. (2012). Hemorrhagic Progression of a Contusion after Traumatic Brain Injury: A Review. *Journal of Neurotrauma* 29:19-31.
- Ling, G.S., Marshall, S.A. (2010). Traumatic Brain Injury. Handbook of Neurocritical Care 307-321. New York: Springer.
- Management of Substance Abuse Alcohol. World Health Organization. (2014). Date Accessed on November 18, 2014.
- Menon, D.K., Schwab, K., Wright, D.W., Maas, A.I. (2010). Position Statement: Definition of Traumatic Brain Injury. *Archives of Physical Medicine and Rehabilitation* 91: 1637- 1640.
- Miller, W.R., Rollnick, S. (1991). Motivational Interviewing: Preparing People to Change Addictive Behavior. New York: Guilford Press.
- Morgenstern, J., Irwin, T. (2007). Alcohol and other drug disorders, *Handbook of clinical interviewing with adults* (pp. 301-316). Los Angeles, CA: Sage.
- Ponsford, J., Whelan-Goodinson, R., Bahar-Fuchs, A. (2007). Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury* 21: 1385-1392.
- Olson-Madden, J.H., Forster, J.E., Huggins, J., Schneider, A. (2012). Traumatic Brain Injury and Substance Use Disorder. *J Head Trauma Rehabil* 27(5): 370-378.
- Reiger, D.A., Farmer, M.E., Rae, D.S., Locke, B.Z., Keith, S.J., Judd, L.L., Goodwin, F.K. (1990). Comorbidity of Mental Disorders with Alcohol and Other Drug Abuse. Results from the Epidemiologic Catchment Area (ECA) Study. *JAMA* 264: 2511-2518.
- Shield, K.D., Parry, C., Rehm, J. (2014). Chronic Diseases and Conditions Related to Alcohol Use. *Alcohol Res.* 35(2):155-177.
- Sparadeo, F. R., Gill, D. (1989). Effects of prior alcohol use on head injury recovery. *The Journal of Head Trauma Rehabilitation*, 4(1), 75-81.
- Sparadeo, F. (1992). Substance abuse, brain injury, and family adjustment. *Neurorehabilitation*, 2: 65-73.
- Staff, J.T. (2013). Mild Traumatic Brain Injury Linked to Increased Risk for Addiction: Study.
- Taylor, L.A., Kreutzer, J.S., Demm, S.R., Meade, M.A. (2003). Traumatic brain injury and substance abuse: A review and analysis of the literature. *Neuropsychological Rehabilitation* 13: 165-188.

Thurman, D.J., Alverson, C., Dunn, K.A., Guerrero, J., Sniezek, J.E. (1999). Traumatic Brain Injury in the United States: A Public Health Perspective. *Journal of Head Trauma Rehabilitation* 14: 602-615.

Traumatic Brain Injury. Centers for Disease Control and Prevention. (2014). Date Accessed on September 21, 2014.

Walker, R., Staton, M., Leukfeld, C.G. (2001). History of head injury among substance users: Preliminary findings. *Substance Use & Misuse* 36: 757-770.

Walker, R., Hiller, M., Staton, M., Leukefeld, C.G. (2003). Head Injury among Drug Abusers: An Indicator of Co-Occurring Problems. *Journal of Psychoactive Drugs* 35 (3): 343-353.

Walker, R., Cole, J. E., Logan, T. K., Corrigan, J. D. (2007). Screening substance abuse treatment clients for traumatic brain injury: prevalence and characteristics. *The Journal of head trauma rehabilitation*, 22(6), 360-367.

Werner, C., Engelhard, K. (2007). Pathophysiology of traumatic brain injury. *British Journal of Anaesthesia* 99: 4-9.

West, S. L., Graham, C. W., Cifu, D. X. (2009). Rates of substance abuse treatment denials to persons with physical disabilities due to accessibility concerns. *Alcoholism Treatment Quarterly*, 27, 305–316.

West, S.L. (2011). Substance use among persons with traumatic brain injury: A review. *Substance Use and Neurotrauma* 29: 1-8.

## VITA

Karon Elizabeth Wynne was born in Leesburg, Virginia, on August 7, 1987. She is the daughter of Linda Ann Wynne and Donald Malcolm Wynne and the sister of Alexander Joseph Wynne and Kevin Charles Wynne. She graduated from Fleetwood High School in 2005. Following high school she obtained a Bachelor's of Science degree in Mathematics from West Virginia University in 2009. During that time she worked for the U.S. Department of Energy, which she continued to work for until she matriculated into the neuroscience PhD program at UTMB in the fall of 2010. At UTMB she worked under the guidance of Dr. Douglas DeWitt for her dissertation work, defending her dissertation in July 2015. During her time at UTMB she was recognized as an Albert Schweitzer Fellow, a Columbia Crew Memorial Scholar and inducted into Sigma Xi, the scientific honorary society.

### Education

B.S. Mathematics, December 2009, *West Virginia University, Morgantown, WV*

Ph.D. Neuroscience, July 2015, *University of Texas Medical Branch, Galveston, TX*

### Publications

- 1) Petrov, A., **Wynne, K.E.**, Parsley, M.A., Petrov, I.Y., Petrov, Y., Ruppert, K.A., Prough, D.S., DeWitt, D.S., Esenaliev, R.O. (2014) "Optoacoustic detection of intra- and extracranial hematomas in rats after blast injury." *Photoacoustics* 2:75-80.

### Dissertation

- 2) **Wynne, K.E.** The Effects of Dendro[60]fullerene on neurobehavioral outcome and cerebral vascular function after traumatic brain injury. PhD dissertation. University of Texas Medical Branch. July 2015.

## Refereed Conference Manuscript

- 3) Petrov, I.Y., Fonseca, R.A., Richardson, C.J., Petrov, Y., Prough, D.S., Petrov, A., **Wynne, K.E.**, Westermann, S., Esenaliev, R.O. “Monitoring cerebral venous blood oxygenation in neonates with a medical-grade optoacoustic system.” International Society for Optics and Photonics (SPIE) Photonics West 2015, San Francisco, California, February 7-12, 2015.
- 4) Petrov, A., **Wynne, K.E.**, Prough, D.S., DeWitt D.S., Petrov, Y., Parsley, M.A., Esenaliev, R.O. Optoacoustic detection and monitoring of blast-induced intracranial hematomas in rats. International Society for Optics and Photonics (SPIE) Photonics West 2015, San Francisco, California, February 1-6, 2014.
- 5) **Wynne, K.E.**, DeWitt, D.S., Petrov, Y.Y., Petrov, I.Y.H., Petrov, A., Parsley, M.A., Esenaliev, R.O., Prough, D.S. “Noninvasive optoacoustic monitoring of cerebral venous blood oxygenation in rats with blast-induced traumatic brain injury.” International Society for Optics and Photonics (SPIE) Photonics West 2013, San Francisco, California, February 2-7, 2013.
- 6) D.K. Weiss, N.S. Levine, E.K. Beutel, N. De Munster, L.G. Barajas, **K.E. Wynne**, A. Stein, C. Runyon, “Mapping Rover Routes and Hydrous Soil Location on the Mars Desert Research Station.” 43<sup>rd</sup> Lunar & Planetary Science Conference 2012, Woodlands, Texas, March 19 – 23, 2012.
- 7) I.Y. Petrov, **K.E. Wynne**, Y.Y. Petrov, R.O. Esenaliev, C.J. Richardson, D.S. Prough, “Noninvasive Optoacoustic Monitoring of Cerebral Venous Blood Oxygenation in Newborns.” International Society for Optics and Photonics (SPIE) Photonics West 2012, San Francisco, California, January 21 – 26, 2012.
- 8) D. Tucker, A. Manivannan, D. Haynes, H. Abernathy, N. Miller, **K. Wynne**, A. Matos, “Evaluating Methods for Infiltration of LSCF Cathodes With Mixed Electronic/Ionic Conductors for Improved Oxygen Exchange,” ASME 2010 8<sup>th</sup> International Conference on Fuel Cell Science, Brooklyn, New York, June 14 -16 2010.

## Abstracts

- 8) Petrov, I.Y., Fonseca, R.A., Richardson, C.J., Petrov, Y., Prough, D.S., Petrov, A., **Wynne, K.E.**, Westermann, S., Esenaliev, R.O. “Monitoring cerebral venous blood oxygenation in neonates with a medical-grade optoacoustic system.” International Society for Optics and Photonics (SPIE) Photonics West 2015, San Francisco, California, February 7-12, 2015.

- 9) **K.E. Wynne**, Y.P., Zeng, D.S. Prough, D.S. DeWitt, “Effects of Dendro[60]fullerene on Neurobehavioral Outcome and Cerebral Vascular Function After Traumatic Brain Injury.” Mission Connect Symposium 2014, Houston, Texas, December 2014.
  
- 10) **Wynne, K.E.**, Prough, D.S., DeWitt, D.S. “Investigation of the effects of Carboxyfullerene Nanoparticles on the cerebral vasculature after fluid percussion injury.” National Neurotrauma Symposium 2014, San Francisco, California June 29 – July 2, 2014.
  
- 11) Petrov, A., **Wynne, K.E.**, Prough, D.S., DeWitt, D.S., Petrov, Y.Y., and monitoring of blast-induced intracranial hematomas in rats.” International Society for Optics and Photonics (SPIE) Photonics West 2014, San Francisco, California, February 2014.
  
- 12) **K.E. Wynne**, D.S. Prough, D.S. DeWitt, “Investigation of Behavioral Changes Carboxyfullerene Nanoparticles Treatment after Moderate Traumatic Brain Injury,” Mission Connect Symposium 2013, Houston, Texas, December 2013.
  
- 13) **K.E. Wynne**, D.S. Prough, D.S. DeWitt, “Treatment with Carboxyfullerene Nanoparticles Reduces Cognitive Deficits after Moderate Traumatic Brain Injury,” National Neurotrauma Symposium 2013, Nashville, Tennessee August 4 – 7, 2013.
  
- 14) **Wynne, K.E.**, DeWitt, D.S., Petrov, Y.Y., Petrov, I.Y.H., Petrov, A., Parsley, M.A., Esenaliev, R.O., Prough, D.S. “Noninvasive optoacoustic monitoring of cerebral venous blood oxygenation in rats with blast-induced traumatic brain injury.” International Society for Optics and Photonics (SPIE) Photonics West 2013, San Francisco, California, February 2-7, 2013.
  
- 15) **K.E. Wynne**, Y. Zeng, D.S. Prough, D.S. DeWitt, “Carboxyfullerene Nanoparticles Reduce Oxidative Stress After Rapid Stretch Injury In Vascular Smooth Muscle Cells, in vitro” Mission Connect Symposium 2012, Houston, Texas, December 7, 2012.
  
- 16) **K.E. Wynne**, D.S. Dewitt, Y. Petrov, I.Y. Petrov, A. Petrov, M.A. Parsley, K.A. Ruppert, R.O. Esenaliev, D.S. Prough, “The Use of Optoacoustic Monitoring to Measure Cerebral Oxygen Saturation in Rats Subjected to Blast-Induced Neurotrauma,” Mission Connect Symposium 2012, Houston, Texas, December 7, 2012.

- 17) **K.E. Wynne**, Y. Zeng, D.S. Prough, D.S. DeWitt, “Carboxyfullerene Nanoparticles Reduce Oxidative Stress after Rapid Stretch Injury in Vascular Smooth Muscle Cells,” National Neurotrauma Symposium 2012, Phoenix, Arizona July 22 – 25, 2012.
  
- 18) D.K. Weiss, N.S. Levine, E.K. Beutel, N. De Munster, L.G. Barajas, **K.E. Wynne**, A. Stein, C. Runyon, “Mapping Rover Routes and Hydrous Soil Location on the Mars Desert Research Station.” 43<sup>rd</sup> Lunar & Planetary Science Conference 2012, Woodlands, Texas, March 19 – 23, 2012.
  
- 19) **K.E. Wynne**, D.S. Dewitt, Y.Y. Petrov, I.Y. Petrov, M.A. Parsley, R.O. Esenaliev, D.S. Prough, “Optoacoustic Monitoring of Cerebral Venous Blood Oxygenation in Rats With Traumatic Brain Injury.” (Oral Presentation) International Society for Optics and Photonics (SPIE) Photonics West 2012, San Francisco, California, January 21 – 26, 2012
  
- 20) I.Y. Petrov, **K.E. Wynne**, Y.Y. Petrov, R.O. Esenaliev, C.J. Richardson, D.S. Prough, “Noninvasive Optoacoustic Monitoring of Cerebral Venous Blood Oxygenation in Newborns.” International Society for Optics and Photonics (SPIE) Photonics West 2012, San Francisco, California, January 21 – 26, 2012
  
- 21) **K.E. Wynne**, J. Richardson, R. Esenaliev, I. Petrov, Y. Petrov, D.S. Prough, “Optoacoustic Monitoring of Oxygen Saturation in the Superior Sagittal Sinus of Neonates,” (Oral Presentation) Mission Connect Symposium 2011, Houston, Texas, December, 2011
  
- 22) **K.E. Wynne**, J. Richardson, R. Esenaliev, I. Petrov, Y. Petrov, D.S. Prough, “Optoacoustic Monitoring of Oxygen Saturation in the Superior Sagittal Sinus of Neonates,” American Society of Anesthesiology 2011, Chicago, Illinois, October 15 – 19, 2011
  
- 23) **K.E. Wynne**, J. Richardson, R. Esenaliev, I. Petrov, Y. Petrov, D.S. Prough, “Optoacoustic Monitoring of Oxygen Saturation in the Superior Sagittal Sinus of Neonates,” National Neurotrauma Symposium 2011, Fort Lauderdale, Florida, July 10 - 13, 2011
  
- 24) D. Tucker, A. Manivannan, D. Haynes, H. Abernathy, N. Miller, **K. Wynne**, A. Matos, “Evaluating Methods for Infiltration of LSCF Cathodes With Mixed Electronic/Ionic Conductors for Improved Oxygen Exchange,” ASME 2010 8<sup>th</sup> International Conference on Fuel Cell Science, Brooklyn, New York, June 14 -16 2010

Permanent address: 532 Walnut Tree Drive; Blandon, PA 19510

This capstone was typed by Karon Wynne.