Spring 2015

Traumatic experiences and cognition: How do static and dynamic variables contribute to current functioning?

Cathryn Richmond
James Madison University

Approved and recommended for acceptance as a thesis in partial fulfillment of the requirements for the degree of Master of Arts

Special committee directing the thesis work of Cathryn Richmond

____

Bernice Marcopulos Date

____

Gregg Henriques Date

____

Lennis Echterling Date

Received by The Graduate School

____

Date

Traumatic Experiences and Cognition:

How Do Static and Dynamic Variables

Contribute to Current Functioning?

Cathryn E. Richmond

A thesis submitted to the Graduate Faculty of

JAMES MADISON UNIVERSITY

In

Partial Fulfillment of the Requirements

for the degree of

Master of Arts

Psychological Sciences

May 2015

Acknowledgements

I would like to express my sincere gratitude to my advisor Dr. Bernice Marcopulos for her continuous support of my research and her guidance throughout the program. I would also like to thank the other members of my committee, Dr. Gregg Henriques and Dr. Lennie Echterling for their kind encouragement, insightful comments, and constructive criticism. Additionally, I would like to thank Dr. Beth Caillouet-Arredondo for her amazing mentorship throughout this entire process and her knowledgeable assistance in improving the quality of both this project as well as my research background as a whole. I would also like to extend my gratitude to fellow students Shannon Kovach and Chris Hill for their assistance in the data collection process, as well as Kate Pinder for her frequent statistical guidance. In addition, I would like to thank my supervisor, Dena Pastor, and my fellow colleagues at CARS for their patience and helping to ensure I was properly prepared to undertake this endeavor. Last but not least, I would like to thank my parents, Dan and Laurey Richmond, for their amazing support and encouragement throughout my entire academic career.

Table of Contents

Acknowledgements ii

List of Tables vi

Abstract viii

Section I: Overview of Literature Review 1

1.1 Neurological Basis 4

1.2 Stress as a Mediator 10

1.3 Current Study 15

Section II: Research Questions 17

Section III: Description of Measurement and Methods 23

Section IV: Description of Data Analysis 33

Section V: Results 37

5.1 Primary Analyses 39

5.1.1 Static factors versus dynamic factors 40

5.1.2 Best models, impact of any trauma 41

5.2 Supplementary Analyses 43

5.2.1 RCFT and TMT model comparison 43

5.2.2 Psychotic diagnosis 44

5.3 Secondary Analyses 45

5.3.1 Impact of psychological trauma 46

5.3.2 Impact of polyvictimization 47

5.3.3 Impact of type of trauma (intentional/unintentional) 48

Section VI: Discussion 50

6.1 Primary Hypotheses 51
6.1.1 Static factors versus dynamic factors 51
6.1.2 Best models, impact of any trauma 51
6.2 Supplementary Hypotheses 52
6.2.1 RCFT and TMT model comparison 52
6.2.2 Psychotic diagnosis 53
6.3 Secondary Hypotheses 53
6.3.1 Impact of psychological trauma 53
6.3.2 Impact of polyvictimization 54
6.3.3 Impact of type of trauma (intentional/unintentional) 54
6.4 Conclusion 55
6.5 Strengths, Limitations, and Implications 57
Section VII: References 65
Section VIII: Tables 84
Appendix A: Life Events Checklist, Modified Trauma Thesis Version 106
Appendix B: RCFT Figure 107
Appendix C: TMT Part A 108
Appendix D: TMT Part B 109
Appendix E: Supplementary Results 110
E1. Interactions 110
E2. Best Model 111
E3. RCFT and TMT 112
E4. IQ 113

E5. Psychosis.. 114

List of Tables

1. Correlations Among Scores and Predictors 84

2. Dynamic Predictors of BQSS Copy Score 85

3. Static Predictors of BQSS Copy Score 85

4. Dynamic Predictors of BQSS Organization Score 86

5. Static Predictors of BQSS Organization Score 86

6. Predictors of BQSS Copy Score 87

7. Predictors of BQSS Organization Score 88

8. Predictors of RCFT Score 89

9. Predictors of TMT Part B Score 90

10. Predictors of TMT Part A Score 91

11. Comparison of Performance Across Measures Based on Presence of Secondary Gain 92

12. Comparison of Performance Across Measures Based on History of Any Trauma/Head Injury 92

13. Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses 93

14. Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses 94

15. Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses 95

16. Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses .. 96

17. Comparison of BQSS Copy and Organization Performance Based on Diagnosis and History of Psychological Trauma 97

18. Polyvictimization and Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma 98

19. Polyvictimization and Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma 99

20. Polyvictimization and Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma 100

21. Polyvictimization and Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma 101

22. Type of Trauma and Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma 102

23. Type of Trauma and Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma 103

24. Type of Trauma and Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma 104

25. Type of Trauma and Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma 105

Abstract

Approximately 25% of children in the United States will witness or experience a traumatic event before age 4, and individuals with a history of trauma, particularly traumatic events in childhood, have a much higher likelihood of developing psychopathology in adulthood. Prior research indicates that the vast majority of individuals with a serious mental illness, particularly those in community mental health centers and psychiatric inpatient settings, have experienced at least one traumatic event in their lifetime. These individuals require special consideration in treatment planning, and a large range of neurodevelopmental and environmental factors must be taken into account when interpreting results of neuropsychological assessment. The current study examines the impact of both “dynamic” factors such as age and diagnosis as well as “static” factors such as trauma history and IQ on performance on two executive functioning measures in a psychiatric inpatient population. Results suggest that while performance is impacted by IQ for executive functioning assessments as a whole, factors such as a history of trauma, the type of traumatic events experienced, psychosis, and the presence of secondary gain may differentially impact performance depending on the specific cognitive functioning abilities being assessed (e.g., basic versus executive).

Keywords: Trauma, neuropsychological assessment, stress, cognition, executive functioning, serious mental illness, psychosis, IQ, secondary gain, intentional, polyvictimization

Childhood Traumatic Experiences and Cognition: How Do Static and Dynamic Variables Contribute to Current Functioning?

Section I: Overview of Literature Review

The American Psychological Association defines trauma as, "an emotional response to a terrible event like an accident, rape or natural disaster" (2014, para. 1). Childhood trauma refers to any traumatic experiences that occur prior to age 18. However, the question of specific events that are considered "traumatic" is a subject of great debate in the field of psychology, particularly in regards to potential differentiation between stressful life events and traumatic life events (McHugo et al., 2005). For instance, the Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria for Posttraumatic Stress Disorder (PTSD) differentiate between stressful and traumatic life events, defining trauma as a stressor in which the person "was exposed to: death, threatened death, actual or threatened serious injury, or actual or threatened sexual violence," either directly or indirectly (American Psychiatric Association, 2000, p. 467). However, this definition is rather restrictive, excluding events such as divorce and emotional and verbal abuse that can result in detrimental outcomes beyond the criteria for a PTSD diagnosis.

In response to such gaps in many trauma definitions, a number of prior researchers (Breslau & Davis, 1987; McHugo et al., 2005; Solomon & Canino, 1990) have argued that any stressful life events can be considered traumatic, and this relationship is dependent on whether the individual is able to appropriately cope with the stressor. Specifically, it is suggested that a traumatic event is any event that is shocking to the individual, regardless of form, and as such produces symptoms of traumatic stress. These events can take a variety of forms, including natural disasters, fires or explosions, vehicle or work accidents, physical or sexual assault, combat or war-zone exposure, captivity, life-threatening illness or injury, severe human suffering, sudden violent or unexpected deaths, and causing harm to others (Weathers et al., 2013). For the purposes of the present article, trauma is defined in the context of this latter conceptualization referring to any life events that result in severe and/or chronic stress.

The prevalence of individuals experiencing a traumatic event is devastatingly high. For example, Norris (1992) conducted an interview-based study of 1,000 participants from four large cities in North Carolina, South Carolina, and Georgia, with the final sample consisting of approximately equal numbers of Caucasians and African-Americans, males and females, and young, middle-aged, and older participants. This study assessed the frequency and impact of nine traumatic life events: robbery, physical assault, sexual assault, tragic death, motor vehicle crash, combat, fire, other disaster, and other hazard.

Results indicated that across demographic groups, nearly 70% of the sample had experienced at least one traumatic event in their lifetime, including 21% of the sample experiencing a violent event in the last year alone. Moreover, younger participants were more likely to exhibit the symptoms of PTSD after experiencing a traumatic event compared to middle-aged and older participants (Norris, 1992). Furthermore, Norris' data suggest that a given individual is more likely to experience a traumatic event at least once in their lifetime than not experiencing any trauma. These results highlight both the high prevalence of traumatic histories across demographic groups as well as the need for early intervention, particularly for younger individuals.

Approximately 25% of children in the United States will witness or experience a traumatic experience before age 4 (Briggs-Gowan, Ford, Fraleigh, McCarthy, & Carter, 2010). Estimates of the occurrence of childhood maltreatment in the entire population have ranged from approximately 15 to 55%. However, though this wide range is most frequently due to reporting error (e.g., caregivers withholding information, memory effects), differing samples (e.g., children of varying ages, retrospective studies in adults, consumers of an array of mental health services, individuals with a variety of comorbid complications), and differing operational definitions of childhood maltreatment utilized across studies (e.g., assessment only of physical and sexual abuse, inclusion or exclusion of emotional trauma), the majority of prior research estimates approximately 30-45% of the population have experienced childhood maltreatment.

Briere and Elliott's (2003) study of 934 adults from the general population indicated that 37% of their sample reported experiencing physical or sexual maltreatment in childhood. Copeland, Keeler, Angold, and Costello's (2007) study found that of their sample of 1420 children (aged 9, 11, and 13 upon enrollment), 30.8% had experienced a traumatic event (not limited to maltreatment) by age 16 and 37.0% of those participants had experienced two or more traumatic events, as measured by the Child and Adolescent Psychiatric Assessment (CAPA).

Prior research has indicated that trauma exposure is one of the strongest predictors of subsequent mental health status (Arnow, 2004; Nemeroff et al., 2003; Schoedl et al., 2010; Steel, Silove, Phan, & Bauman, 2002; Wingenfeld et al., 2011). Young children exposed to five or more significant adverse experiences in the first three years of childhood face an approximated 76% likelihood of having one or more delays in their language, emotional or brain development (Substance Abuse and Mental Health Services Administration, 2011). As the number of traumatic events experienced during childhood increases, the risk for health problems in adulthood increases, including depression, alcoholism, drug abuse, suicide attempts, heart and liver diseases, pregnancy problems, high stress, uncontrollable anger, and family, financial, and job problems (Centers for Disease Control and Prevention, 2014; Edwards et al., 2005).

1.1 Neurological Basis

The term allostasis refers to the concept of "maintaining stability…through change" where the body adapts to stress in order to return to a state of homeostasis (McEwen, 2000). The allostatic model (McEwen & Stellar, 1993) posits that during periods of chronic stress, the brain engages in allostatic accommodation, in which the structures involved in the stress response are constantly readjusting in an attempt to find the best balance between homeostasis and the stress response in order to cope with the presented stressor (Broderick & Blewitt, 2014). This results in an allostatic load in which the related structures experience fatigue as a result of the brain being unable to return to homeostasis (Broderick & Blewitt, 2014). This allostatic load can develop due to repeated reactions to multiple novel stressors, lack of adaptation, or inadequate or prolonged stress responses (McEwen, 2000), all of which are frequent occurrences for those that experience trauma.

Glucocorticoids such as cortisol are instrumental in terminating the stress response, such that insufficient or excessive cortisol levels are related to a hyperactive stress response and are viewed as indicators of chronic stress (Broderick & Blewitt, 2014; Frodl & O'Keane, 2013). The majority of trauma survivors react to traumatic experiences in one of two ways: either maintaining a constant state of severe stress in which cortisol levels are increased, or attempting to suppress the feeling of stress while maintaining the stressor itself, resulting in decreased cortisol levels. Though both are maladaptive methods of coping, the former relates to an attempt to *return to* allostasis in which the individual is "overwhelmed" by the stressor, resulting in little to no regulation of stress. The latter relates to an attempt to *maintain* allostasis in which the individual is "hiding" from, and therefore over-regulating, stress.

For example, stress has been found to initially induce a significant increase in cortisol (Heim et al., 2000), and those with a history of trauma typically have lower overall cortisol levels compared to those without trauma exposure (Yehuda, Halligan, Golier, Grossman, & Bierer, 2004), particularly for individuals with mediating symptomology such as depression. For instance, children who experience sexual abuse before age 5, primarily those with higher internalizing symptoms, often demonstrate a slower decrease in cortisol levels after experiencing a stressor and an atypical flattening (i.e., lack of change) of overall daily cortisol levels compared to those that experienced abuse after age 5 and/or with lower internalizing symptoms (Cicchetti, Rogosch, Gunnar, & Toth, 2010). Similarly, women with a history of sexual abuse and current depression frequently have higher cortisol levels after experiencing a stressor compared to those without a history of sexual abuse and/or without current depression (Heim et al., 2000).

Furthermore, abnormal cortisol levels have been found to negatively impact the size and functionality of the hippocampus (McEwen & Sapolsky, 1995). The hippocampus is part of the limbic system, which is the portion of the brain that is intimately involved in the processing of emotions (Broderick & Blewitt, 2014). Specifically, the hippocampus is critical in memory functioning (Broderick & Blewitt, 2014; Squire, 1992), evaluating the context of life events (Fink, 2009), and inhibiting the stress response once activated by the amygdala (McEwen & Gianaros, 2010).

Prior research has demonstrated a relationship between increased cortisol responses and decreased hippocampal volume, particularly in individuals with low self-esteem and external locus of control (Pruessner et al., 2005), and elevated glucocorticoid levels and long-term cortisol increases have been found to be associated with hippocampal damage and dysfunction in addition to volume and atrophy (Lupien et al., 1998). In particular, there is support for a relationship between these abnormal hormone levels and decreased dendritic functionality in the hippocampus in a variety of species, including rats (Watanabe, Gould, Daniels, Cameron, & McEwen, 1992), tree shrews (Magariños, McEwen, Flügge, & Fuchs, 1996), and monkeys (Sapolsky, Uno, Reber, & Finch, 1990). For example, results of Woolley, Gould, and McEwen's (1990) study supported a relationship between high cortisol levels and dendritic atrophy, specifically decreased dendritic branching and dendritic length.

Dendritic spines are the primary sites for synaptic input and have been hypothesized to be fundamental in synaptic plasticity (Anderson, Spencer-Smith, & Wood, 2011; Crick, 1982; Engert & Bonhoeffer, 1999), such that dendritic structure greatly influences the functionality of the brain. As stated by Kolb and Wishaw (1998, pp. 59-60), "[Those] with extensive dendritic growth… show facilitated performance on many types of behavioral measures. In contrast, [those] with atrophy in dendritic arborization show a decline in behavioral capacity. Similarly, factors that enhance dendritic growth…facilitate behavioral outcome, whereas factors that block dendritic growth…retard functional outcomes…If neurons have more connections, they are hypothesized to have more influence on the observed behavior." For instance, a lack of dendritic spine maturation is a common feature of intellectual disabilities (Harris, 1999), suggesting that dendrites are critical to cognitive functioning, particularly executive functioning.

Moreover, prior research suggests an association between dendritic branching and the quality of the environment, such that a lack of stimulation or an adverse environment has been demonstrated to negatively impact the structure and frequency of dendrites, particularly in the hippocampus. For example, Yasumatsu, Matsuzaki, Miyazaki, Noguchi, and Kasai's (2008) study supported rapid responses of hippocampal dendritic spines to stimulation and indicated a significant correlation between dendritic spine volume with memory, age, and life expectancy.

Prior research has found support for increased dendritic branching in all cortical layers of brains that develop in enriched environments compared to standard or isolated environments (Bryan & Riesen, 1989; Coleman & Riesen, 1968; Greenough & Volkmar, 1973; Johansson & Belichenko, 2002), suggesting that negative environments are harmful to dendritic development. In addition, results of Engert and Bonhoeffer's (1999) study suggest that spine formation on developing dendrites may be related to highly selective activation, such as long-term enhancement of synapses fostered by the environment. Results of the 2009 study by Livneh, Feinstein, Klein, and Mizrahi suggest not only that synaptogenesis is continuously regulated but also that neuronal development is impacted by sensory activity such as odor, such that higher levels of activity are related to increased numbers of potential synapses.

As stress negatively impacts the structure, presence, and functionality of dendrites, the connectivity between various parts of the brain is compromised, resulting in negative impacts on learning new information. This is particularly damaging when it occurs during critical developmental phases of crucial brain domains, such as the pre-frontal cortex (Broderick & Blewitt, 2014; Frodl & O'Keane, 2013). The frontal lobe, particularly the pre-frontal cortex, receives input from all major sensory afferent systems (Passler, Isaac, & Hynd, 1985) and is widely regarded as the primary brain structure involved in goal-directed behavior, commonly referred to as executive functioning (Best & Miller, 2010). Executive functions are critical to overall functioning, including planning, organization, impulse control, and set-shifting (Welsh, Pennington, & Groisser, 1991), and build upon more basic functions of the sensory systems such as motor skills and verbal speech.

Critical structural organization of the brain develops prenatally and during childhood (Anderson et al., 2011; Perry, 1997). This sequential development assists in mediating reactive impulses characteristic of the more primitive areas of the brain responsible for lower-level processing, such that children with normal brain development are able to react less impulsively in a more socially-acceptable manner as they age. This development of behavioral inhibition is dependent on environmental cues, such that lack or disruption of these cues can result in abnormal brain development and thus diminished functionality in the disrupted areas. For example, Perry's (1997) preliminary studies demonstrated cortical atrophy of neglected children, suggesting that lack of type and quality of stimulation is associated with underdevelopment of the cortex, such that children who have experienced global environmental neglect were found to have smaller cortical and sub-cortical areas that subsequently atrophied. As a result of the sequential nature of the development, disturbances earlier in life result in more severe dysfunction throughout the lifespan due to each stage of brain development being dependent on successful development during prior stages.

As the development of the pre-frontal cortex occurs after the development of other brain structures and continues into adulthood, disruptions in typical development of any brain structure due to complications such as trauma can negatively impact the functionality of the pre-frontal cortex, such as executive functioning. However, as executive functioning is so complex and such a critical component of overall functioning, these deficits can manifest in a variety of ways, including verbal fluency, set-shifting, mental flexibility, and visual reconstruction.

Welsh et al. (1991) suggest that there are three major stages in development of executive functioning skills – age six, age ten, and adolescence. Planning and organization are in development at age six, complex organization and impulse control develops until age ten, and verbal fluency, complex planning, and motor sequencing continue developing throughout adolescence (Welsh et al., 1991). For example, results of Becker, Issac, and Hynd's (1987) study indicated that younger children up to eight years old had more difficulty inhibiting motor behaviors and retaining order of nonverbal, visual designs than older children, suggesting that the related processes are still developing until age eight. Furthermore, Best and Miller's (2010) study supported the concept that the most improvement in inhibition, working memory, and hand motor responses occurs in preschool between ages three and four.

Taken together, prior research indicates that traumatic settings, such as neglectful or abusive environments, result in an allostatic load that contributes to abnormal cortisol levels. These abnormal hormone levels restrict or even hinder dendritic branching in developing brains, particularly the hippocampus and related brain structures, which results in dysfunction of these structures, such as deficits in higher-level functioning including executive functioning (Frodl & O'Keane, 2013).

1.2 Stress as a Mediator

Three distinct types of stress have been identified by Harvard University's Center on the Developing Child (2012) – positive, tolerable, and toxic. The positive stress response is the brief "normal" response that is typical of healthy development, while the tolerable stress response is the stronger though temporary response typically seen when a person experiences the loss of a loved one, a natural disaster, etc. The toxic stress reaction, however, is a severe, prolonged reaction that occurs with intense and/or frequent adverse events such as abuse, specifically in the absence of social support, which disrupts the development of the brain.

It has been suggested by Perry (1997) among others that a persistently active stress response during development will result in the development of an essentially cumulative stress response that is both hypersensitive and overactive. Though this chronic stress response is beneficial to a child in a chaotic environment as the child will be hypervigilant and hypersensitive to external stimuli, it will hinder the child in other environments. For example, many children that were exposed to chronic neurodevelopmental trauma are diagnosed with Attention Deficit Hyperactivity Disorder (ADHD) due to their hypervigilance resulting from a use-dependent organization of the brain involved in the stress response (Perry, 1997). Similarly, Frodl and O'Keane (2013) describe a direct association between chronic stress and later psychopathology, such as depression. In addition, in Steel et al.'s (2002) study, participants who had been exposed to more than three traumatic events were at a significantly higher risk of developing mental illness over the following ten years compared to those without trauma exposure.

Traumatic experiences in childhood are particularly devastating as defensive capabilities increase with age, such that children have limited capability to cope with these events (Perry, 1997). For example, childhood traumatization is significantly related to a range of psychopathology in later adulthood, such as panic/anxiety disorders, PTSD, depression, and substance abuse (Arnow, 2004). Similarly, Schoedl et al.'s (2010) study found that those who experienced their first trauma before age 12 were more likely to experience depression in adulthood. Moreover, the 2011 study by Wingenfeld et al. supported a strong relationship between general traumatization and dissociation; emotional abuse and depression; and sexual abuse and depression, dissociation, and symptoms of Borderline Personality Disorder and PTSD.

In addition, results of Nemeroff et al.'s (2003) study suggest that the etiology and pathology of chronic depression in adulthood differs based on the presence or absence of a trauma history. Participants without a history of trauma were equally likely to benefit from either psychotherapy or pharmacotherapy, and were much more likely to benefit from a combination of the two. Participants with a traumatic history, however, benefited far more from psychotherapy than pharmacotherapy, and the majority demonstrated limited benefit of a combination compared to psychotherapy alone (Nemeroff et al., 2003). This suggests that the etiology of adult mental illnesses may be more heavily impacted by traumatic experiences as compared to individual neurological differences, and that this etiology is critical component of effective treatment.

In addition, research suggests that the type of trauma has been found to impact subsequent functioning. For example, results of DiScala, Sege, Li, and Reece's (2000) 10-year retrospective study of children that were hospitalized for blunt trauma indicated that there were significant group differences between the children that experienced abuse compared to an accidental injury. In particular, abused children tended to be significantly younger and more likely to have obtained an injury in infancy compared to children that were victims of accidental injury, and these injuries were more likely to be severe, occur in the home, and impact multiple body regions. Furthermore, children in the abuse group were more likely to sustain injury to the thorax and abdomen, have an intracranial injury, and be diagnosed with a retinal hemorrhage compared to children in the accidental group. Child victims of abuse were also more likely to have a prior medical history and/or concomitant medical complications at the time of hospitalization compared to those that were victims of accidental injury. In addition, social deprivation is believed to be associated with "intentional" traumas such as physical or sexual abuse but not "unintentional" traumas such as a car accident or natural disaster (Hamel, Pampalon, & Institut national de santé publique du Québec, 2002).

These relationships are mediated by a variety of potential risk and resilience factors that can impact a child's likelihood of developing subsequent psychopathology. For example, Wingenfeld et al.'s (2011) study suggested that perceived stress is a strong predictor of psychopathology, even when controlling for traumatic experiences. Furthermore, Nikulina and Widom's (2013) study found support for PTSD symptoms mediating the relationship between childhood maltreatment and higher-order adult functioning on the Trail Making Test (TMT) Part A, an executive functioning assessment that primarily focuses on psychomotor speed and sequencing. Moreover, results of Ford and Kidd's (1998) study suggest that a history of childhood trauma is highly correlated with Disorders of Extreme Stress Not Otherwise Specified, and that these disorders are highly predictive of poor outcomes for PTSD treatment even when controlling for trauma history. These findings suggest that the relationship between trauma and psychopathology may be mediated by stress, and that this stress response is related to a disruption of brain activity in situations of severe and chronic early life stress.

Prior research has found support for the presence of a stable, supportive adult figure as a protective factor for traumatized children, particularly those exposed to violence (Perry, 1997). Similarly, positive peer relationships have been demonstrated to be a resilience factor for children in stressful situations, such as those affected by HIV/AIDS (Mann, 2002). Moreover, community support such as neighborhoods, schools, and governmental support can be instrumental in a child's ability to cope with adverse events (Boyden & Mann, 2005), suggesting that any form of positive social support can serve as a protective factor for traumatized children. Abilities such as reading skills in elementary school have been found to be strong predictors of resiliency in high-risk children (Werner, 1993), and characteristics such as high self-esteem and internal locus of control have been found to frequently be related to decreased cortisol levels in response to psychosocial stress as well as increased hippocampus volume (Pruessner et al., 2005), decreasing the likelihood of subsequent psychopathology.

Though there has been a wide variety of research related to the association between trauma and psychopathology, to the author's knowledge there is only one prior study examining the relationship between childhood trauma and adult executive functioning. The 2013 study by Nikulina and Widom assessed the relationship between childhood maltreatment and executive functioning in adulthood via the TMT. Results for Part A indicated that childhood maltreatment, including neglect, physical abuse, and sexual abuse, was a strong predictor of poor executive functioning performance. Results for Part B indicated that childhood neglect predicted poorer performance, though the data did not demonstrate a significant relationship between executive functioning and childhood sexual or physical abuse.

However, the sample included far more participants who had experienced neglect compared to physical or sexual abuse, suggesting the study might have lacked sufficient power needed to detect a significant relationship. In addition, their sample was not restricted to participants with mental illnesses. Prior research indicates that the vast majority of individuals with a serious mental illness, particularly those in community mental health centers and psychiatric inpatient settings, have experienced at least one traumatic event in their lifetime (Cusack, Frueh, & Brady, 2004; Switzer et al., 1999). For example, Mueser et al.'s (1998) study of individuals with serious mental illness receiving public mental health services indicated that a staggering 98% of their sample had experienced at least one traumatic event. Cusack, Grubaugh, Knapp, and Frueh's (2006) study of individuals with serious mental illness in a psychosocial rehabilitation program indicated that 87% of their sample had been exposed to a traumatic event. Davies-Netzley, Hurlburt, and Hough's (1996) study of homeless women indicated that 76.7% of participants had experienced either physical or sexual abuse in childhood alone.

The prevalence of traumatic histories in individuals with serious mental illness is perhaps the highest in psychiatric inpatient settings. For example, Escalona, Tupler, Saur, Krishnan, and Davidson's (1997) study of 343 psychiatric inpatients indicated that 84% of the sample had experienced at least one traumatic event, with an average of two to three events per consumer. Draijer and Langeland's (1999) research suggested that trauma histories are more common in inpatient psychiatric settings compared to the general population, including 42.1% of the 160 participants reporting experiencing sexual and/or physical abuse alone.

1.3 Current Study

Taken together, the existing literature suggests that the earlier a traumatic event occurs, the less cognitive and psychological resources the individual has to cope with the trauma, and this difficulty coping results in an increase in chronic stress. This increase in chronic stress often occurs during critical periods of development of the prefrontal cortex, widely regarding as a primary area utilized for executive functioning. As a result of the chronic stress, the connections between the prefrontal cortex and hippocampus do not develop properly, leading to deficits in overall functionality, particularly executive functioning abilities.

Nevertheless, there appear to be troubling gaps in the existing research related to trauma and subsequent functioning. Many of these gaps relate to the varying definitions of traumatic experiences, particularly as such a large proportion of previous studies utilize the DSM definition of trauma, which excludes emotionally traumatic experiences. However, given the empirical support for differential impacts of the presence or absence of a trauma history, the type and frequency of traumatic experiences, the age at which events were experienced, and stress overall on brain development and current functioning, the DSM definition of trauma is perhaps insufficient in best assessing the effects of traumatic experiences on subsequent functioning.

In addition, as executive functioning plays such a critical role in overall functioning and childhood trauma is believed to be one of the strongest predictors of overall functioning, particularly executive functioning abilities and development of subsequent psychopathology, the limited research related to the impact of childhood trauma on adult executive functioning is problematic. This discrepancy is even further worrisome when taken in the context of the overwhelming prevalence of trauma histories, particularly in childhood, in the overall population and specifically in mental health settings.

As prior research suggests that trauma exposure is one of the strongest predictors of subsequent mental health status and overall functionality, further examination of the relationship between trauma and specific aspects of functionality such as executive functioning as well as the interaction of multiple potentially mediating factors is needed to better understand this complex relationship. Furthermore, given the extremely high prevalence of traumatic histories in individuals with serious mental illness, examination of the impact of factors unique to this population (e.g., diagnosis, duration of illness, age of onset) as well as their interactions with more universal factors that are also common to the larger population of individuals without a serious mental illness (e.g., trauma, age, IQ, socio-economic status) would be beneficial in informing effective treatment. This would be particularly useful for consumers of psychiatric inpatient settings as these individuals generally experience extreme difficulties functioning effectively in the general population and typically have the largest disease burden and thus the largest number of factors that should be considered in treatment planning.

Given the current status of knowledge, research assessing the impact of any traumatic experiences, including those outside of the DSM definition of trauma, on adult executive functioning in a psychiatric inpatient population that considers trauma occurrence, frequency, type, and age of experience as well as a variety of other neurological, environmental, and individual factors is warranted. Therefore, the purpose of the current study is to extend the prior research using an additional measure of executive functioning in an inpatient psychiatric setting among a large sample of consumers with mental illness who were referred for neuropsychological evaluation.

Section II: Research Questions

As executive functioning is so complex, the current study primarily focuses on a single, more fundamental skill involving basic components of executive functioning – visual reconstruction. Stiles and Tada (1996) suggest that children ages three to five years are capable of analyzing spatial forms, though they so do differently than adults, such that they are able to copy a figure accurately overall, but have more difficulty with the details (Tada & Stiles-Davis, 1989). For example, Akshoomoff and Stiles' 1995 study of copy drawings from the Rey-Osterrieth Complex Figure Test (RCFT) suggests that by age six, children are able to accurately include most aspects of the figure, and by age nine they are able to accurately include nearly all details. Furthermore, children ages five to eight are able to distinguish specific details but are unable to organize them to properly copy the RCFT figure, while children age eight and older demonstrate far more advanced visuomotor organization (Karapetsas & Kantas, 1991). In addition, children can generally accurately reproduce the entire RCFT figure by age nine (Waber & Holmes, 1985).

In populations of consumers of mental health services that have a serious mental illness, there are a number of difficulties impacting the interpretation of results of neuropsychological assessment. In addition to current diagnoses, multiple neurocognitive and environmental risk factors may also contribute to the manifestation of deficits exhibited in testing. Determination of the etiology of these impairments is critical to proper treatment and clarification of diagnoses, yet extremely difficult given the wide variety of potential factors that may contribute. For instance, a consumer diagnosed with schizophrenia may demonstrate executive functioning deficits unrelated to this diagnosis, such as those arising from complications of prior head injury, substance abuse, developmental or learning disorders, or low educational level. As a result, much of the prior research has focused on “cleaner,” less impaired populations, such as outpatients with a higher level of education, no history of substance abuse or head injury, and average IQ levels (Heim et al., 2000; Nikulina & Widom, 2013; Swanson, Gur, Bilker, Petty, & Gur, 1998). However, the exclusion of such factors results in a worrisome gap in our understanding of the true etiology of deficits demonstrated by the vast majority of consumers with mental illness.

For example, though research has indicated that consumers of public psychiatric hospitals are less likely to have a high school education and more likely to have a learning disability (Fuller et al., 2002), these individuals are often excluded from samples examining the etiology of neuropsychological deficits. However, prior research has indicated that premorbid deficits in early childhood prior to disease onset may explain educational under-achievement in consumers diagnosed with mental illness (Johnstone et al., 2000; Swanson et al., 1998). Similarly, low intellectual ability, educational level, and/or IQ are risk factors for schizophrenia and other psychoses, learning disabilities, and overall psychopathology (Bora, Yucel, & Pantelis, 2009a, 2009b; Brewer et al., 2005; David, Malmberg, Brandt, Allebeck, & Lewis, 1997; Fillenbaum, Hughes, Heyman, George, & Blazer, 1988; Kolb & Wishaw, 2009; Leeson et al., 2010). In addition, the relationship between low IQ and psychopathology may differ depending on specific diagnosis and the type of IQ being assessed, such as overall, performance, or verbal IQ (Kolb & Wishaw, 2009).

In addition, individuals with a history of substance abuse are often excluded from such research, though prior research has indicated that mentally ill patients are three to six times more likely to have a substance abuse problem than the general population (Regier et al., 1990). Similarly, consumers with co-morbid substance abuse and schizophrenia often show greater number of neurocognitive deficits (Serper et al., 2000) compared to consumers with schizophrenia but no history of substance abuse.

Much of prior research has also excluded consumers with head injuries, though many previous studies have supported an association between serious mental illness and head injury. For example, patients with schizophrenic-spectrum disorder have a higher prevalence of head injury than non-psychotic patients (Wilcox & Nasrallah, 1987), and brain injuries may increase the development of the disease in genetically susceptible individuals (Koponen et al., 2002; van Reekum, Cohen, & Wong, 2000). Furthermore, research has supported an association between experiencing a traumatic brain injury and subsequently being diagnosed with a mental illness (Deb, Lyons, Koutzoukis, Ali, & McCarthy, 1999; Fann et al., 2004; Flashman, McAllister, & Ferrell R., 2012; Whelan-Goodinson, Ponsford, Johnston, & Grant, 2009).

Basic demographic characteristics are also often overlooked in prior research. However, factors such as biological sex have been found to greatly impact the effects of other factors on functionality (Lehman, Rachuba, & Postrado, 1995; Qin, Agerbo, & Mortensen, 2003), and prior research suggests significant differences in type of diagnosis, likelihood of hospitalization, and quality of life based on race and age (Fillenbaum et al., 1988; Lehman et al., 1995; Strakowski et al., 1994, 1996). Furthermore, research suggests a potential skew in the distribution of age in public mental hospitals, with 30% of inpatients aged 65 or older with only 2% of outpatients in this age group, suggesting that age may be an important covariate to consider particularly in psychiatric inpatient settings (Kramer, Taube, & Starr, 1968). Similarly, factors specific to psychiatric populations such as age of illness onset and duration of illness have been indicated as being strong predictors of subsequent functionality (Kramer et al., 1968) and behaviors such as suicide attempts (Qin et al., 2003).

In addition, few prior studies assess the impact of diagnosis on current functioning, with even fewer studies examining the specific impact of psychosis-related diagnoses. As the majority of prior research excludes individuals with serious mental illness, the impact of these diagnoses and the related symptomology are frequently not assessed even though these factors may have dramatic impacts on presentation and performance. In addition, of those studies that have attempted to assess the impact of diagnosis, nearly all tend to differentiate between major diagnostic categories, such as the impact of Schizophrenia-Spectrum Disorders compared to Major Affective Disorders.

However, prior research has supported the presence of a general cognitive deficit across psychotic disorders including Schizophrenia, Schizoaffective, Schizophreniform, Bipolar, Psychotic, and Major Depressive with psychotic features disorder (Bora et al., 2009b; Hill et al., 2009; Park et al., 2004; Reilly & Sweeny, 2014; Salvatore et al., 2009). Furthermore, results of prior research indicate that individuals with a psychotic disorder such as Schizophrenia or Bipolar I may exhibit more severe and long-term deficits in neuropsychological functioning compared to individuals without a psychiatric diagnosis or individuals with a non-psychotic affective disorder, such as Bipolar II and Major Depression (Blumenfeld, 2010; Gruber, Rathgeber, Bräunig, & Gauggel, 2007; Johnson-Selfridge & Zalewski, 2001; Strakowski et al., 1996).

Results of previous research also support a linkage between negative life events (such as adverse environments and periods of isolation) and the subsequent development of psychotic symptoms manifesting both cognitively and emotionally (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). In particular, positive psychotic symptoms such as hallucinations and delusions are the most common symptoms leading to a psychotic diagnosis and are perhaps the most prominent at onset (Garety et al., 2001; Strakowski et al., 1996), and negative symptoms such as flat affect and a lack of motivation may contribute to more severe cognitive impairment (Bora et al., 2009a).

Presence of secondary gain is rarely assessed as well, though research has indicated that secondary gain may impact performance on executive functioning measures even for individuals that have passed a symptom or performance validity test (Marcopulos, Caillouet, Bailey, Tussey, Kent, & Frederick, 2014). The term "secondary gain" refers to the presence of external motivation for the consumer to perform below his or her true ability level on neuropsychological assessments. For instance, clients in forensic cases may receive a shorter or suspended sentence or avoid execution if their performance suggests that they have a serious mental illness or severe cognitive deficits, such that they may intentionally and systematically downwardly bias their responses. Performance or symptom validity tests are often utilized as a screening measure to attempt to detect a negative response bias on behalf of the consumer, with biased responses for individuals with secondary gain suspected as being a result of malingering (Marcopulos et al., 2014).

Failure of a performance or symptom validity test generally indicates that results of cognitive assessments are unable to be validly interpreted (Marcopulos et al., 2014) and is therefore a crucial factor to consider in the neuropsychological assessment process. Furthermore, assessment of symptom or performance validity is particularly critical for individuals with potential for secondary gain (such as a shorter criminal sentence) as these individuals may have external motivation for intentionally manipulating their performance (Marcopulos & Fujii, 2012).

Therefore, the purpose of the current study is designed with the intent to better understand the influence of specific potentially confounding factors that contribute to cognitive impairment in mentally ill populations. The primary hypotheses are as follows: (a) static factors (defined as developmental and historical factors that remain relatively stable throughout the lifespan, such as history of trauma, IQ, and education level) as a whole will more strongly predict performance on an executive functioning measure compared to dynamic factors (defined as current factors that may fluctuate throughout the lifespan and therefore differ depending on the time of assessment, such as age, duration of illness, and secondary gain) as a whole; (b) the best model for prediction of performance will include a combination of both static and dynamic factors; and (c) history of any trauma will be one of the most salient predictors of performance after controlling for the factors included in the best model (i.e., the significant predictors as determined by the initial primary analyses).

Secondary hypotheses are as follows: (a) consumers with a history of "psychological trauma" (defined as events that meet the current definition of a traumatic life experience) that occurs during any period throughout their lives will more consistently demonstrate deficits on executive functioning assessments compared to consumers without a history of psychological trauma; (b) within the consumers with histories of psychological trauma, more severe deficits will be exhibited by those who were polyvictimized (i.e., experienced multiple traumatic events) compared to those who experienced a single traumatic incident; and (c) within the consumers with histories of psychological trauma, more severe deficits will be observed in those that experienced "intentional" trauma compared to those who experienced "unintentional" trauma.

Section III: Description of Measurement and Methods

This study utilized archival data from an approximately 250-bed southeastern public state inpatient adult psychiatric hospital licensed and operated by the state government. The mission of the hospital is to provide safe and effective individualized treatment in a recovery-focused environment for consumers for whom community brief outpatient services are insufficient. The hospital primarily provides long-term treatment and symptom management to individuals in surrounding areas across the state that have a serious, persistent mental illness and/or are a danger to themselves or others.

The current sample included all consumers referred for neuropsychological evaluation who completed a RCFT copy drawing (and ideally a TMT) between 2003 and 2010, with a sample size of 183. Referral sources were members of the consumer's treatment team, primarily medical doctors and/or psychiatrists, and most commonly requested a confirmation of diagnosis, evaluation of current consumer functioning (typically for treatment or discharge planning), or clarification of neurological factors impacting presentation. This archival data study was reviewed and approved by both the hospital's and James Madison University's Institutional Review Boards (IRB).

The population includes individuals age 18 or older and represents a variety of consumer ages, ethnicities, diagnoses, duration of illnesses, age of illness onsets, current symptoms, education levels, learning/developmental disorders, IQs, trauma histories, substance abuse histories, previous brain injuries, and secondary gain categories. However, it is important to note that the majority of consumers have been diagnosed with a schizophrenia-spectrum disorder, and as the sample was drawn from a public inpatient hospital, many of the consumers come from a background of low socio-economic status, including fewer resources such as housing, employment, education and support, as well as frequently lower IQ. In addition, though admission can be either voluntary or involuntary, the majority of the consumers are admitted involuntarily (i.e., court-ordered).

The current study utilized a subset of a database maintained by the hospital which includes all demographic, intake, and test data from all consumers seen in the Neuropsychology Laboratory since its initiation in 1979. This database has previously been utilized for multiple publications and scholarly presentations. However, one of the major, though unfortunately unavoidable, limitations of the database and therefore the current study relates to the restriction of data to those which were collected upon intake with the exception of neuropsychological testing sessions. This is particularly problematic in regards to trauma data. Though basic information is collected regarding prior traumatic experiences (e.g., past experiences such that the consumer should not be restrained), a formal measure relating to trauma history is not administered. Furthermore, PTSD is dramatically under-diagnosed in populations of consumers of community mental health services (Brady, Rierdan, Penk, Losardo, & Meschede, 2003; Cusack et al., 2006; Zimmerman & Mattia, 1999) and in psychiatric inpatient settings (Dansky, Roitzsch, Brady, & Saladin, 1997; Escalona et al., 1997; van Zyl, Oosthuizen, & Seedat, 2008), restricting the data to those which were self-reported by the consumer upon intake.

Though self-report is subjective by definition, it is the nature of trauma research. Opportunities to collect both pre- and post-event quantitative, objective data are nearly nonexistent, such that the majority of current trauma literature relies on self-reported data. However, a number of studies examining the psychometric properties of utilizing self-report measures of trauma in psychiatric populations have found self-report to be generally reliable and valid overall (Goodman et al., 1999; McHugo et al., 2005; Mueser et al., 2001). Furthermore, as previously mentioned, prior research has indicated that trauma and PTSD are dramatically under-documented in the public mental health system, suggesting that self-report may be far more comprehensive and accurate than medical records.

Trauma information is available in an open-text field in the hospital database which includes any self-reported head injuries or trauma that the consumer noted upon intake or during a neuropsychological evaluation interview. This information was coded using a modified version of the Life Events Checklist (LEC), as displayed in **Appendix A**. A single rater (the author) reviewed the entries and coded as appropriate to collect data where available regarding the occurrence of trauma, the type of trauma, and the frequency with which it occurred.

Each traumatic experience within the database was categorized individually, such that a single consumer could have multiple traumatic experiences. Missing or unclear information and information regarding chronic medical conditions were not coded as traumatic experiences to avoid making inaccurate inferences. Consumers were considered as having been polyvictimized if the data indicated more than one traumatic event excluding suicide attempts.

Suicide attempts were excluded from this calculation as research suggests that these experiences may differ in their etiology and impact on functioning compared to other traumatic experiences. In particular, suicidality often develops post-trauma (Ferrada-Noli, Asberg, Ormstad, Lundin, & Sundbom, 1998; Pfeffer et al., 1997; Simpson & Tate, 2005; Teasdale & Engberg, 2001) and may be most impacted by factors such as global functionality (Tejedor, Diaz, Castillon, & Pericay, 1999). In addition, while the experience of most traumatic events is typically primarily environmental, research suggests that factors such as age, biological sex, genetics, mental illness, head injury, and trauma history may differentially impact the likelihood of developing suicidal ideations (Christiansen & Frank Jensen, 2007; Dube et al., 2001; Ferrada-Noli et al., 1998; Nordström, Samuelsson, & Åsberg, 1995; Perroud et al., 2008; Pfeffer et al., 1997; Roy, Gorodetsky, Yuan, Goldman, & Enoch, 2010; Roy, Hu, Janal, & Goldman, 2007; Sarchiapone, Carli, Cuomo, & Roy, 2007; Simpson & Tate, 2005; Teasdale & Engberg, 2001; Zoroglu et al., 2003), such that the etiology of suicidality may differ from that of other traumatic events.

Experiences considered "intentional" include suicide attempts, sexual assault, physical assault, assault with a weapon, and serious injury, harm, or death caused to someone else. Events coded as suicide attempt were overdose, self-inflicted gunshot wound, attempted hanging, attempted suicide by electricity, jumping off a bridge, and suicide attempt with method unspecified. Events coded as sexual assault were sexual abuse or assault. Events coded as physical assault were being struck by a son-in-law, fighting, domestic violence, physical abuse, child abuse, head beat against chair by husband, unspecified assault, head injuries occurring in prison, and being "hit on the head" with the method unspecified. Events coded as assault with a weapon were being kicked in the head with a steel-toed boot, hit with a frying pan, hammer, bat, or lead pipe, pistol-whipped, and gunshot wound that was not indicated as self-inflicted. Events coded as serious injury, harm, or death caused to someone else were assaulting a teacher and attacking a woman.

Experiences considered "unintentional" include combat or exposure to war zone, transportation accident, serious accident at work, home, or during a recreational activity, exposure to a toxic substance, life-threatening illness or injury, sudden, violent death, sudden, unexpected death of someone close, and "other" events. Events coded as combat of exposure to war zone were a tour in Vietnam and active duty in the Army. Events coded as transportation accident were unspecified motor vehicle accident, motorcycle accident, car accident, bike accident, go-kart accident, and being struck by a vehicle (e.g., hit by car, struck by car on bicycle, struck by truck while walking). Events coded as serious accident at work, home, or during a recreational activity were sport injury (e.g., head injury from wrestling, baseball, football), having passed out or fell, and construction accident. Events coded as exposure to toxic substance were solvent injury and carbon monoxide poisoning. Events coded as life-threatening illness or injury were cardiac arrest or heart attack, stroke, struck by lightning, and astrocytoma. Events coded as sudden, violent death were suicide (e.g., daughter, father). Events coded as sudden, unexpected death of someone close were death of mother, father, husband, and grandmother not indicated as suicide. Events coded as "other" were incarceration, having left home in child or adolescence (e.g., has not resided at home since mid-teens, was in state homes during high school, ran away at age 14), loss of job, home, and children, caring for husband who suffered brain damage, divorce, and a series of setbacks and losses not specified. Note that though many of these experiences may very well meet the criteria for "psychological trauma" as defined in the current study, they were coded as unintentional in order to maintain the conservative coding and to attempt to avoid making inaccurate inferences (e.g., though combat exposure is generally traumatic, the events coded in this category were vague such that it was unclear if the consumers experienced combat or were simply military members).

This study utilized the RCFT (**Appendix B**) to assess visual reconstruction and as a measure of executive functioning to assist in assessing the impact of both static and dynamic factors on current functioning. This instrument was originally designed to assess visuospatial constructional ability and visuospatial memory in people with brain injuries and is intended to be administered with strict adherence to the procedures detailed in the manual. The test consists of a single figure encompassing three separate drawing trials: a copy trial, in which the participant is asked to directly copy the figure; a 3-minute immediate recall trial, in which the participant is asked to recall and draw the figure 3 minutes after the copy trial; and a 30-minute delayed recall trial, in which the participant is asked to recall and draw the figure 30 minutes after the original trial. The copy portion of the task assesses fundamental motor skills and executive functioning abilities, particularly planning and organization. For the purposes of this study, only the copy trial was analyzed in order to assess more basic aspects of executive functioning without the confound of memory effects.

The primary normative sample for the RCFT consisted of 601 participants aged 18-89 years who had been screened for learning disability, substance abuse, psychiatric disorders, and depression (Roman, 1996), and 505 who were ages 6-17. The sample included all geographic regions of the U.S. and Canada, including both urban and rural areas, and a subset of the sample was selected to match the current census projections, allowing scores to be compared to both age-corrected scores and the general population. No effects were found based on education or gender, though age had a significant influence on scores.

In the current study, the Boston Qualitative Scoring System (BQSS) was utilized in the coding of all figures. The BQSS attempts to score the drawings more qualitatively to assess the procedural aspects of the drawing production as compared to the quantitative 36-item scoring system included with the RCFT (Stern et al., 1999), allowing specific aspects of executive functioning to be assessed. Specifically, prior to the development of the BQSS there were no scoring systems "created predominantly for adults that provide both a comprehensive set of qualitative rating and important quantitative summary scores. For these reasons, the Boston Qualitative Scoring System for the [RCFT] was developed" (Stern et al., 1999). The BQSS's comprehensive scoring method assesses each drawing for the following: configural presence and accuracy; cluster presence, accuracy and placement; detail presence and placement; fragmentation; planning; neatness; vertical and horizontal expansion; reduction; rotation; perseveration; confabulation; and asymmetry.

The organization of the figure and individual elements, the spatial relationship of the figure on the page, and the order in which the elements were drawn are assessed to create a planning score. This planning score is then combined with the total number of fragmentations of the main elements of the figure to create an overall organization score thought to be reflective of executive functioning deficits.

The configural, cluster, and detail elements are displayed in **Appendix B**. Each element was initially assessed for presence or absence, and accuracy, placement, and fragmentation were subsequently assessed for present elements. Accuracy scores focused on the reproduction of the element itself, while placement scores focused on the placement of the element in relation to the overall figure. Fragmentation scores were simply based on the presence or absence of a fragmented approach to the element with the exception of configural rectangle A, which was scored for number of fragmentations (0-3+). The presence, accuracy, and placement scores are subsequently combined for an overall copy score for the entire figure, thought to be reflective of more basic motor deficits impacting visual reconstruction.

In addition, the BQSS provides specific scores related to perseveration (defined as element replication), confabulation (defined as addition of foreign elements), asymmetry (defined as a lack of detail and/or a higher degree of distortion), and neatness (defined as wavy/tremulous lines, gaps or overshoots, overdrawn lines, cross-outs or attempted corrections, and/or rounded corners). Overall spatial scores are also provided in regards to vertical and horizontal expansion, rotation, and reduction of the entire drawing on the page compared to the original figure.

This approach allows comparisons to be made across 17 different qualitative scores, and is beneficial when attempting to localize the region of the brain in which the dysfunction is occurring. For instance, low planning scores may be indicative of an overall executive functioning deficit, while low neatness scores may be more related to a motor deficit.

Overall, the scores from RCFT using the BQSS have been found to be generally a valid measure of visuospatial constructional ability and visuospatial memory across a variety of populations (Elderkin-Thompson, Boone, Kumar, & Mintz, 2004; Meyers & Meyers, 1999; Somerville, Tremont, & Stern, 2000; Stern et al., 1999). The strongest features of this test include use of a heterogeneous normative sample, generally high interrater reliability across samples, and relatively high correlations with similar measures typically used to assess visuospatial and executive functioning skills. In the present study, all figures were coded by one of three trained raters using the BQSS. Interrater reliability was assessed using a subset of 10 figures coded independently by each rater, with discordant ratings subsequently adjudicated, resulting in an overall Cronbach's Alpha and Intraclass Correlation of .860.

Similarly, prior research has found support for the validity of the both the RCFT and BQSS in past studies. For example, high correlations between items suggest that the RCFT is a strong measure of visuoconstructional ability and visuospatial memory (Meyers & Meyers, 1999; Somerville et al., 2000). Furthermore, higher specificity and sensitivity percentages compared to the 36-point system as found in Stern et al.'s (1999) study supports the use of the BQSS for the RCFT. In addition, results also indicated extremely high correlations with similar executive functioning measures (Elderkin-Thompson et al., 2004; Stern et al., 1999) and low correlations with indirect measures of executive functioning (Somerville et al., 2000).

In addition, though the RCFT is used as the primary endpoint, these data are also supplemented by results of the TMT Parts A and B when available to attempt to determine the predictive validity of multiple factors for executive functioning abilities (e.g., planning, organization, set-shifting) compared to more basic abilities (e.g., motor skills, visual reconstruction). This assessment is designed to assess psychomotor speed, visual scanning, and executive functioning, particularly set-shifting and mental flexibility. Part A (**Appendix C**) involves the participant sequencing numbers 1 through 25 as quickly as possible, while Part B (**Appendix D**) requires the participant to sequence numbers 1 through 13 while following a sequential pattern involving shifting between numbers and letters (e.g., 1-A-2-B). Both parts are scored based on time to completion and take approximately 10 minutes to administer.

The TMT has demonstrated moderate test-retest reliability for intervals of 3 weeks to 1 year (Bornstein, Baker, & Douglass, 1987; Dikmen, Heaton, Grant, & Temkin, 1999; Matarazzo, Wiens, Matarazzo, & Goldstein, 1974) which is anticipated for this type of assessment as it is expected that there may be memory effects, particularly for smaller intervals. In addition, the results of Heilbronner, Kinsella, Ong, and McGregor (1991) suggest that there is a moderate correlation between Parts A and B, which again is anticipated as each part is intended to assess the same basic abilities but differing higher-level abilities. Furthermore, the TMT has been found to correlate relatively highly with other visual search tasks (Ehrenstein, Heister, & Cohen, 1982).

Section IV: Description of Data Analysis

A variety of analyses were conducted to determine if there is a significant relationship between performance on executive functioning measures and multiple static and dynamic risk factors, as well as to determine the relative importance of each factor in prediction of performance believed to be reflective of current executive functioning. Variables were selected based upon prior theory and research as indicated and previously discussed, as well as the availability of the data within the database.

Variables considered to be “static” include developmental and historical factors that generally remain relatively stable throughout the lifetime, though they may increase in frequency. For instance, trauma history is considered “static” in that though additional events may be experienced, once a traumatic history is present it will remain present. Static variables tested included consumer race (Fillenbaum et al., 1988; Lehman et al., 1995; Strakowski et al., 1994, 1996), biological sex (Lehman et al., 1995; Qin et al., 2003), age of illness onset (Kramer et al., 1968; Qin et al., 2003), educational level, prior enrollment in special education courses, receipt of GED (Fillenbaum et al., 1988; Fuller et al., 2002; Johnstone et al., 2000; Swanson et al., 1998), IQ (Bora et al., 2009a, 2009b; Brewer et al., 2005; David, et al., 1997; Leeson et al., 2010), history of head injury or any trauma (Deb et al., 1999; Fann et al., 2004; Flashman et al., 2012; Whelan-Goodinson et al., 2009; Wilcox & Nasrallah, 1987), and psychological trauma variables where available, including presence or absence of a trauma history independent of brain injury (Arnow, 2004; Nemeroff et al., 2003; Schoedl et al., 2010; Steel et al., 2002; Wingenfeld et al., 2011), traumatic event type (DiScala et al., 2000; Hamel et al., 2002), and number of occurrences (Centers for Disease Control and Prevention, 2014; Edwards et al., 2005; Steel et al., 2002; Substance Abuse and Mental Health Services Administration, 2011). For the purposes of the current study, IQ is defined as intelligence level reflective of cognitive ability as assessed by the Wechsler Adult Intelligence Scale (WAIS).

Dynamic variables were obtained from the time point at which the consumer was admitted to the hospital and assessed, and include consumer age, duration of illness (Fillenbaum et al., 1988; Kramer et al., 1968; Qin et al., 2003), presence of secondary gain (defined as pending legal charges and/or application for or receipt of disability funds; Marcopulos & Fujii, 2012; Marcopulos et al., 2014), positive and negative symptoms of psychosis (Bora et al., 2009a; Garety et al., 2001; Strakowski et al., 1996), and presence of absence of a psychotic diagnosis (Blumenfeld, 2010; Bora et al., 2009b; Gruber et al., 2007; Hill et al., 2009; Johnson-Selfridge & Zalewski, 2001; Koponen et al., 2002; Park et al., 2004; Reilly & Sweeny, 2014; Salvatore et al., 2009; Strakowski et al., 1996; van Reekum et al., 2000; Wilcox & Nasrallah, 1987). Positive symptoms of psychosis include moderate to severe delusions, hallucinations, or thought derailment upon intake and/or as measured by the Inpatient Psychiatric Outcomes Scale (IPOS). Negative symptoms of psychosis include restricted/blunted/flat affect, poverty of speech with lack of spontaneity, and diminished social drive with active or passive social withdrawal as measured by the IPOS. Missing data was coded as an absence of symptomology.

Diagnoses considered related to psychosis include Bipolar I (with the exception of those specified as occurring without psychotic features), Major Depressive Disorder with psychotic features, Schizophrenia (including Schizoaffective and Schizophreniform Disorder), and Psychotic Disorders. In addition, one consumer with a diagnosis of unspecified Bipolar was also categorized as having a psychotic diagnosis as this consumer exhibited symptoms of psychosis during the testing session. Consumers without these diagnoses were categorized as having a non-psychotic diagnosis, including consumers with a diagnosis of delusional disorder or psychosis due to substance abuse or medical condition.

As previously discussed, prior research suggests that a wide range of neurological, environmental, and individual factors can contribute to current functioning. As such, all of the above static and dynamic variables are expected to impact executive functioning performance. However, given the prior research related to trauma history and subsequent functioning, the current study hypothesizes that trauma is an important predictor of performance on executive functioning measures and as such differentially impacts performance compared to other static and dynamic factors. Therefore, the current analyses attempted to determine the predictive validity of trauma after controlling for the effects of the remaining static and dynamic variables on current executive functioning performance.

The primary analyses focused on determination of the relative contribution of each individual predictor variable as well as each group of predictor variables (static versus dynamic) to the BQSS copy score and the BQSS organization score via multiple regression. The BQSS copy score includes configural presence and accuracy, cluster presence and accuracy, and detail presence, while the BQSS organization score includes fragmentation and planning. As the former is believed to reflect visuospatial functions while the latter is thought to be reflective of more general executive functioning, these two scores were utilized in the current study to compare the predictive validity of a variety of factors for both basic and global abilities impacting executive functioning.

In particular, these analyses focused on (a) determining if static variables as a whole or dynamic variables as a whole constitute the best model for prediction of performance, (b) determining which combination of individual predictor variables constitutes the best model for prediction of performance, and (c) determining the proportion of the variability in performance that can be attributed to any trauma history after controlling for all other predictor variables.

The best prediction models for the BQSS copy and organization scores were used to assess the predictive validity of these models for the original RCFT score as described by the test manual as well as the TMT scores to determine if the BQSS models similarly predict RCFT and TMT performance compared to BQSS performance. The purpose of these analyses were to determine if the factors impacting BQSS performance differed from those impacting RCFT performance which would support the use of the BQSS in practice, as well as to determine if these factors also impact TMT performance which would suggest that they may reflect more general cognitive functioning assessed across measures. In other words, as both the RCFT and TMT are believed to assess executive functioning as well as other more basic aspects of cognitive functioning, analyses were conducted to confirm if the factors included in the best model for each score differed, suggesting that they are reflective of differing basic abilities, or were similar, suggesting that they are reflective of similar executive functioning abilities. In addition, multiple regressions were conducted to determine the best model for each score, as well as the impact of psychotic diagnosis on performance.

Secondary analyses were conducted via multiple regression to assess the impact of a history of psychological trauma, polyvictimization, and type of trauma after controlling for the significant predictor variables in the best models for the BQSS scores as determined by the primary analyses.

All statistics were calculated using IBM SPSS Statistics for Windows version 21.0 (IBM Corp, 2012) with the exception of confidence intervals for R^2 which were calculated using the R2 program version 1.1 (Steiger & Fouladi, n.d).

Section V: Results

Consumer age ranged from 18 to 64 ($\bar{x}$=37.54, *SD*=14.030), and the sample included 107 males (59.4%) and 73 females (40.6%). The majority of the sample was Caucasian (80.6%), with 95% of the sample identifying as Caucasian or African-American. Age of onset of illness ranged from age 3 to age 61 ($\bar{x}$=23.57, *SD*=12.333) and duration of illness ranged from 0 to 46 years ($\bar{x}$=13.79, *SD*=10.516). In addition, the sample also included nine individuals with a substance abuse diagnosis, five individuals with an intellectual disorder, one individual with a developmental disorder, and six individuals with a learning disability.

The majority of consumers were diagnosed with a schizophrenia-spectrum (50.6%) or mood (35.0%) disorder, with 72.2% having a psychosis-related diagnosis. However, data regarding psychotic symptoms were only available for a subset of the sample. As a result, the available data appears to indicate that psychotic symptoms were present for a minority of the sample, with 30.6% experiencing positive symptoms of psychosis and 5.0% experiencing negative symptoms of psychosis. Nevertheless, as there is such a high prevalence of psychotic diagnoses in the current sample and presence of psychotic symptoms is a criterion that must be met to receive such a diagnosis, it is logical to infer that the majority of the sample exhibited psychotic symptoms, and that the superficial appearance of a lack of psychotic symptoms in the current sample is simply an artifact of missing data regarding the specific type of symptoms (negative versus positive) as well as individuals exhibiting psychotic symptoms post-intake. While this is extremely problematic in regards to validity, post-hoc analyses utilizing the psychotic diagnosis variables were conducted to attempt to better understand the impact of psychosis on performance.

Education level ranged from 6 to 20 years ($\bar{x}$=12.15, *SD*=2.583) and IQ ranged from 56 to 123 ($\bar{x}$=86.83, *SD*=14.336). Of the entire sample, 39 individuals (21.7%) had previously enrolled in special education courses and 23 individuals (12.8%) obtained their GED. Approximately one-third of the sample (36.1%) had presence of secondary gain, the majority of which pertained to legal charges. Of the consumers with secondary gain, three were removed from the sample due to poor performance on a symptom validity test as they were suspected to be malingering such that their data was believed to be invalid, with a final sample size of 180 consumers.

Referral sources were members of the consumer's treatment team, primarily medical doctors and/or psychiatrists, and most commonly requested a confirmation of diagnosis, evaluation of current consumer functioning (typically for treatment planning), or clarification of neurological factors impacting presentation.

The majority of consumers had experienced a head injury or any trauma (68.9%) and nearly half of the sample (47.8%) was coded as experiencing a psychological trauma independent of head injury. While these rates are dramatically lower than those indicated by much of prior research, it is likely that the actual frequencies of such traumatic histories are higher in the current population than the current rates appear to reflect. However, it was unfortunately not feasible to collect new data regarding traumatic experiences in the current study, restricting the data to that which was self-reported by the consumer as previously discussed.

5.1 Primary Analyses

The following predictors were assessed for the primary analyses: biological sex, race, education level (in years), enrollment in special education courses, receipt of a GED, history of head injury/trauma, age of onset of illness (in years), overall IQ, age, duration of illness (in years), presence of secondary gain, and positive and negative symptoms of psychosis. Listwise deletion was used such that consumers were excluded from analysis if they were missing data on any of the variables included in the model being tested.

Correlations among predictors and BQSS scores are displayed in **Table 1**. Though many of the predictor variables were statistically significantly correlated with one another, no predictors were removed from the analysis due to multicollinearity as there were no correlations at or above a value of .80 (Field, 2013).

5.1.1 Static factors versus dynamic factors

Factors considered to be static (*n*=153) included IQ, sex, race, education level, special education, receipt of a GED, age of onset of illness, and head injury/trauma. Factors considered to be dynamic (*n*=170) included age, duration of illness, presence of secondary gain, and negative and positive psychotic symptoms.

For the BQSS copy score, the dynamic model accounts for a large though not statistically significant percentage of score variance [R^2=.052, F(5,164)=1.802, p=.115, R^2 *95% CI:* .000 to .109], with only presence of secondary gain significantly contributing to the model (b=1.968, p=.033, sr^2=.027). These data are presented in **Table 2**. The static model accounts for a statistically significant percentage of score variance [R^2=.210, F(8,144)=4.798, p<.001, R^2 *95% CI:* .066 to .294], with only IQ significantly contributing to the model (b=0.161, p<.000, sr^2=.091). These data are presented in **Table 3**.

For the BQSS organization score, the dynamic model does not account for a significant percentage of score variance [R^2=.025, F(5,164)=0.827, p=.532, R^2 *95% CI:* .000 to .059], with none of the predictors significantly contributing to the model. These data are presented in **Table 4**. The static model accounts for a significant percentage of score variance [R^2=.143, F(8,144)=2.992, p=.004, R^2 *95% CI:* .018 to .214], with both IQ

(b=0.034, p=.003, sr^2=.053) and head injury/trauma significantly (b=0.635, p=.024, sr^2=.031) contributing to the model. These data are presented in **Table 5**.

Composite scores were calculated for each model, and the predictive utility of the dynamic model compared to the static model was assessed using the test for dependent correlations (Steiger, 1980; Steiger & Browne, 1984). For both the BQSS copy and organization scores, the static model was found to account for significantly more variance than the dynamic model, such that the static variables have stronger predictive utility with respect to both BQSS copy score (Z=-2.297, p<.05) and BQSS organization score (Z=-2.143, p<.05).

These results support the primary hypothesis that static variables as a whole are more predictive of performance compared to dynamic variables as a whole.

5.1.2 Best models, impact of any trauma

In order to determine which specific variables were the strongest predictors, potential two-way interactions and group differences were examined. Chi-squares were calculated for 27 pairs of categorical variables to assess group differences. None of these comparisons were statistically significant.

A total of 65 potential two-way interactions with the continuous predictors were assessed for both the BQSS copy and organization scores to ensure that any significant interactions were tested in the final models per statistical best practices. The five continuous variables (age, duration of illness, education, IQ, and age of onset of illness) were centered. Binary categorical variables were re-coded to values of zero and one. Categorical variables with multiple levels were effect coded.

Though six statistically significant interactions were detected, after examination of the significance of the full models including the interactions as well as the effect sizes and related confidence intervals, only one of these interactions was deemed potentially practically and statistically significant. Furthermore, this interaction was related to negative symptoms of psychosis and IQ (b=0.113, p=0.049, sr^2=.023), such that these results and the small effect size are likely artifacts of the extremely limited variability of the negative symptom variable as previously discussed. These interactions are detailed in **Appendix E1**.

5.1.2.1 Significant predictors (n=153)

For the BQSS copy score, the best model for prediction of performance consists of presence of secondary gain and IQ as displayed in **Table 6** [R^2=.225, F(2,150)=21.737, p<.001, R^2 *95% CI:* .111 to .340]. The predictive validity of the full model including all variables was not significantly stronger than that of the reduced model including only secondary gain and IQ [$R^2\Delta$=.036, $F\Delta$(11,139)=0.622, p=.807].

For the BQSS organization score, the best model for prediction of performance consists of IQ and head injury/trauma as displayed in **Table 7** [R^2=.086, F(2,150)=7.063, p=.001, R^2 *95% CI:* .015 to .179]. The predictive validity of the full model including all variables was not significantly stronger than that of the reduced model including only IQ and head injury/trauma [$R^2\Delta$=.094, $F\Delta$(11,139)=1.441, p=.161].

Information regarding the way in which these models were determined is available in **Appendix E2**.

As specific disabilities are thought to often be a confounding factor in assessment of mental illness, both the full and reduced models were also tested after excluding consumers with an intellectual disorder (*n*=5), developmental disorder (*n*=1), or learning disability (*n*=6). The reduced models remained significant predictors compared to the full models for these limited samples, suggesting that these disabilities are not confounding factors in prediction of BQSS copy or organization score when controlling for IQ and secondary gain or head injury/trauma, respectively.

Taken together, results indicate that a combination of both static and dynamic variables has the strongest predictive validity for BQSS copy score, with the best model consisting of presence of secondary gain and IQ. However, results suggest that a combination of only static factors has the strongest predictive validity for BQSS organization score, with the best model consisting of IQ and head injury/trauma.

These results partially support the primary hypothesis that a combination of both static and dynamic variables represents the best model for prediction of performance, as well as the primary hypothesis that history of any trauma is a significant predictor of performance after controlling for the other predictor variables.

5.2 Supplementary Analyses

5.2.1 RCFT and TMT model comparison

The best model for prediction of both RCFT [R^2=.291, $F(2,150)=30.724$, $p<.000$, R^2 *95% CI:* .167 to .408] and TMT Part B [R^2=.224, $F(2,144)=20.778$, $p<.000$, R^2 *95% CI:* .108 to .342] performance is a combination of IQ and presence of secondary gain as displayed in **Tables 8 and 9**, respectively. However, the best model for prediction of TMT Part A performance simply includes IQ [R^2=.098, $F(1,148)=16.094$, $p<.000$, R^2 *95% CI:* .026 to .202] as displayed in **Table 10**. Information regarding the way in which these models were determined is available in **Appendix E3**.

Though IQ was found to be the strongest predictor in all models tested, the practical significance of these results must be taken into account. IQ accounts for less than approximately 10% of the variance in BQSS organization (sr^2=.071) and TMT Part A (sr^2=.017) score. However, the effect of IQ is much larger for BQSS copy (sr^2=.189), RCFT (sr^2=.237), and TMT Part B (sr^2=.200) scores. Additional analyses comparing the predictive validity of Performance IQ and Full IQ are available in **Appendix E4**.

In addition, independent sample t-tests were conducted to assess if performance on any of the previously tested scores differed based on presence of secondary gain or a history of head injury/trauma. Both the BQSS copy (t=-2.540, p=.012, d=.375, $\bar{x}\Delta$ *95% CI:* -3.651 to -0.458) and RCFT (t=-2.568, p=.011, d=.382, $\bar{x}\Delta$ *95% CI:* -4.372 to -0.571) scores significantly differed based on presence of secondary gain, while there were no statistically significant differences in BQSS organization or any TMT score based on presence of secondary gain. These results are presented in **Table 11**. There were no statistically significant differences in any score based on history of head injury/trauma. These results are presented in **Table 12**.

Taken together, these results suggest that not only may IQ be a significant covariate for executive functioning assessments as a whole, but that these assessments may be differentially impacted by factors such as presence of secondary gain and occurrence of a head injury or trauma.

5.2.2 Psychotic diagnosis

As the literature supports psychosis being an important clinical variable often associated with more severe cognitive impairment, additional analyses were conducted to assess the predictive validity of IQ, secondary gain, and head injury/trauma for RCFT score for those with a psychotic (*n*=113) diagnosis compared to those with a non-psychotic (*n*=40) diagnosis (coded as previously described).

For individuals without a psychotic diagnosis, the best model for prediction of the BQSS copy score includes IQ, secondary gain, and negative symptoms of psychosis [R^2=.236, $F(3,36)=3.713$, p=.020, R^2 *95% CI:* .005 to .440]. These results are displayed in **Table 13**. For consumers with a psychotic diagnosis, the best model for prediction of the BQSS copy score simply includes IQ [R^2=.253, $F(1,111)=37.587$, p<.001, R^2 *95% CI:* .122 to .394]. These results are displayed in **Table 14**.

For individuals without a psychotic diagnosis, none of the models tested accounted for a significant percentage of variance in BQSS organization score, with the strongest model including only GED [R^2=.058, $F(1,38)=2.332$, p=.135, R^2 *95% CI:* .000 to .259]. These results are displayed in **Table 15**. For individuals with a psychotic diagnosis, the best model for prediction of the BQSS organization score includes only IQ and head injury/trauma [R^2=.103, $F(2,110)=6.305$, p=.003, R^2 *95% CI:* .015 to .218]. These results are displayed in **Table 16**.

Information regarding the way in which these models were determined is available in **Appendix E5**.

5.3 Secondary Analyses

Additional analyses were conducted to assess the specific predictive validity of a history of psychological trauma for BQSS copy and organization performance after controlling for the significant predictors as determined by the prior analyses. There were no statistically significant differences in the presence of secondary gain (χ^2=0.108, p=.743, R^2=.001), negative symptoms of psychosis (χ^2=1.355, p=.244, R^2=.008), or IQ (*t*=-0.613, *p*=.541, *d*=.096, *x̄Δ 95% CI:* -5.770 to 3.037) based on the presence or absence of a history of psychological trauma alone.

5.3.1 Impact of psychological trauma

An independent samples T-test was conducted to assess the potential impact of psychological trauma on the relationship between psychotic diagnosis and BQSS copy and organization scores. These results are presented in **Table 17**.

For consumers with a history of psychological trauma, the BQSS organization score significantly differed based on presence or absence of a psychotic diagnosis (*t*=-2.149, *p*=.035, *d*=.437, *x̄Δ 95% CI:* -1.222 to -0.047). There was no statistically significant difference in BQSS organization score for consumers without a history of psychological trauma based on psychotic diagnosis, nor were there differences in BQSS copy score for consumers with or without a history of trauma based on psychotic diagnosis.

Furthermore, a chi-square analysis was conducted to verify if there are significant group differences based on psychosis diagnosis and history of psychological trauma alone. This analysis was not significant (χ^2=0.137, *p*=.711, R^2=.001), indicating that there is likely no direct causal relationship between history of psychological trauma and psychotic diagnosis.

As such, the results of the t-test suggest that the relationship between performance and history of psychological trauma may be mediated by the presence or absence of a psychotic diagnosis depending on the assessment and type of score, partially supporting the secondary hypothesis that psychological trauma differentially impacts executive functioning compared to the other static and dynamic variables assessed in the prior analyses.

In addition, multiple regressions were conducted to assess the predictive validity of polyvictimization and type of trauma on BQSS copy and organization score after controlling for the previously significant predictors in a subset of the sample including only those consumers with a history of psychological trauma (coded as previously described, n=86). Of this subset, 23 individuals had a non-psychotic diagnosis and 58 individuals had a psychotic diagnosis.

5.3.2 Impact of polyvictimization

For the BQSS copy score, the best model for consumers without a psychotic diagnosis includes only secondary gain [R^2=.207, $F(1,21)$=5.469, p=.029, R^2 *95% CI:* .000 to .520]. Polyvictimization did not account for a significant proportion of variance in BQSS copy score after controlling for secondary gain [$R^2\Delta$=.045, $F\Delta(1,20)$=1.208, p=.285], nor did the addition of IQ and negative symptoms of psychosis [$R^2\Delta$=.115, $F\Delta(2,18)$=1.636, p=.222]. This data is presented in **Table 18**.

For consumers with a psychotic diagnosis, the best model for prediction of BQSS copy score includes simply IQ [R^2=.186, $F(1,56)$=12.760, p=.001, R^2 *95% CI:* .037 to .381]. Polyvictimization did not account for a significant proportion of variance in BQSS copy score after controlling for IQ [$R^2\Delta$=.013, $F\Delta(1,55)$=0.881, p=.352]. This data is presented in **Table 19**.

For the BQSS organization score, as none of the previously tested models were statistically significant for consumers without a psychotic diagnosis, the model included in the current analysis includes GED (the strongest predictor for this group) as well as IQ and head/injury trauma (the best model for the entire sample for prediction of BQSS organization score). For consumers without a psychotic diagnosis, the best model for prediction of BQSS organization score simply includes IQ [R^2=.241, F(1,21)=6.662, p=.017, R^2 *95% CI:* .007 to .551]. Polyvictimization did not account for a significant proportion of variance in BQSS organization score after controlling for IQ [$R^2\Delta$=.013, $F\Delta$(1,20)=0.340, p=.566], nor did the addition of GED and head injury/trauma [$R^2\Delta$=.104, $F\Delta$(2,18)=1.462, p=.258]. This data is presented in **Table 20**.

For consumers with a psychotic diagnosis, the strongest though not significant model of those tested for prediction of BQSS organization score simply includes IQ [R^2=.018, F(1,56)=1.055, p=.309, R^2 *95% CI:* .000 to .142]. Polyvictimization did not account for a significant proportion of variance in BQSS organization score after controlling for IQ [$R^2\Delta$=.001, $F\Delta$(1,55)=0.083, p=.775], nor did the addition of head injury/trauma [$R^2\Delta$=.002, $F\Delta$(1,54)=0.102, p=.751]. This data is presented in **Table 21**.

These results are contrary to the secondary hypothesis that polyvictimization differentially impacts performance compared to the other predictor variables.

5.3.3 Impact of type of trauma (intentional/unintentional)

For the BQSS copy score, the best model for consumers without a psychotic diagnosis includes occurrence of intentional trauma in addition to secondary gain [R^2=.373, F(2,20)=5.946, p=.009, R^2 *95% CI:* .032 to .638]. This model accounts for a significantly larger percentage of variance in BQSS copy score compared to a model including secondary gain alone [$R^2\Delta$=.166, $F\Delta$(1,20)=5.302, p=.032]. In addition, unintentional trauma (b=-4.342, p=.205, sr^2=.051), IQ (b=-0.034, p=.827, sr^2=.001) and negative symptoms of psychosis (b=-13.231, p=.095, sr^2=.092) did not account for a significant proportion of variance in BQSS copy score after controlling for secondary gain and intentional trauma. These data are presented in **Table 22**.

For consumers with a psychotic diagnosis, the best model for prediction of BQSS copy score includes occurrence of intentional trauma in addition to IQ [R^2=.288, F(2,55)=11.112, p<.001, R^2 *95% CI:* .089 to .473]. This model accounts for a significantly larger percentage of variance in BQSS copy score compared to a model including IQ alone [$R^2\Delta$=.102, $F\Delta$(1,55)=7.892, p=.007]. In addition, unintentional trauma (b=-0.500, p=.656, sr^2=.003) did not account for a significant proportion of variance in BQSS copy score after controlling for IQ and intentional trauma. These data are presented in **Table 23**.

For the BQSS organization score, the best model for consumers without a psychotic diagnosis simply includes IQ [R^2=.241, F(1,21)=6.662, p=.017, R^2 *95% CI:* .007 to .551]. Intentional trauma did not account for a significant proportion of variance in BQSS organization score after controlling for IQ [$R^2\Delta$=.024, $F\Delta$(1,20)=0.659, p=.426], nor did the addition of unintentional trauma, GED, and head injury/trauma [$R^2\Delta$=.161, $F\Delta$(3,17)=1.588, p=.229]. This data is presented in **Table 24**.

For consumers with a psychotic diagnosis, the strongest though not significant model of those tested for prediction of BQSS organization score simply includes IQ [R^2=.018, F(1,56)=1.055, p=.309, R^2 *95% CI:* .000 to .142]. Intentional trauma did not account for a significant proportion of variance in BQSS organization score after controlling for IQ [$R^2\Delta$=.001, $F\Delta$(1,55)=0.072, p=.789], nor did the addition of unintentional trauma and head injury/trauma [$R^2\Delta$=.022, $F\Delta$(2,53)=0.619, p=.542]. This data is presented in **Table 25**.

These results support the secondary hypothesis that type of trauma, particularly intentional trauma, differentially impacts performance compared to the other predictor variables.

Section VI: Discussion

The purpose of the current study was to examine the potential impact of a variety of static and dynamic factors as suggested by prior research on current executive functioning in a sample of consumers with serious mental illness. In particular, the predictive validity of factors related to a history of psychological trauma were assessed as prior research indicates a high prevalence of traumatic histories in populations of consumers with serious mental illness as well as a wide range of potentially related deficits in subsequent functionality after experiencing trauma.

Results suggest that for the current sample, the predictive validity of a number of factors for performance on executive functioning measures differs depending on the presence or absence of a history of psychological trauma as well as psychotic diagnosis. Taken together, it appears that more fundamental skills such as visual reconstruction as assessed by the BQSS copy score are differentially impacted by presence of secondary gain, a history of intentional trauma, negative symptoms of psychosis, and IQ, while more global executive functioning skills such as planning and set-shifting as assessed by the BQSS organization score are differentially impacted by IQ, a history of any head injury or trauma, and other factors not assessed in the current study.

In particular, it appears that secondary gain may impact visual reconstruction for individuals with a non-psychotic diagnosis but not those with a non-psychotic diagnosis, while a history of intentional trauma may impact performance regardless of diagnosis.

Furthermore, the effects of IQ may be diminished for individuals with a history of psychological trauma and a non-psychotic diagnosis, and the presence of negative symptoms of psychosis may only impact performance for individuals with a non-psychotic diagnosis and no history of psychological trauma.

In addition, it appears that IQ may impact organization for individuals with a non-psychotic diagnosis and history of psychological trauma as well as individuals with a psychotic diagnosis and no history of psychological trauma, while organization may be particularly impacted by factors not included in the current analyses for individuals with a non-psychotic diagnosis and no history of trauma as well as individuals with a psychotic diagnosis and no history of trauma. Furthermore, a history of any trauma or head injury may only impact performance for individuals with a psychotic diagnosis and no history of psychological trauma.

6.1 Primary Hypotheses

6.1.1 Static factors versus dynamic factors

Results indicated that for both BQSS copy and organization scores, the static variable model was a significantly stronger predictor of performance compared to the dynamic variable model. The data support the hypothesis that overall, static factors may be more predictive of performance than dynamic factors.

6.1.2 Best models, impact of any trauma

In particular, presence of secondary gain and overall IQ appear to be the strongest predictors of BQSS copy score, while a history of head injury/trauma and overall IQ appear to be the strongest predictors of BQSS organization score for the entire sample.

This partially supports the primary hypothesis that a combination of static and dynamic variables constitute the best model for prediction of performance, as a combination of static and dynamic variables appear to contribute to the best model for prediction of BQSS copy score, while only static variables appear to contribute to the best model for prediction of BQSS organization score.

In addition, this partially supports the primary hypothesis that a history of any trauma will be a strong predictor of performance after controlling for the other factors, as presence or absence of a history of any trauma or head injury appears to significantly impact BQSS organization score while not impacting BQSS copy score.

6.2 Supplementary Hypotheses

6.2.1 RCFT and TMT model comparison

Supplementary analyses suggest that the factors impacting performance on BQSS organization and TMT Part A scores may differ from those impacting performance on other measures such that these scores may represent distinct constructs, while the BQSS copy, RCFT, and TMT Part B scores may be impacted by similar factors such that they may represent a single construct. Specifically, it appears that while IQ impacts performance across measures, the impact of IQ is much larger for the BQSS copy, RCFT, and TMT Part B scores compared to the BQSS organization and TMT Part A scores. In addition, presence of secondary gain may only impact BQSS copy, RCFT, and TMT Part B scores, while a history of any head injury or trauma may only impact BQSS organization score.

6.2.2 Psychotic diagnosis

One of the most significant findings of the current study relates to the impact of a psychosis-related diagnosis, suggesting that the contribution of the predictor variables greatly differs for those with a psychotic versus non-psychotic diagnosis for both BQSS copy and organization score.

Results suggest that IQ may significantly impact BQSS copy score for consumers regardless of diagnosis, though the impact may be much larger for consumers with a psychotic diagnosis. In addition, the presence of secondary gain and negative symptoms of psychosis may differentially impact BQSS copy score for individuals without a psychotic diagnosis compared to those with a psychotic diagnosis.

Results also suggest that IQ, history of head/injury trauma, and/or receipt of a GED may differentially impact BQSS organization score for individuals without a psychotic diagnosis compared to those with a psychotic diagnosis. Furthermore, BQSS organization score may be more reflective of the influence of other constructs beyond the predictor variables tested for consumers without a psychotic disorder compared to those with a psychotic disorder.

6.3 Secondary Hypotheses

6.3.1 Impact of psychological trauma

Results suggest that the relationship between psychotic diagnosis and BQSS organization performance may be mediated by the presence or absence of a history of psychological trauma, while this history may not impact BQSS copy performance. This partially supports the secondary hypothesis that a history of psychological trauma differentially impacts performance depending on the measure and impact of other factors.

These results help to extend the findings of prior research suggesting that trauma exposure and the resulting toxic stress are among the strongest predictors of subsequent cognitive functioning and mental health (Arnow, 2004; Frodl and O'Keane, 2013; Nemeroff et al., 2003; Schoedl et al., 2010; Steel et al., 2002; Wingenfeld et al., 2011).

6.3.2 Impact of polyvictimization

Polyvictimization did not appear to significantly impact performance on either BQSS copy or organization score. This does not support the secondary hypothesis that number of traumatic experiences is a significant factor in prediction of cognitive functioning, nor does it replicate prior research suggesting that the risk for health problems in adulthood increases with the number of traumatic events experienced (Edwards et al., 2005; Steel et al., 2002). However, of the 86 consumers with a history of psychological trauma, only 21 were coded as being polyvictimized, 11 of whom had a non-psychotic diagnosis and 13 of whom had a psychotic diagnosis. As such, the lack of significance of polyvictimization in prediction of performance in the current study is likely a result of a lack of statistical power due to limited sample size and variability, as well as the conservative coding of the trauma data as later described in the limitations section.

6.3.3 Impact of type of trauma (intentional/unintentional)

One of the most exciting findings of the current study relates to the impact of type of trauma for individuals with a history of psychologically traumatic experiences. Results indicate that a history of experiencing intentional trauma is a significant predictor of BQSS copy score while not significantly impacting BQSS organization score. This supports the secondary hypothesis and reinforces prior research suggesting that the type of traumatic experience may differentially impact functioning beyond the presence of a traumatic history alone (DiScala et al., 2000; Hamel et al., 2002).

6.4 Conclusion

Taken together, the data suggest that a wide variety of factors may not only contribute to poor performance on executive functioning measures overall, but may differentially impact performance depending on other individual factors and the type of cognitive functioning being assessed (e.g., basic versus executive).

The data indicate that BQSS copy score may be significantly impacted by secondary gain for consumers with a non-psychotic diagnosis, but that these factors may not impact performance for individuals with a psychotic diagnosis. However, further research is needed to assess if these results are due to a diagnostic bias (e.g., consumers with secondary gain are less likely to receive a psychotic diagnosis such that this is a spurious relationship) or malingering (e.g., consumers without a psychotic diagnosis that have secondary gain are more likely to feign poor performance).

In addition, results suggest that the BQSS organization score may be most impacted by IQ and a history of head injury or trauma for individuals with a psychotic diagnosis, while this score may be significantly impacted by other factors not assessed in the current study for individuals without a psychotic diagnosis. However, further research is needed to assess if these results are due to a multi-level interaction among these factors (e.g., the relationship between IQ and performance is almost entirely moderated by the interaction of head injury/trauma and psychotic diagnosis), a more sequential, causal association (e.g., individuals with low IQ are more likely to then experience head injury/trauma which then makes them more likely to develop psychotic symptoms), or spurious relationships (e.g., a factor not assessed in the current study moderates the relationship between psychotic diagnosis and performance regardless of IQ and/or trauma history).

Furthermore, it appears that compared to a history of unintentional or any trauma, a history of intentional trauma may have stronger predictive validity for quantitative measures (such as the BQSS copy score) than qualitative measures (such as the BQSS organization score) of executive functioning regardless of the presence or absence of a psychotic diagnosis. However, further research is needed to determine the specific aspects of executive functioning assessed by the BQSS copy versus organization scores that may be most impacted by a history of intentional trauma (e.g., visual reconstruction versus planning).

Moreover, the data suggest that IQ may not only impact performance across measures, but that it may differentially impact performance depending on psychotic diagnosis and history of psychological trauma. However, further research is needed to assess significance of potentially multi-level interactions with IQ and other predictive variables on performance on a variety of executive functioning measures, as well as the impact of specific types of IQ such as performance or verbal IQ.

In addition, the best models for prediction of RCFT and TMT Part B scores are identical to the best model for prediction of BQSS copy score for the entire sample, suggesting that these scores may provide similar information, particularly in regards to IQ and presence of secondary gain. The best model for prediction of TMT Part A score includes IQ but not secondary gain, suggesting that this score may also provide similar information in regards to IQ but may not be impacted by the presence of secondary gain.

Furthermore, the best model for BQSS organization score for the entire sample was the only model that included a history of head injury or any trauma, suggesting that BQSS organization score may provide information relating to a history of trauma not represented by the other scores.

These results help extend those of the 2013 study by Nikulina and Widom suggesting that the factors impacting TMT Part A may differ from those impacting the TMT Part B. This also supports prior researchers' position that the relationship between low IQ and psychopathology may be mediated by a variety of other factors rather than being a direct causation (Kolb & Wishaw, 2009). Furthermore, these data suggest that the BQSS copy and RCFT scores provide similar information, while the BQSS organization score may provide additional information beyond the scope of the RCFT score, further supporting the use of the BQSS in practice (Stern et al., 1999).

The strength of these models did not appear to differ based on intellectual disability, developmental disorder, or learning disability. This differs from the results of prior research, suggesting that specific disabilities such as learning disorders and abilities such as reading skills may have strong predictive validity for subsequent functionality such as executive functioning in adulthood (Bora et al., 2009a, 2009b; Brewer et al., 2005; David et al., 1997; Kolb & Wishaw, 2009; Leeson et al., 2010; Werner, 1993). However, the lack of impact of such disabilities on functionality in the current study is almost certainly simply an artifact of the extremely limited sample size for the current study, as only 12 of the 180 consumers in the sample were diagnosed with an intellectual, developmental, or learning disorder or disability.

6.5 Strengths, Limitations, and Implications

The current study has a number of strengths contributing to its generalizability. In particular, a wide variety of static and dynamic factors suggested by prior research were assessed, including biological sex, race, education, enrollment in special education courses, receipt of a GED, history of head injury/trauma, age of onset of illness, overall IQ, age, duration of illness, presence of secondary gain, positive and negative symptoms of psychosis, presence or absence of a psychotic diagnosis, history of psychological trauma and polyvictimization, and type of trauma.

In addition, the sample included a sample more representative of the population than much of prior research (Heim et al., 2000; Nikulina & Widom, 2013; Swanson et al., 1998), including consumers with low education levels, history of substance abuse, history of head injury, and low IQ levels. This is particularly beneficial in the current sample of individuals with serious mental illness, as much of prior research indicates this population is more likely to have lower levels of education (Fuller et al., 2002), substance abuse problems (Regier et al., 1990), prior head injury (Deb et al., 1999; Fann et al., 2004; Whelan-Goodinson et al., 2009), and low IQ (Bora et al., 2009a, 2009b; Brewer et al., 2005; David et al., 1997; Leeson et al., 2010).

The assessment of the impact of secondary gain was also extremely useful for this population, as prior research as well as the current results suggest that secondary gain may impact performance on executive functioning measures even for individuals that have passed a symptom or performance validity test (Marcopulos et al., 2014). Furthermore, the administration of a performance validity test and the subsequent removal of the three participants that failed this assessment from the current sample prior to analyses strengthen the inferences that can be made from the resulting data as this greatly reduced the likelihood of detecting spurious relationships as a result of malingering.

Similarly, the differentiation between psychotic and non-psychotic diagnoses in the current study allows more direct comparisons to be made to the recent cognitive endophenotype literature suggesting that neuropsychological functioning may be more similar for individuals with psychosis-related diagnoses such as Schizophrenia and Bipolar I compared to individuals with non-psychotic diagnoses such as Bipolar II and depression without psychotic features (Blumenfeld, 2010; Bora et al., 2009b; Gruber et al., 2007; Hill et al., 2009; Johnson-Selfridge & Zalewski, 2001; Park et al., 2004; Reilly & Sweeny, 2014; Salvatore et al., 2009).

Moreover, the assessment of specific symptoms of psychosis in addition to diagnosis enabled the assessment of the interrelationships between traumatic history, psychosis-related diagnosis, both positive and negative psychotic symptoms, and current cognitive functionality. This is particularly beneficial for the current study as prior research suggests that there are significant relationships between negative life events, psychotic symptoms, and subsequent cognitive impairment (Bora et al., 2009a; Garety et al., 2001).

Similarly, two separate scores assessing different skills were utilized for a single assessment, enabling more direct comparisons to be made without additional confounding effects from multiple testing administrations (e.g., both scores were based upon a single drawing, decreasing the likelihood of confounding effects such as memory or time delays). Moreover, the comparison of these two scores to a third, more basic score for the same assessment, as well as to two additional scores for a separate assessment focusing on differing skills, contributes to the validity of the inferences made regarding the impact of factors on both basic and more global executive functioning abilities.

The current study also utilized a more broad definition of trauma than much of prior research, enabling the effects of emotional trauma to be assessed in addition to physical trauma. This is particularly critical as many of the findings of previous research have suggested that trauma histories are overwhelmingly prevalent in psychiatric populations and that the characteristics of these histories may be more predictive of subsequent functionality than the presence of the history itself.

However, a number of limitations of the current study impacting its generalizability must be recognized. One of the largest limitations is the restriction of trauma data to that which was self-reported by the consumer, as previously discussed. As PTSD is so dramatically underdiagnosed in the public mental health system, the current sample was unable to be stratified by PTSD diagnosis as a measure of the presence or absence of a traumatic history. Though this information would ideally be collected objectively, systematically, and quantitatively, the nature of the population and services restrict the data to those which were self-reported by the consumers after the trauma occurred. In addition, the sample was limited to consumers referred for neuropsychological evaluation, such that the sample is not representative of the entire hospital population.

Similarly, information regarding positive psychotic symptoms was collected at intake for a subset of the sample rather than at the same time point at which the consumer completed the RCFT and TMT, such that these symptoms may have no longer been present at the time of the neuropsychological assessment session. Moreover, psychotic symptom data was not available for all consumers, particularly in regards to the negative symptom information which was only available for a subset of consumers that completed the IPOS during their testing session.

In addition, the information utilized from the database was not collected by the current author with the exception of the BQSS and trauma coding, such that data regarding standardization of testing sessions or threats to performance validity for the specific measures assessed are not available. Furthermore, as PTSD is underdiagnosed in mental health populations and the current study uses a broad definition of trauma and archival data, inferences were made based on the available data regarding specific events to consider as traumatic as well as which events to consider intentional or unintentional. As such, though all events coded as trauma have a potential to be traumatic, they may not have been traumatic for the specific consumer in question, particularly as prior research suggests that the relationship between trauma and psychopathology is likely mediated by perceived stress (Ford & Kidd, 1998; Nikulina & Widom, 2013; Wingenfeld et al., 2011). Moreover, trauma information was only coded when explicitly specified in the database, such that the actual frequencies of psychological trauma histories, number of experiences, and types of trauma are likely significantly higher than indicated by the extremely conservative coding utilized in the current study.

An additional limitation relates to the limited variability in the current sample for a number of factors, such that the effects of such factors were unable to be accurately assessed. For example, of the entire sample of 180 consumers, only nine individuals were diagnosed with substance abuse. As a result, the impact of substance abuse on cognitive functioning was unable to be detected, though prior research suggests that a history of trauma is significantly related to subsequent substance abuse (Arnow, 2004), and co-morbid substance abuse is thought to often result in a greater number of neurocognitive deficits for individuals with serious mental illnesses such as schizophrenia (Serper et al., 2000).

Similarly, the sample included only five individuals with an intellectual disorder, one individual with a developmental disorder, and six individuals with a learning disability. Though research suggests that consumers of mental health services are more likely to have a learning disability (Fuller et al., 2002) and that the relationships between such disabilities and functionality may be mediated by IQ (Kolb & Wishaw, 2009), the limited variability of these factors in the current sample resulted in their effects being unable to be accurately detected.

In addition, though the current sample only included those individuals that passed a performance validity test, the potential impact of malingering on performance cannot be entirely eliminated as research suggests that secondary gain may impact performance even for individuals that pass such validity measures (Marcopulos et al., 2014), further limiting the inferences that can be made from the resulting data.

Additional limitations include the assessment of only two measures of executive functioning; lack of availability of data relating to other potentially predictive factors such as socioeconomic status; and analyses related to correlational rather than causational relationships.

Furthermore, one of the main limitations of psychology research as a whole and therefore the current study relates to the lack of a unified theoretical approach in the field of psychology (Henriques, 2014). As the core concepts of psychology are differentially defined across and within topic areas, the efficacy of research attempting to extend the knowledge base regarding specific phenomena is limited and convoluted.

For instance, the theoretical basis of the current study focuses on a very narrow body of research relating to the potential effects of traumatic experiences, the foundation of which tends to utilize a framework and methodology that greatly differs from other, presumably interrelated topic areas in both research and practice. Furthermore, even within the narrow confines of trauma research, the conceptual basis and methodology utilized greatly differs across studies even in the most foundational aspects, such as the definition of a traumatic life experience.

Similarly, though the current study did attempt to assess the impact of a variety of factors in addition to trauma on performance, the underlying assumption upon which the methodology was based that trauma impacts functionality results in an inherent bias in the study design and inferences made from the resulting data. Moreover, though the factors in the primary analyses were selected based on prior theory and research, the factors included in the secondary analyses were selected based on the data from the primary analyses, leading to a potential disconnect between the results of the current study and the underlying theory upon which it was based.

As a result, though there is an immense variety of existing “psychology” research, the contribution of such research to a better understanding of psychology as a field and a workable theory of the human condition is extremely lacking, as much of such research tends to focus on elaboration of a current theory such that new, potentially better theories are overlooked or ignored, or introduction of a new theory without incorporation of previous theories, resulting in unnecessary repetition of foundational concepts and a lack of cohesion across studies.

The current study has a wide range of implications for neuropsychological assessment, treatment in mentally ill populations, and future research. When assessing cognitive functioning, clinicians should consider the impact of a variety of contributing factors, including IQ and secondary gain. In addition, interpretation of results of neuropsychological assessments should consider the contribution of psychosis, history of trauma, and type of trauma to performance and specific deficits manifested during testing. In addition, clinicians should utilize an assortment of cognitive functioning assessments as performance may be differentially impacted by these factors depending on the specific abilities being assessed (e.g., basic versus executive).

Furthermore, treatment of cognitive impairment would benefit from incorporation of treatment for the impact of these factors. This would enable the treatment team to formulate a treatment plan that is in the best interests of the consumer, including addressing specific deficits as well as underlying historical, developmental, and current factors that may impact performance.

Future research is needed to assess other aspects of executive functioning as well as the impact of more systematically collected trauma history data in larger samples and across multiple settings, differentiating between psychotic and non-psychotic diagnoses and including assessment of additional factors such as perceived stress and SES.

Section VII: References

Akshoomoff, N. A., & Stiles, J. (1995). Developmental trends in visuospatial analysis and planning: I. Copying a complex figure. *Neuropsychology*, *9(*3), 364.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, text revision (DSM-IV-TR)*. American Psychiatric Association.

American Psychological Association, (2014). *Trauma.* Retrieved from http://www.apa.org/topics/trauma.

Anderson, V., Spencer-Smith, M., & Wood, A. (2011). Do children really recover better? Neurobehavioural plasticity after early brain insult. *Brain*, awr103.

Arnow, B. A. (2004). Relationships between childhood maltreatment, adult health and psychiatric outcomes, and medical utilization. *Journal of Clinical Psychiatry*, *65*, 10-15.

Becker, M. G., Isaac, W., & Hynd, G. W. (1987). Neuropsychological development of nonverbal behaviors attributed to "frontal lobe" functioning. *Developmental Neuropsychology*, *3(*3-4), 275-298.

Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development*, *81(*6), 1641-1660.

Blumenfeld, H. (2010). *Neuroanatomy through clinical cases* (2nd ed., 856-858). Sunderland, MA: Sinauer.

Bora, E., Yucel, M., & Pantelis, C. (2009a). Cognitive functioning in schizophrenia, schizoaffective disorder and affective psychoses: Meta-analytic study. *The British Journal of Psychiatry*, *195*(6), 475-482.

Bora, E., Yücel, M., & Pantelis, C. (2009b). Cognitive impairment in schizophrenia and affective psychoses: Implications for DSM-V criteria and beyond. *Schizophrenia Bulletin*, sbp094.

Bornstein, R. A., Baker, G. B., & Douglass, A. B. (1987). Short-term retest reliability of the Halstead-Reitan Battery in a normal sample. *Journal of Nervous and Mental Disease*, *175*, 229-232.

Boyden, J., & Mann, G. (2005). Children's risk, resilience, and coping in extreme situations. In Ungar, M. (Ed.), *Handbook for working with children and youth: Pathways to resilience across cultures and contexts* (3-26). Location: Sage Publications.

Brady, S., Rierdan, J., Penk, W., Losardo, M., & Meschede, T. (2003). Post-traumatic stress disorder in adults with serious mental illness and substance abuse. *Journal of Trauma and Dissociation*, *4(*4), 77-90.

Breslau, N., & Davis, G. (1987). Post-traumatic stress disorder: The stressor criterion. *Journal of Nervous and Mental Disease*, *175*, 255-264.

Brewer, W. J., Francey, S. M., Wood, S. J., Jackson, H. J., Pantelis, C., Phillips, L. J., Yung, A. R., Anderson, V. A., & McGorry, P. D. (2005). Memory impairments identified in people at ultra-high risk for psychosis who later develop first-episode psychosis. *Memory*, *162*(1).

Briere, J., & Elliott, D. M. (2003). Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. *Child Abuse and Neglect*, *27(*10), 1205-1222.

Briggs-Gowan, M. J., Ford, J. D., Fraleigh, L., McCarthy, K., & Carter, A. S. (2010). Prevalence of exposure to potentially traumatic events in a healthy birth cohort of very young children in the northeastern United States. *Journal of Traumatic Stress*, *23*, 725–733.

Broderick, P. C., & Blewitt, P. (2014). *The life span: Human development for helping professionals.* New York, NY: Pearson.

Bryan, G. K., & Riesen, A. H. (1989). Deprived somatosensory-motor experience in stumptailed monkey neocortex: Dendritic spine density and dendritic branching of layer IIIB pyramidal cells. *Journal of Comparative Neurology*, *286(*2), 208-217.

Centers for Disease Control and Prevention (2014). *Major findings.* The ACE Study. Retrieved from http://www.cdc.gov/violenceprevention/acestudy/findings.html.

Christiansen, E., & Frank Jensen, B. (2007). Risk of repetition of suicide attempt, suicide or all deaths after an episode of attempted suicide: A register-based survival analysis. *Australasian Psychiatry*, *41*(3), 257-265.

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81(*1), 252-269.

Coleman, P. D., & Riesen, A. H. (1968). Environmental effects on cortical dendritic fields: I. Rearing in the dark. *Journal of Anatomy*, *102(*Pt. 3), 363.

Copeland, W. E., Keeler, G., Angold, A., & Costello, E. J. (2007). Traumatic events and posttraumatic stress in childhood. *Archives of General Psychiatry*, *64(*5), 577-584.

Crick, F. (1982). Do dendritic spines twitch? *Trends in Neurosciences*, *5*, 44-46.

Cusack, K. J., Frueh, B. C., & Brady, K. T. (2004). Trauma history screening in a community mental health center. *Psychiatric Services*, *55(*2), 157-162.

Cusack, K. J., Grubaugh, A. L., Knapp, R. G., & Frueh, B. C. (2006). Unrecognized trauma and PTSD among public mental health consumers with chronic and severe mental illness. *Community Mental Health Journal*, *42(*5), 487-500.

Dansky, B. S., Roitzsch, J. C., Brady, K. T., & Saladin, M. E. (1997). Posttraumatic stress disorder and substance abuse: Use of research in a clinical setting. *Journal of Traumatic Stress*, *10(*1), 141-148.

David, A. S., Malmberg, A., Brandt, L., Allebeck, P., & Lewis, G. (1997). IQ and risk for schizophrenia: A population-based cohort study. *Psychological Medicine*, *27(*6), 1311-1323.

Davies-Netzley, S., Hurlburt, M. S., & Hough, R. L. (1996). Childhood abuse as a precursor to homelessness for homeless women with severe mental illness. *Violence and Victims*, *11(*2), 129-142

Deb, S., Lyons, I., Koutzoukis, C., Ali, I., & McCarthy, G. (1999). Rate of psychiatric illness 1 year after traumatic brain injury. *American Journal of Psychiatry*, *156(*3), 374-378.

Dikmen, S. S., Heaton, R. K., Grant, I., & Temkin, N. R. (1999). Test-retest reliability and practice effects of expanded Halstead-Reitan Neuropsychological Test Battery. *Journal of the International Neuropsychological Society*, *5(*4), 346-356.

DiScala, C., Sege, R., Li, G., & Reece, R. M. (2000). Child abuse and unintentional injuries: A 10-year retrospective. *Archives of Pediatrics and Adolescent Medicine*, *154(*1), 16-22.

Draijer, N., & Langeland, W. (1999). Childhood trauma and perceived parental dysfunction in the etiology of dissociative symptoms in psychiatric inpatients. *American Journal of Psychiatry, 156(*3), 379-385.

Dube, S. R., Anda, R. F., Felitti, V. J., Chapman, D. P., Williamson, D. F., & Giles, W. H. (2001). Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span: findings from the Adverse Childhood Experiences Study. *Jama*, *286*(24), 3089-3096.

Edwards, V. J., Anda, R. F., Dube, S. R., Dong, M., Chapman, D. F., & Felitti, V. J. (2005). The wide-ranging health consequences of adverse childhood experiences. *Child Victimization: Maltreatment, Bullying, and Dating Violence Prevention and Intervention, Kingston, NJ: Civic Research Institute*, 8-1.

Ehrenstein, W. H., Heister, G., & Cohen, R. (1982). Trail Making Test and visual search. *Archiv für Psychiatrie und Nervenkrankheiten*, *231*, 333-338.

Elderkin-Thompson, V., Boone, K. B., Kumar, A., & Mintz, J. (2004). Validity of the Boston Qualitative Scoring System for the Rey–Osterrieth Complex Figure among depressed elderly patients. *Journal of Clinical and Experimental Neuropsychology*, *26(*5), 598-607.

Engert, F., & Bonhoeffer, T. (1999). Dendritic spine changes associated with hippocampal long-term synaptic plasticity. *Nature*, *399(*6731), 66-70.

Escalona, R., Tupler, L. A., Saur, C. D., Krishnan, K. R. R., & Davidson, J. R. (1997). Screening for trauma history on an inpatient affective-disorders unit: A pilot study. *Journal of Traumatic Stress*, *10(*2), 299-305.

Fann, J. R., Burington, B., Leonetti, A., Jaffe, K., Katon, W. J., & Thompson, R. S. (2004). Psychiatric illness following traumatic brain injury in an adult healthmaintenance organization population. *Archives of General Psychiatry*, *61(*1), 53-61.

Ferrada-Noli, M., Asberg, M., Ormstad, K., Lundin, T., & Sundbom, E. (1998). Suicidal behavior after severe trauma. Part 1: PTSD diagnoses, psychiatric comorbidity, and assessments of suicidal behavior. *Journal of Traumatic Stress*, *11*(1), 103-112.

Field, A. (2013). *Discovering statistics using IBM SPSS statistics*. Washington D.C.: Sage.

Fillenbaum, G. G., Hughes, D. C., Heyman, A., George, L. K., & Blazer, D. G. (1988). Relationship of health and demographic characteristics to Mini-Mental State Examination score among community residents. *Psychological Medicine*, *18*(03), 719-726.

Fink, G. (Ed.). (2010). *Stress science: Neuroendocrinology*. San Diego, CA: Academic Press.

Flashman, L. A., McAllister, T., & Ferrell R. (2012). Traumatic brain injury and schizophrenia. In Marcopulos, B. A., & Kurtz, M. M. (Eds.), *Clinical and neuropsychological foundations of schizophrenia* (181-206). New York: Psychology Press.

Ford, J. D., & Kidd, P. (1998). Early childhood trauma and disorders of extreme stress as predictors of treatment outcome with chronic posttraumatic stress disorder. *Journal of Traumatic Stress*, *11(*4), 743-761.

Frodl, T., & O'Keane, V. (2013). How does the brain deal with cumulative stress? A review with focus on developmental stress, HPA axis function and hippocampal structure in humans. *Neurobiology of Disease*, *52*, 24-37.

Fuller, R., Nopoulos, P., Arndt, S., O'Leary, D., Ho, B. C., & Andreasen, N. C. (2002). Longitudinal assessment of premorbid cognitive functioning in patients with schizophrenia through examination of standardized scholastic test performance. *American Journal of Psychiatry*, *159(*7), 1183-1189.

Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine*, *31*(02), 189-195.

Goodman, L. A., Thompson, K. M., Weinfurt, K., Corl, S., Acker, P., Mueser, K. T., & Rosenberg, S. D. (1999). Reliability of reports of violent victimization and posttraumatic stress disorder among men and women with serious mental illness. *Journal of Traumatic Stress*, *12(*4), 587-599.

Greenough, W. T., & Volkmar, F. R. (1973). Pattern of dendritic branching in occipital cortex of rats reared in complex environments. *Experimental Neurology*, *40(*2), 491-504.

Gruber, S., Rathgeber, K., Bräunig, P., & Gauggel, S. (2007). Stability and course of neuropsychological deficits in manic and depressed bipolar patients compared to patients with major depression. *Journal of Affective Disorders*, *104*(1), 61-71.

Hamel, D., Pampalon, R., & Institut national de santé publique du Québec. (2002). *Trauma and deprivation in Québec*. [Québec]: Institut national de santé publique du Québec.

Harris, K. M. (1999). Structure, development, and plasticity of dendritic spines. *Current Opinion in Neurobiology*, *9(*3), 343-348.

Harvard University's Center on the Developing Child (2012). *Key concepts: Stress response*. Retrieved from http://developingchild.harvard.edu/index.php/key_concepts/toxic_stress_response.

Heilbronner, R. L., Kinsella, G. J., Ong, B., & McGregor, J., (1991). Lateralized brain damage and performance on Trail Making A and B, Digit Span Forward and Backward, and TPT memory and location. *Archives of Clinical Neuropsychology, 6*, 251-258.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *The Journal of the American Medical Association*, *284(*5), 592-597.

Henriques, G. (2014, December 13). The unified theory: A blog tour [Web log post]. Retrieved from https://www.psychologytoday.com/blog/theory-knowledge/201412/the-unified-theory-blog-tour.

Hill, S. K., Reilly, J. L., Harris, M. S., Rosen, C., Marvin, R. W., DeLeon, O., & Sweeney, J. A. (2009). A comparison of neuropsychological dysfunction in first-episode psychosis patients with unipolar depression, bipolar disorder, and schizophrenia. *Schizophrenia Research*, *113*(2), 167-175.

Johansson, B. B., & Belichenko, P. V. (2002). Neuronal plasticity and dendritic spines: Effect of environmental enrichment on intact and postischemic rat brain. *Journal of Cerebral Blood Flow and Metabolism*, *22(*1), 89-96.

Johnson-Selfridge, M., & Zalewski, C. (2001). Moderator variables of executive functioning in schizophrenia: Meta-analytic findings. *Schizophrenia Bulletin*, *27*(2), 305-316.

Johnstone, E. C., Abukmeil, S. S., Byrne, M., Clafferty, R., Grant, E., Hodges, A., Lawrie, S. M., & Owens, D. G. (2000). Edinburgh high risk study—findings after four years: Demographic, attainment and psychopathological issues. *Schizophrenia Research*, *46(*1), 1-15.

Karapetsas, A., & Kantas, A. (1991). Visuomotor organization in the child: A neuropsychological approach. *Perceptual and Motor Skills*, *72(*1), 211-217.

Kolb, B., & Whishaw, I. Q. (1998). Brain plasticity and behavior. *Annual Review of Psychology*, *49(*1), 43-64.

Kolb, B., & Whishaw, I. Q. (2009). *Fundamentals of human neuropsychology* (6th ed., 698-812). New York: Macmillan.

Koponen, S., Taiminen, T., Portin, R., Himanen, L., Isoniemi, H., Heinonen, H., Hinkka, S., & Tenovuo, O. (2002). Axis I and II psychiatric disorders after traumatic brain injury: A 30-year follow-up study. *American Journal of Psychiatry*, 159(8), 1315-1321.

Kramer, M., Taube, C., & Starr, S. (1968). Patterns of use of psychiatric facilities by the aged: Current status, trends, and implications. *Psychiatric Research Reports*, *23,* 89-150.

Leeson, V. C., Barnes, T. R., Harrison, M., Matheson, E., Harrison, I., Mutsatsa, S. H., Ron, M. A., & Joyce, E. M. (2010). The relationship between IQ, memory, executive function, and processing speed in recent-onset psychosis: 1-year stability and clinical outcome. *Schizophrenia Bulletin*, *36*(2), 400-409.

Lehman, A. F., Rachuba, L. T., & Postrado, L. T. (1995). Demographic influences on quality of life among persons with chronic mental illnesses. *Evaluation and Program Planning, 18*(2), 155-164.

Livneh, Y., Feinstein, N., Klein, M., & Mizrahi, A. (2009). Sensory input enhances synaptogenesis of adult-born neurons. *The Journal of Neuroscience*, *29(*1), 86-97.

Lupien, S. J., de Leon, M., De Santi, S., Convit, A., Tarshish, C., Nair, N. P. V., Thakur, M., McEwen, B. S., Hauger, R. L., & Meaney, M. J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience*, *1(*1), 69-73.

Magariños, A. M., McEwen, B. S., Flügge, G., & Fuchs, E. (1996). Chronic psychosocial stress causes apical dendritic atrophy of hippocampal CA3 pyramidal neurons in subordinate tree shrews. *The Journal of Neuroscience, 16(*10), 3534-3540.

Mann, G. (2002). *Family matters: The care and protection of children affected by HIV/AIDS in Malawi*. Stockholm, Sweden: Save the Children.

Marcopulos, B. A., & Fujii, D. (2012). Neuropsychological assessment of persons with schizophrenia. In Marcopulos, B. A., & Kurtz, M. M. (Eds.), *Clinical and neuropsychological foundations of schizophrenia* (55-79). New York: Psychology Press.

Marcopulos, B. A., Caillouet, B. A., Bailey, C. M., Tussey, C., Kent, J. A., & Frederick, R. (2014). Clinical decision making in response to performance validity test failure in a psychiatric setting. *The Clinical Neuropsychologist*, *28*(4), 633-652.

Matarazzo, J. D., Wiens, A. N., Matarazzo, R. G., & Goldstein, S. G. (1974). Psychometric and clinical test-retest reliability of the Halstead impairment index in a sample of healthy, young, normal men. *Journal of Nervous and Mental Disease*, *158*, 37-49.

McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology*, *22(*2), 108-124.

McEwen, B. S., & Gianaros, P. J. (2010). Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences*, *1186(*1), 190-222.

McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. *Current Opinion in Neurobiology*, *5(*2), 205-216.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, *153(*18), 2093-2101.

McHugo, G. J., Caspi, Y., Kammerer, N., Mazelis, R., Jackson, E., Russell, L., Clark, C., Liebschutz, J., & Kimerling, R. (2005). The assessment of trauma history in women with co-occurring substance abuse and mental disorders and a history of interpersonal violence. *The Journal of Behavioral Health Services and Research*, *32(*2), 113-127.

Meyers, J. E., & Meyers, K. R. (1999). *Rey complex figure test and recognition trial: Manual*. Psykologiförl.

Mueser, K. T., Goodman, L. B., Trumbetta, S. L., Rosenberg, S. D., Osher, F. C., Vidaver, R., Auciello, P., & Foy, D. W. (1998). Trauma and posttraumatic stress disorder in severe mental illness. *Journal of Consulting and Clinical Psychology*,*66(*3), 493.

Mueser, K. T., Rosenberg, S. D., Fox, L., Salyers, M. P., Ford, J. D., & Carty, P. (2001). Psychometric evaluation of trauma and posttraumatic stress disorder assessments in persons with severe mental illness. *Psychological Assessment*, *13(*1), 110.

Nemeroff, C. B., Heim, C. M., Thase, M. E., Klein, D. N., Rush, A. J., Schatzberg, A. F., Ninan, P. T., McCullough, J. P., Jr., Weiss, P. M., Dunner, D. L., Rothbaum, B. O., Kornstein, S., Keitner, G., & Keller, M. B. (2003). Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proceedings of the National Academy of Sciences*, *100(*24), 14293-14296.

Nikulina, V., & Widom, C. S. (2013). Child maltreatment and executive functioning in middle adulthood: A prospective examination. *Neuropsychology*, *27(*4), 417.

Nordström, P., Samuelsson, M., & Åsberg, M. (1995). Survival analysis of suicide risk after attempted suicide. *Acta Psychiatrica Scandinavica*, *91*(5), 336-340.

Norris, F. H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology*, *60(*3), 409.

Park, N., Juo, S. H., Cheng, R., Liu, J., Loth, J. E., Lilliston, B., Nee, J., Grunn, A., Kanyas, K., Lerer, B., Endicott, J., Gilliam, T. C., & Baron, M. (2004). Linkage analysis of psychosis in bipolar pedigrees suggests novel putative loci for bipolar disorder and shared susceptibility with schizophrenia. *Molecular Psychiatry*, *9*(12), 1091-1099.

Passler, M. A., Isaac, W., & Hynd, G. W. (1985). Neuropsychological development of behavior attributed to frontal lobe functioning in children. *Developmental Neuropsychology*, *1(*4), 349-370.

Perroud, N., Courtet, P., Vincze, I., Jaussent, I., Jollant, F., Bellivier, F., Leboyer, M., Baud, P., Buresi, C., & Malafosse, A. (2008). Interaction between BDNF Val66Met and childhood trauma on adult's violent suicide attempt. *Genes, Brain and Behavior*, *7*(3), 314-322.

Perry, B. D. (1997). Incubated in terror: Neurodevelopmental factors in the "cycle of violence." *Children in a Violent Society*, 124-148.

Pfeffer, C. R., Martins, P., Mann, J., Sunkenberg, M., Ice, A., Damore, J. P., Gallo, C., Karpenos, I., & Jiang, H. (1997). Child survivors of suicide: Psychosocial characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(1), 65-74.

Pruessner, J. C., Baldwin, M. W., Dedovic, K., Renwick, R., Mahani, N. K., Lord, C., Meaney, M., & Lupien, S. (2005). Self-esteem, locus of control, hippocampal volume, and cortisol regulation in young and old adulthood. *Neuroimage*, *28(*4), 815-826.

Qin, P., Agerbo, E., & Mortensen, P. B. (2003). Suicide risk in relation to socioeconomic, demographic, psychiatric, and familial factors: A national register-based study of all suicides in Denmark, 1981-1997. *American Journal of Psychiatry*, *160*(4), 765-772.

Regier, 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. *Journal of the American Medical Association*, *264(*19), 2511-25

Reilly, J. L., & Sweeney, J. A. (2014). Generalized and specific neurocognitive deficits in psychotic disorders: Utility for evaluating pharmacological treatment effects and as intermediate phenotypes for gene discovery. *Schizophrenia Bulletin*, sbu013.

Roman, D. D. (1996). Review of the Rey Complex Figure Test and Recognition Trial. *Mental Measurement Yearbook*, 14.

Roy, A., Gorodetsky, E., Yuan, Q., Goldman, D., & Enoch, M. A. (2010). Interaction of FKBP5, a stress-related gene, with childhood trauma increases the risk for attempting suicide. *Neuropsychopharmacology*, *35*(8), 1674-1683.

Roy, A., Hu, X. Z., Janal, M. N., & Goldman, D. (2007). Interaction between childhood trauma and serotonin transporter gene variation in suicide. *Neuropsychopharmacology*, *32*(9), 2046-2052.

Salvatore, P., Baldessarini, R. J., Tohen, M., Khalsa, H. M. K., Sanchez-Toledo, J. P., Zarate, C. A., Jr., Vieta, E., & Maggini, C. (2009). The McLean-Harvard first episode project: Two-year stability of DSM-IV diagnoses in 500 first-episode psychotic disorder patients. *The Journal of Clinical Psychiatry*, *70*(4), 458.

Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *The Journal of Neuroscience*, *10(*9), 2897-2902.

Sarchiapone, M., Carli, V., Cuomo, C., & Roy, A. (2007). Childhood trauma and suicide attempts in patients with unipolar depression. *Depression and Anxiety*, *24*(4), 268-272.

Schoedl, A., Costa, M., Mari, J. J., Mello, M., Tyrka, A. R., Carpenter, L. L., & Price, L. H. (2010). The clinical correlates of reported childhood sexual abuse: An association between age at trauma onset and severity of depression and PTSD in adults. *Journal of Child Sexual Abuse, 19*(2), 156-170.

Serper, M. R., Bergman, A., Copersino, M. L., Chou, J. C., Richarme, D., & Cancro, R. (2000). Learning and memory impairment in cocaine-dependent and comorbid schizophrenic patients. *Psychiatry Research*, *93(*1), 21-32.

Simpson, G., & Tate, R. (2005). Clinical features of suicide attempts after traumatic brain injury. *The Journal of Nervous and Mental Disease*, *193*(10), 680-685.

Solomon, S. D., & Canino, G. (1990). The appropriateness of DSM-IHR criteria for post-traumatic stress disorder. *Comprehensive Psychiatry, 31,* 227-237

Somerville, J., Tremont, G., & Stern, R. A. (2000). The Boston Qualitative Scoring System as a measure of executive functioning in Rey-Osterrieth Complex Figure performance. *Journal of Clinical and Experimental Neuropsychology*, *22(*5), 613-621.

Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, *99(*2), 195.

Steel, Z., Silove, D., Phan, T., & Bauman, A. (2002). Long-term effect of psychological trauma on the mental health of Vietnamese refugees resettled in Australia: A population-based study. *The Lancet*, *360(*9339), 1056-1062

Steiger, J. H. (1980). Tests for comparing elements of a correlation matrix. *Psychological Bulletin*, *87*, 245 - 251.

Steiger, J. H., & Browne, M. W. (1984). The comparison of interdependent correlations between optimal linear composites. *Psychometrika, 49*, 11 - 24.

Steiger, J.H., & Fouladi, R.T. (n.d.) *R2 user's guide: Version 1.1.* University of British Columbia.

Stern, R. A., Javorsky, D. J., Singer, E. A., Singer-Harris, N. G., Somerville, J. A., Duke, L. M., Thompson, J. A., & Kaplan, E. (1999). *The Boston Qualitative Scoring System for the Rey-Osterrieth complex figure: Professional manual.* PAR.

Stiles, J., & Tada, W. L. (1996). Developmental change in children's analysis of spatial patterns. *Developmental Psychology*, *32(*5), 951.

Strakowski, S. M., Flaum, M., Amador, X., Bracha, H. S., Pandurangi, A. K., Robinson, D., & Tohen, M. (1996). Racial differences in the diagnosis of psychosis. *Schizophrenia Research*, *21*(2), 117-124.

Strakowski, S. M., Lonczak, H. S., Sax, K. W., West, S. A., Crist, A., Mehta, R., & Thienhaus, O. J. (1995). The effects of race on diagnosis and disposition from a psychiatric emergency service. *Journal of Clinical Psychiatry*, *56*(3), 101-107.

Substance Abuse and Mental Health Services Administration (2011). *Building resilience in children and youth dealing with trauma.* Retrieved from http://archive.samhsa.gov/children/trauma_resilience.asp.

Swanson, C. L., Jr., Gur, R. C., Bilker, W., Petty, R. G., & Gur, R. E. (1998). Premorbid educational attainment in schizophrenia: Association with symptoms, functioning, and neurobehavioral measures. *Biological Psychiatry*, *44(*8), 739-747.

Switzer, G. E., Dew, M. A., Thompson, K., Goycoolea, J. M., Derricott, T., & Mullins, S. D. (1999). Posttraumatic stress disorder and service utilization among urban mental health center clients. *Journal of Traumatic Sstress*, *12(*1), 25-39.

Tada, W. L., & Stiles-Davis, J. (1989). Children's analysis of spatial patterns: An assessment of their “errors” in copying geometric forms. *Cognitive Development*, *4(*2), 177-195.

Teasdale, T. W., & Engberg, A. W. (2001). Suicide after traumatic brain injury: A population study. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*(4), 436-440.

Tejedor, M. C., Diaz, A., Castillon, J. J., & Pericay, J. M. (1999). Attempted suicide: repetition and survival findings of a follow-up study. *Acta Psychiatrica Scandinavica*, *100*(3), 205-211.

van Reekum, R., Cohen, T., & Wong, J. (2000). Can traumatic brain injury cause psychiatric disorders? *Journal of Neuropsychiatry and Clinical Neurosciences*, *12(*3), 316-327.

van Zyl, M., Oosthuizen, P. P., & Seedat, S. (2008). Post traumatic stress disorder: Undiagnosed cases in a tertiary inpatient setting. *African Journal of Psychiatry*, *11(*2), 119-122.

Waber, D. P., & Holmes, J. M. (1985). Assessing children's copy productions of the Rey-Osterrieth Complex Figure. *Journal of Clinical and Experimental Neuropsychology*, *7(*3), 264-280.

Watanabe, Y., Gould, E., Daniels, D. C., Cameron, H., & McEwen, B. S. (1992). Tianeptine attenuates stress-induced morphological changes in the hippocampus. *European Journal of Pharmacology*, *222(*1), 157-162.

Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B. P., & Keane, T. M. (2013). *The Life Events Checklist for DSM-5 (LEC-5)*. Instrument available from the National Center for PTSD at www.ptsd.va.gov.

Welsh, M. C., Pennington, B. F., & Groisser, D. B. (1991). A normative-developmental study of executive function: A window on prefrontal function in children. *Developmental Neuropsychology*, *7(*2), 131-149.

Werner, E. E. (1993). Risk, resilience, and recovery: Perspectives from the Kauai longitudinal study. *Development and Psychopathology*, *5(*04), 503-515.

Whelan-Goodinson, R., Ponsford, J., Johnston, L., & Grant, F. (2009). Psychiatric disorders following traumatic brain injury: Their nature and frequency. *The Journal of Head Trauma Rehabilitation*, *24(*5), 324-332.

Wilcox, J. A., & Nasrallah, H. A. (1987). Childhood head trauma and psychosis. *Psychiatry Research*, *21(*4), 303-306.

Williams, L. M. (1994). Recall of childhood trauma: A prospective study of women's memories of child sexual abuse. *Journal of Consulting and Clinical Psychology*, *62(*6), 1167.

Wingenfeld, K., Schaffrath, C., Rullkoetter, N., Mensebach, C., Schlosser, N., Beblo, T., & Meyer, B. (2011). Associations of childhood trauma, trauma in adulthood and previous-year stress with psychopathology in patients with major depression and borderline personality disorder. *Child Abuse and Neglect*, *35(*8), 647-654.

Woolley, C. S., Gould, E., & McEwen, B. S. (1990). Exposure to excess glucocorticoids alters dendritic morphology of adult hippocampal pyramidal neurons. *Brain Research*, *531(*1), 225-231.

Yasumatsu, N., Matsuzaki, M., Miyazaki, T., Noguchi, J., & Kasai, H. (2008). Principles of long-term dynamics of dendritic spines. *Journal of Neuroscience,* 28(50), 13592-13608.

Yehuda, R., Halligan, S. L., Golier, J. A., Grossman, R., & Bierer, L. M. (2004). Effects of trauma exposure on the cortisol response to dexamethasone administration in PTSD and major depressive disorder. *Psychoneuroendocrinology,* 29(3), 389-404.

Zimmerman, M., & Mattia, J. I. (1999). Is posttraumatic stress disorder underdiagnosed in routine clinical settings? *Journal of Nervous and Mental Disease*, *187(*7), 420-428.

Zoroglu, S. S., Tuzun, U., Sar, V., Tutkun, H., Savaçs, H. A., Ozturk, M., Alyanak, B., & Kora, M. E. (2003). Suicide attempt and self-mutilation among Turkish high school students in relation with abuse, neglect and dissociation. *Psychiatry and Clinical Neurosciences*, *57*(1), 119-126.

Section VIII: Tables

Table 1

Correlations Among Scores and Predictors

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	--																			
2	.281**	--																		
3	-.053	-.054	--																	
4	-.011	-.113	.032	--																
5	.228**	.137	.228**	-.098	--															
6	-.231**	.021	-.050	.113	-.216**	--														
7	.045	.053	-.011	.002	-.296**	.001	--													
8	-.045	.129	-.032	.029	-.112	-.142	.077	--												
9	-.033	-.053	.330**	-.066	.259**	-.345**	-.076	-.031	--											
10	.451**	.228**	.030	-.082	.537**	-.369**	.022	-.142	.207**	--										
11	-.074	-.123	.240**	-.162*	.235**	-.409**	-.033	.079	.680**	.130	--									
12	-.093	-.076	-.079	-.141	.014	-.120	.014	.164*	-.264**	-.071	.525**	--								
13	.171*	.013	-.056	.115	-.021	.054	-.011	-.094	-.221**	-.012	-.141	-.012	--							
14	.036	-.008	.140	-.024	.071	.061	-.001	-.127	.064	-.056	.137	.033	.003	--						
15	-.055	-.052	.122	-.024	.017	.003	-.011	-.011	.062	-.137	.024	-.019	-.119	.180*	--					
16	.167*	.059	-.069	.099	.060	-.065	.014	-.042	-.034	.101	.051	.050	-.024	.277**	.085	--				
17	.040	.012	-.065	-.101	-.003	-.125	.034	.475**	.153*	.048	.165*	.041	-.024	-.079	.087	-.028	--			
18	.019	-.001	-.053	.016	-.062	.019	.068	.207**	-.036	.006	-.051	-.015	.051	-.016	.155*	-.122	.380**	--		
19	-.002	-.001	-.070	.021	-.095	-.040	.120	.318**	-.012	.090	.049	.060	.032	-.034	.089	.005	.495**	.588**	--	
20	.077	-.057	-.137	-.026	-.043	-.054	-.029	.360**	.033	-.015	.029	-.009	.100	-.049	.103	-.014	.604**	.350**	.091	--

1=BQSS Copy Score; 2=BQSS Organization Score; 3=Biological Sex; 4=Race; 5=Education Level; 6=Special Education; 7=Receipt of GED; 8=History of Any Head Injury/Trauma; 9=Age of Onset of Illness; 10=IQ; 11=Age; 12=Duration of Illness; 13=Presence of Secondary Gain; 14=Positive Symptoms of Psychosis; 15=Negative Symptoms of Psychosis; 16=Psychotic Diagnosis; 17=History of Psychological Trauma; 18=History of Polyvictimization; 19=History of Intentional Trauma; 20=History of Unintentional Trauma.

* p<.05. **p<.01.

Table 2

Dynamic Predictors of BQSS Copy Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	1.802	.115	.052					
Age				-.009	.815	-.082	.064	-.018
Duration of illness (years)				-.045	.353	-.140	.050	-.071
Presence of secondary gain				1.968	.033	.161	3.774	.163
Positive symptoms of psychosis				.941	.327	-.949	2.831	.075
Negative symptoms of psychosis				-2.396	.250	-6.498	1.705	-.088

Note. N=170.

Table 3

Static Predictors of BQSS Copy Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	4.798	<.001	.210					
Biological sex				.027	.977	-1.818	1.873	.002
Race				.365	.531	-.784	1.514	.046
Education level (years)				.116	.588	-.307	.540	.040
Special education				-1.368	.277	-3.846	1.110	-.081
Receipt of GED				.384	.786	-2.402	3.170	.020
History of any head injury/trauma				-.111	.908	-1.999	1.778	-.009
Age of onset of illness				-.075	.053	-.152	.001	-.145
IQ				.161	<.001	.083	.239	.301

Note. N=153.

Table 4

Dynamic Predictors of BQSS Organization Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	.827	.532	.025					
Age				-.008	.430	-.029	.012	-.061
Duration of illness (years)				-.006	.651	-.033	.021	-.035
Presence of secondary gain				.110	.668	-.397	.617	.033
Positive symptoms of psychosis				.245	.362	-.285	.776	.070
Negative symptoms of psychosis				-.738	.207	-1.889	.412	-.098

Note. *N*=170.

Table 5

Static Predictors of BQSS Organization Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	2.992	.004	.095					
Biological sex				-.161	.555	-.698	.377	-.046
Race				-.296	.083	-.631	.039	-.135
Education level (years)				.034	.592	-.090	.157	.041
Special education				.630	.087	-.092	1.351	.133
Receipt of GED				.258	.531	-.553	1.069	.048
History of any head injury/trauma				.635	.024	.086	1.185	.176
Age of onset of illness				-.010	.399	-.032	.013	-.065
IQ				.034	.003	.011	.057	.230

Note. *N*=153.

Table 6

Predictors of BQSS Copy Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	21.737	<.001	.225					
Presence of secondary gain				2.382	.006	.709	4.055	.202
IQ				.176	<.001	.118	.233	.435
2	3.778	<.001	.261					
Presence of secondary gain				2.071	.024	.272	3.869	.166
IQ				.162	<.001	.082	.241	.292
Age				-.115	.820	-1.112	.882	-.017
Duration of illness (years)				.055	.915	-.956	1.065	.008
Positive symptoms of psychosis				1.492	.120	-.394	3.379	.114
Negative symptoms of psychosis				-.025	.991	-4.221	4.171	-.001
Biological sex				-.086	.927	-1.944	1.772	-.007
Race				.025	.966	-1.132	1.182	.003
Education level (years)				.092	.669	-.334	.518	.031
Special education				-1.869	.147	-4.403	.666	-.106
Receipt of GED				.436	.755	-2.318	3.191	.023
History of any head injury/trauma				.353	.713	-1.538	2.243	.027
Age of onset of illness				.039	.939	-.959	1.036	.006

Note. N=153.

Table 7

Predictors of BQSS Organization Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	7.063	.001	.086					
IQ				.030	.001	.013	.048	.267
History of any head injury/trauma				.553	.045	.011	1.095	.157
2	2.341	.007	.180					
IQ				.030	.012	.007	.054	.195
History of any head injury/trauma				.729	.011	.172	1.285	.199
Age				.088	.553	-.205	.382	.046
Duration of illness (years)				-.116	.442	-.414	.181	-.059
Presence of secondary gain				.189	.480	-.340	.719	.054
Positive symptoms of psychosis				.252	.370	-.303	.808	.069
Negative symptoms of psychosis				-.389	.534	-1.625	.846	-.048
Biological sex				-.168	.545	-.715	.379	-.047
Race				-.358	.040	-.699	-.017	-.160
Education level (years)				.042	.513	-.084	.167	.050
Special education				.421	.266	-.325	1.167	.086
Receipt of GED				.319	.438	-.492	1.130	.060
Age of onset of illness				-.104	.486	-.397	.190	-.054

Note. *N*=153.

Table 8

Predictors of RCFT Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	30.724	<.001	.291					
IQ				.228	<.001	.165	.292	.487
Presence of secondary gain				3.389	<.001	1.530	5.249	.248
2	20.793	<.001	.295					
IQ				.233	<.001	.169	.297	.492
Presence of secondary gain				3.436	<.001	1.574	5.299	.251
History of any head injury/trauma				.981	.331	-1.007	2.970	.067
3	6.140	<.001	.365					
IQ				.217	<.001	.132	.303	.339
Presence of secondary gain				2.841	.004	.904	4.779	.196
History of any head injury/trauma				1.187	.251	-.849	3.223	.078
Age				.156	.775	-.919	1.230	.019
Duration of illness (years)				-.263	.633	-1.352	.825	-.032
Positive symptoms of psychosis				2.013	.052	-.019	4.046	.132
Negative symptoms of psychosis				1.175	.608	-3.346	5.695	.035
Biological sex				-1.169	.250	-3.171	.832	-.078
Race				-.109	.863	-1.355	1.137	-.012
Education level (years)				.153	.512	-.306	.612	.044
Special education				-2.103	.130	-4.834	.627	-.103
Receipt of GED				.579	.700	-2.388	3.547	.026
Age of onset of illness				-.272	.618	-1.346	.803	-.034

Note. *N*=153.

Table 9

Predictors of TMT Part B Score

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	20.778	<.001	.224					
IQ				2.306	<.001	1.556	3.056	.446
Presence of secondary gain				26.828	.016	5.081	48.575	.179
2	13.792	<.001	.224					
IQ				2.291	<.001	1.532	3.050	.439
Presence of secondary gain				26.655	.017	4.805	48.505	.178
History of any head injury/trauma				-3.411	.772	-26.672	19.850	-.021
3	5.290	<.001	.341					
IQ				2.687	<.001	1.706	3.668	.381
Presence of secondary gain				17.914	.114	-4.385	40.212	.112
History of any head injury/trauma				2.885	.807	-20.437	26.208	.017
Age				-9.735	.113	-21.789	2.319	-.112
Duration of illness (years)				7.864	.205	-4.356	20.085	.090
Positive symptoms of psychosis				12.993	.280	-10.690	36.675	.076
Negative symptoms of psychosis				-7.894	.775	-62.332	46.544	-.020
Biological sex				15.124	.193	-7.719	37.966	.092
Race				-2.646	.708	-16.608	11.316	-.026
Education level (years)				-1.740	.503	-6.870	3.389	-.047
Special education				-9.646	.543	-40.921	21.629	-.043
Receipt of GED				1.508	.928	-31.620	34.637	.006
Age of onset of illness				7.941	.196	-4.136	20.017	.092

Note. *N*=147.

Table 10

Predictors of TMT Part A Score

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	16.094	<.001	.098					
IQ				.450	<.001	.228	.671	.313
2	5.464	.001	.101					
IQ				.452	<.001	.227	.676	.312
History of any head injury/trauma				-.507	.885	-7.425	6.411	-.011
Presence of secondary gain				2.168	.510	-4.326	8.663	.052
3	4.515	<.001	.301					
IQ				.472	.001	.194	.750	.241
History of any head injury/trauma				-.450	.893	-7.033	6.133	-.010
Presence of secondary gain				-2.003	.530	-8.290	4.285	-.045
Age				-.724	.680	-4.184	2.737	-.030
Duration of illness (years)				.048	.978	-3.458	3.555	.002
Positive symptoms of psychosis				2.124	.529	-4.527	8.774	.045
Negative symptoms of psychosis				-1.551	.834	-16.134	13.031	-.015
Biological sex				3.452	.296	-3.059	9.964	.075
Race				.816	.688	-3.192	4.823	.029
Education level (years)				-.537	.473	-2.010	.937	-.052
Special education				-11.235	.013	-20.073	-2.398	-.180
Receipt of GED				3.700	.444	-5.830	13.231	.055
Age of onset of illness				-.018	.992	-3.485	3.449	-.001

Note. *N*=150.

Table 11

Comparison of Performance Across Measures Based on Presence of Secondary Gain

	Group										
	No Secondary Gain			Secondary Gain			95% CI for $\bar{x}\Delta$				
Score	$\bar{x}$	*SD*	*n*	$\bar{x}$	*SD*	*n*	Lower bound	Upper bound	*t*	*df*	*p*
BQSS Copy	27.530	6.290	115	29.580	4.489	65	-3.651	-.458	-2.540	168.420	.012
BQSS Organization	2.896	1.656	115	2.939	1.467	65	-.515	.429	-.179	146.689	.858
RCFT	26.840	7.452	115	29.320	5.370	65	-4.372	-.571	-2.568	167.642	.011
TMT Part A	91.071	18.427	112	92.234	24.038	64	-8.048	5.722	-.335	105.653	.738
TMT Part B	264.883	77.314	111	278.871	63.520	62	-35.539	7.563	-1.283	147.656	.202

Note. Values reflect equal variances not assumed as Levene's Test for Equality of Variances was significant for at least one comparison.

Table 12

Comparison of Performance Across Measures Based on History of Any Trauma/Head Injury

	Group										
	No History of Any Trauma			History of Any Trauma			95% CI for $\bar{x}\Delta$				
Score	$\bar{x}$	*SD*	*n*	$\bar{x}$	*SD*	*n*	Lower bound	Upper bound	*t*	*df*	*p*
BQSS Copy	28.660	5.428	56	28.100	5.942	124	-1.275	2.403	.605	178	.546
BQSS Organization	2.607	1.288	56	3.048	1.691	124	-.943	.060	-1.737	178	.084
RCFT	27.720	6.091	56	27.740	7.208	124	-2.205	2.168	-.017	178	.987
TMT Part A	92.600	18.329	55	90.992	21.585	121	-5.013	8.230	.479	174	.632
TMT Part B	275.889	65.498	54	267.177	75.982	119	-14.896	32.321	.728	171	.467

Note. Values reflect equal variances assumed as Levene's Test for Equality of Variances was not significant for any comparisons.

Table 13

Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	3.982	.053	.095					
IQ				.187	.053	-.003	.377	.308
2	3.713	.020	.236					
IQ				.143	.127	-.043	.329	.227
Presence of secondary gain				4.120	.075	-.438	8.678	.267
Negative symptoms of psychosis				-11.486	.128	-26.454	3.482	-.227
3	1.090	.408	.353					
IQ				.024	.851	-.233	.281	.030
Presence of secondary gain				4.309	.106	-.980	9.598	.264
Negative symptoms of psychosis				-14.166	.140	-33.311	4.980	-.240
Age				-1.201	.849	-14.009	11.608	-.030
Duration of illness (years)				1.078	.865	-11.807	13.963	.027
Positive symptoms of psychosis				-2.208	.643	-11.895	7.478	-.074
Biological sex				-1.149	.683	-6.857	4.560	-.065
Race				.533	.815	-4.092	5.158	.037
Education level (years)				.749	.415	-1.109	2.607	.131
Special education				-3.096	.505	-12.507	6.316	-.107
Receipt of GED				6.665	.204	-3.841	17.170	.206
History of any head injury/trauma				-3.778	.301	-11.136	3.580	-.167
Age of onset of illness				1.076	.863	-11.661	13.813	.027

Note. *N*=40.

Table 14

Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	37.587	<.001	.253					
IQ				.164	<.001	.111	.216	.503
2	19.696	<.001	.264					
IQ				.148	<.001	.090	.206	.413
Special education				-1.373	.208	-3.522	.777	-.104
3	4.305	<.001	.361					
IQ				.171	<.001	.097	.244	.369
Special education				-2.395	.043	-4.712	-.079	-.165
Age				-.127	.756	-.936	.682	-.025
Duration of illness (years)				.063	.879	-.758	.885	.012
Presence of secondary gain				1.477	.098	-.279	3.233	.134
Positive symptoms of psychosis				1.527	.078	-.173	3.226	.143
Negative symptoms of psychosis				1.071	.561	-2.573	4.715	.047
Biological sex				.442	.632	-1.380	2.263	.039
Race				.006	.990	-1.038	1.051	.001
Education level (years)				-.096	.623	-.482	.290	-.040
Receipt of GED				-1.671	.186	-4.164	.821	-.107
History of any head injury/trauma				.996	.250	-.713	2.706	.093
Age of onset of illness				.056	.892	-.756	.867	.011

Note. *N*=113.

Table 15

Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	2.332	.135	.058					
Receipt of GED				1.028	.135	-.335	2.390	.240
2	.399	.957	.166					
Receipt of GED				1.560	.125	-.464	3.584	.284
Age				-.400	.742	-2.867	2.068	-.060
Duration of illness (years)				.380	.756	-2.102	2.862	.056
Presence of secondary gain				-.079	.875	-1.098	.940	-.028
Positive symptoms of psychosis				.574	.533	-1.292	2.440	.113
Negative symptoms of psychosis				.208	.909	-3.480	3.896	.021
Biological sex				-.455	.403	-1.555	.644	-.152
Race				-.087	.842	-.978	.804	-.036
Education level (years)				.168	.343	-.190	.526	.173
Special education				-.258	.772	-2.071	1.555	-.052
History of any head injury/trauma				.341	.625	-1.076	1.759	.089
Age of onset of illness				.383	.751	-2.071	2.836	.057
IQ				.006	.801	-.043	.056	.046

Note. *N*=40.

Table 16

Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	6.305	.003	.103					
IQ				.033	.002	.013	.054	.288
History of any head injury/trauma				.632	.056	-.018	1.281	.174
2	4.883	.003	.118					
IQ				.032	.003	.011	.053	.276
History of any head injury/trauma				.628	.057	-.018	1.275	.173
Race				-.280	.167	-.679	.119	-.125
3	2.157	.017	.221					
IQ				.041	.006	.012	.070	.251
History of any head injury/trauma				.888	.010	.218	1.559	.233
Race				-.424	.043	-.833	-.014	-.182
Age				.054	.736	-.263	.372	.030
Duration of illness (years)				-.086	.596	-.409	.236	-.047
Presence of secondary gain				.272	.435	-.416	.961	.070
Positive symptoms of psychosis				.284	.401	-.383	.950	.075
Negative symptoms of psychosis				-.626	.387	-2.055	.803	-.077
Biological sex				.021	.954	-.694	.735	.005
Education level (years)				-.002	.978	-.154	.149	-.002
Special education				.591	.200	-.318	1.499	.114
Receipt of GED				.008	.988	-.970	.985	.001
Age of onset of illness				-.076	.636	-.395	.242	-.042

Note. *N*=113.

Table 17

Comparison of BQSS Copy and Organization Performance Based on Diagnosis and History of Psychological Trauma

	Group																	
	No History of Psychological Trauma									History of Psychological Trauma								
	Non-Psychotic Diagnosis (n=25)		Psychotic Diagnosis (n=69)		95% CI for $\bar{x}\Delta$					Non-Psychotic Diagnosis (n=25)		Psychotic Diagnosis (n=61)		95% CI for $\bar{x}\Delta$				
Score	$\bar{x}$	*SD*	$\bar{x}$	*SD*	Lower bound	Upper bound	*t*	*df*	*p*	$\bar{x}$	*SD*	$\bar{x}$	*SD*	Lower bound	Upper bound	*t*	*df*	*p*
BQSS Copy	26.72	7.40	28.54	4.96	-5.07	1.44	-1.138	32.145	0.264	26.72	7.98	29.25	4.61	-1.49	30.80	0.148	-5.996	0.945
BQSS Organization	3.04	1.54	2.84	1.53	-0.53	0.92	0.556	42.319	0.581	2.48	0.87	3.12	1.86	-1.22	-0.05	-2.149	82.419	0.035

Note. Values reflect equal variances not assumed as Levene's Test for Equality of Variances was significant for at least one comparison.

Table 18

Polyvictimization and Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	5.469	.029	.207					
Presence of secondary gain				7.369	.029	.816	13.923	.455
2	5.869	.010	.370					
Presence of secondary gain				7.267	.020	1.263	13.271	.448
History of polyvictimization				6.654	.034	.556	12.753	.404
3	3.708	.023	.452					
Presence of secondary gain				6.310	.042	.239	12.380	.381
History of polyvictimization				5.393	.092	-.971	11.757	.311
IQ				.083	.544	-.199	.366	.108
Negative symptoms of psychosis				-9.438	.221	-25.062	6.187	-.221

Note. *N*=23.

Table 19

Polyvictimization and Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	12.760	.001	.186					
IQ				.132	.001	.058	.206	.431
2	7.535	.001	.215					
IQ				.127	.001	.053	.200	.412
History of polyvictimization				-1.942	.156	-4.648	.765	-.172

Note. *N*=58.

Table 20

Polyvictimization and Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	6.662	.017	.241					
IQ				.037	.017	.007	.067	.491
2	3.179	.063	.241					
IQ				.038	.025	.005	.070	.473
History of polyvictimization				-.036	.924	-.804	.732	-.019
3	2.376	.091	.346					
IQ				.037	.035	.003	.072	.435
History of polyvictimization				-.011	.976	-.788	.765	-.006
Receipt of GED				-.912	.152	-2.192	.368	-.285
History of any head injury/trauma				-.350	.591	-1.696	.996	-.104

Note. *N*=23.

Table 21

Polyvictimization and Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	1.055	.309	.018					
IQ				.018	.309	-.017	.053	.136
2	.518	.598	.018					
IQ				.018	.315	-.017	.053	.135
History of polyvictimization				.005	.994	-1.291	1.301	.001
3	.366	.777	.020					
IQ				.018	.307	-.017	.054	.139
History of polyvictimization				-.017	.979	-1.334	1.300	-.004
History of any head injury/trauma				.289	.778	-1.756	2.335	.038

Note. *N*=58.

Table 22

Type of Trauma and Predictors of BQSS Copy Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	5.469	.029	.207					
Presence of secondary gain				7.369	.029	.816	13.923	.455
2	5.946	.009	.373					
Presence of secondary gain				6.053	.052	-.054	12.159	.366
History of intentional trauma				6.847	.032	.644	13.049	.408
3	3.406	.026	.500					
Presence of secondary gain				7.378	.037	.486	14.269	.387
History of intentional trauma				6.311	.080	-.849	13.471	.319
History of unintentional trauma				-4.342	.205	-11.286	2.601	-.226
IQ				-.034	.827	-.355	.287	-.038
Negative symptoms of psychosis				-13.231	.095	-29.012	2.550	-.303

Note. *N*=23.

Table 23

Type of Trauma and Predictors of BQSS Copy Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma

						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	12.760	.001	.186					
IQ				.132	.001	.058	.206	.431
2	11.112	<.001	.288					
IQ				.130	<.001	.060	.200	.425
History of intentional trauma				-2.907	.007	-4.980	-.833	-.320
3	7.367	<.001	.290					
IQ				.129	.001	.058	.199	.417
History of intentional trauma				-3.119	.009	-5.413	-.824	-.312
History of unintentional trauma				-.500	.656	-2.741	1.740	-.051

Note. *N*=58.

Table 24

Type of Trauma and Predictors of BQSS Organization Score for Consumers with Non-Psychotic Diagnoses and History of Psychological Trauma

Step and Predictor	*F*	*p*	R^2	*B*	*p*	95% CI for *B* Lower bound	95% CI for *B* Upper bound	*sr*
1	6.662	.017	.241					
IQ				.037	.017	.007	.067	.491
2	3.607	.046	.265					
IQ				.045	.017	.009	.081	.498
History of intentional trauma				-.332	.426	-1.185	.521	-.156
3	2.523	.070	.426					
IQ				.037	.054	-.001	.074	.380
History of intentional trauma				-.165	.685	-1.009	.679	-.076
History of unintentional trauma				-.505	.167	-1.245	.234	-.265
History of any head injury/trauma				.025	.969	-1.352	1.402	.007
Receipt of GED				-.946	.127	-2.191	.299	-.295

Note. *N*=23.

Table 25

Type of Trauma and Predictors of BQSS Organization Score for Consumers with Psychotic Diagnoses and History of Psychological Trauma

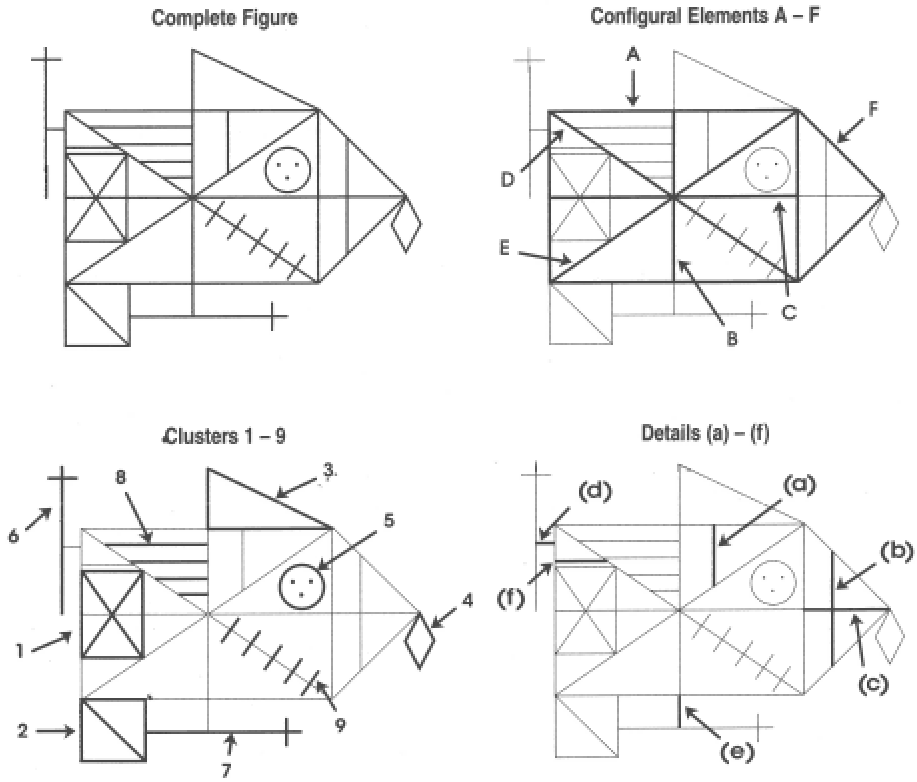
						95% CI for *B*		
Step and Predictor	*F*	*p*	R^2	*B*	*p*	Lower bound	Upper bound	*sr*
1	1.055	.309	.018					
IQ				.018	.309	-.017	.053	.136
2	.555	.577	.020					
IQ				.018	.315	-.017	.053	.135
History of intentional trauma				-.140	.789	-1.181	.902	-.036
3	.583	.676	.042					
IQ				.018	.329	-.018	.053	.132
History of intentional trauma				-.511	.414	-1.756	.734	-.111
History of unintentional trauma				-.653	.295	-1.891	.586	-.142
History of any head injury/trauma				.862	.452	-1.421	3.145	.102

Note. *N*=58.

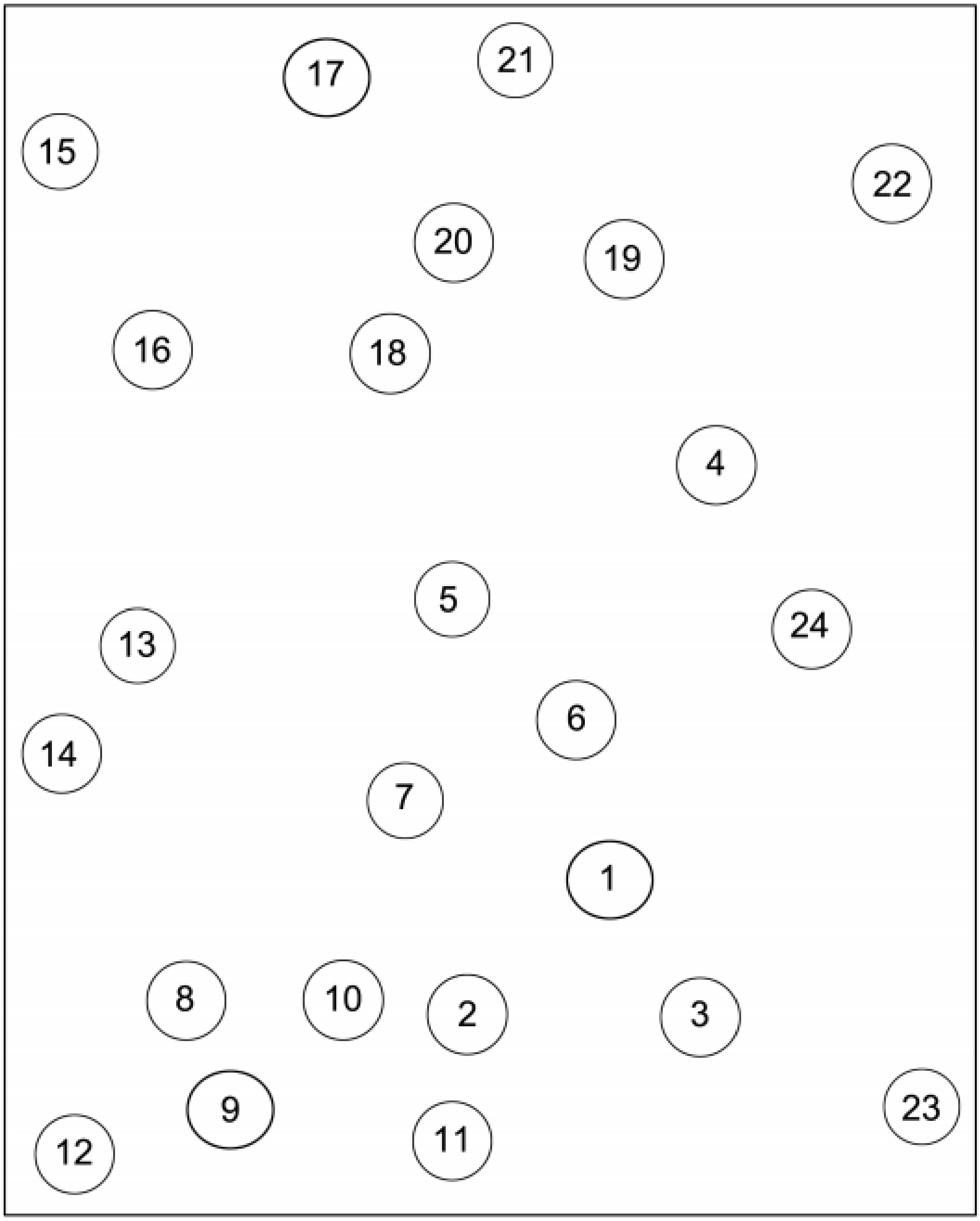
Appendix A: Life Events Checklist, Modified Trauma Thesis Version

Event	Patient Experienced	Number of Occurrences*
1. Natural disaster (for example, flood, hurricane, tornado, earthquake)		
2. Fire or explosion		
3. Transportation accident (for example, car accident, boat accident, train wreck, plane crash)		
4. Serious accident at work, home, or during recreational activity		
5. Exposure to toxic substance (for example, dangerous chemicals, radiation)		
6. Physical assault (for example, being attacked, hit, slapped, kicked, beaten up)		
7. Assault with a weapon (for example, being shot, stabbed, threatened with a knife, gun, bomb)		
8. Sexual assault (rape, attempted rape, made to perform any type of sexual act through force or threat of harm)		
9. Other unwanted or uncomfortable sexual experience		
10. Combat or exposure to a war-zone (in the military or as a civilian)		
11. Captivity (for example, being kidnapped, abducted, held hostage, prisoner of war)		
12. Life-threatening illness or injury		
13. Severe human suffering		
14. Sudden, violent death (for example, homicide, suicide)		
15. Sudden, unexpected death of someone close to you		
16. Serious injury, harm, or death you caused to someone else		
17. Suicide attempt		
18. Any other very stressful event or experience		
	Total Number of Event Types	Total Number of Events*

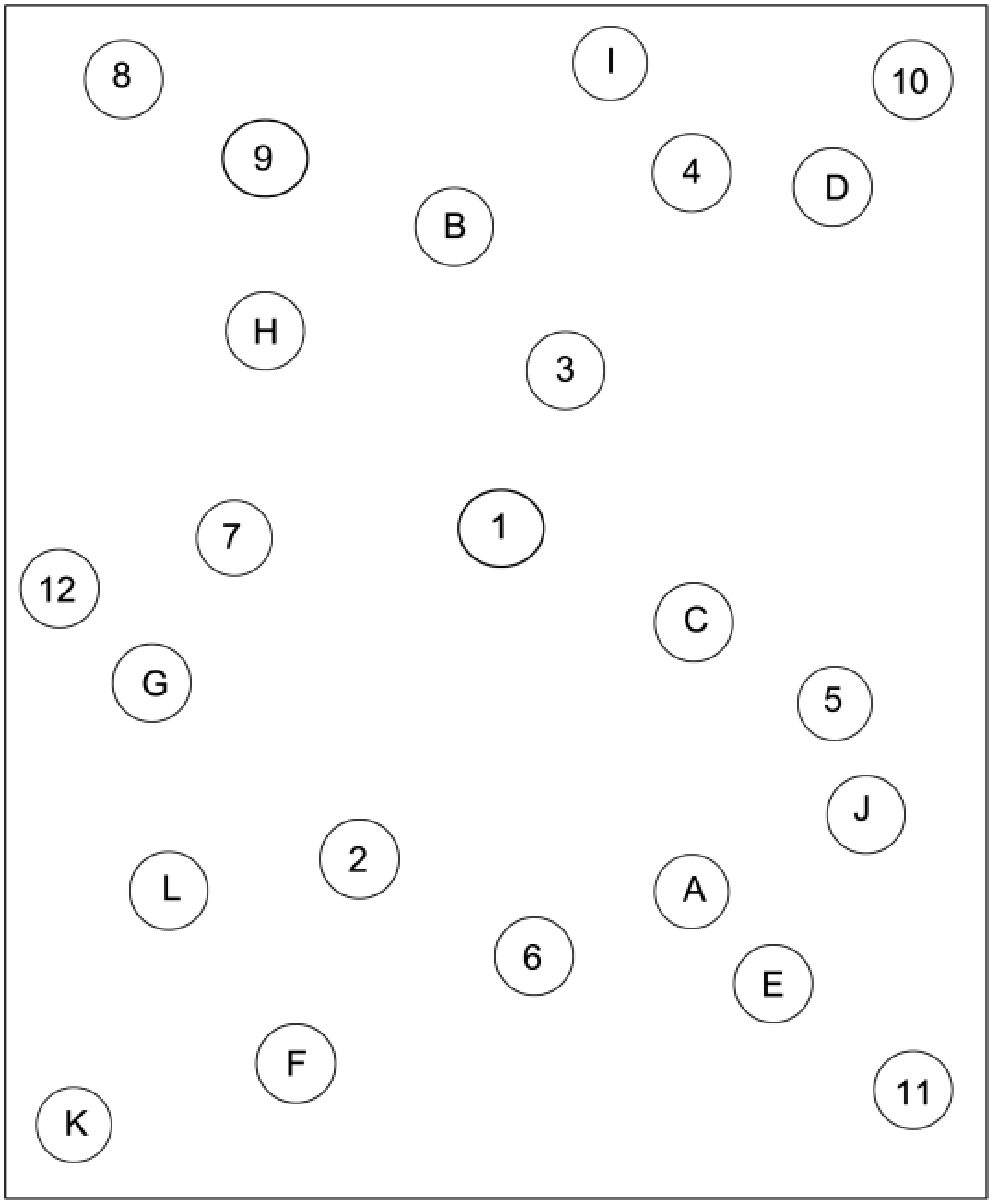
Appendix B: RCFT Figure



Appendix C: TMT Part A



Appendix D: TMT Part B



Appendix E: Supplementary Results

E1. Interactions

For the BQSS copy score, only age and age of onset of illness were indicated as having a significant interaction (*b*=-0.007, *p*=0.31, sr^2=.027). However, this effect was small and the overall model (*n*=170) including the interaction was not significant [R^2=.038, *F*(3,166)=2.211, *p*=.089, R^2 *95% CI:* .000 to .098]. There were no other significant two-way interactions for BQSS copy score.

For the BQSS organization score, age of onset of illness and duration of illness were indicated as having a statistically significant interaction (*b*=-0.003, *p*=0.018, sr^2=.033). However, this effect was small and the overall model (*n*=170) including the interaction was not significant [R^2=.045, *F*(3,166)=2.585, *p*=.055, R^2 *95% CI:* .000 to .109].

In addition, secondary gain was found to significantly interact with duration of illness (*b*=0.047, *p*=0.048, sr^2=.023), with the relationship between BQSS organization score and duration of illness increasing with the presence of secondary gain. However, this effect was small and the overall model (*n*=170) including the interaction was not significant [R^2=.032, *F*(3,166)=1.840, *p*=.142, R^2 *95% CI:* .000 to .088].

Duration of illness was also found to significantly interact with both positive symptoms of psychosis (*b*=-0.055, *p*=0.037, sr^2=.026) and negative symptoms of psychosis (*b*=-0.202, *p*=0.015, sr^2=.035), with the relationship between BQSS organization score and duration of illness decreasing with the presence of positive or negative psychotic symptoms. The overall model (*n*=170) including the interaction was not significant for positive symptoms [R^2=.033, *F*(3,166)=1.914, *p*=.129, R^2 *95% CI:* .000

to .090], though it was statistically significant for negative symptoms [R^2=.049, *F*(3,166)=2.855, *p*=.039, R^2 *95% CI:* .000 to .116]. However, these effects are small and as the confidence intervals include zero, indicating that there may not be true practical significance in the larger population.

Negative symptoms of psychosis were found to significantly interact with IQ (*b*=0.113, *p*=0.049, sr^2=.023) with the relationship between BQSS organization score and IQ increasing with the presence of negative psychotic symptoms. In addition, the overall model (*n*=166) including the interaction was statistically significant [R^2=.076, *F*(3,162)=4.411, *p*=.005, R^2 *95% CI:* .008 to .156], and though this effect is small the confidence interval does not include zero, suggesting that there may likely be true practical significance in the larger population.

None of these interactions remained statistically or practically significant when included in the final models.

E2. Best Model

For the BQSS copy score, the full model including all of the indicated predictor variables (*n*=153) was found to account for a statistically significant percentage of score variance [R^2=.261, *F*(13,139)=3.778, *p*<.001, R^2 *95% CI:* .081 to .325]. However, only secondary gain (*b*=2.071, *p*=0.024, sr^2=.028) and IQ (*b*=0.162, *p*<.001, sr^2=.085) significantly contributed to the model, which is in line with the results of the regressions calculated in the first primary analysis. As a result, a multiple regression was conducted to determine the predictive validity of a reduced model including only secondary gain and IQ compared to the full model.

For the BQSS organization score, both the full basic model [R^2=.180, F(13,139)=2.341, p=.007, R^2 *95% CI:* .015 to .230] as well as the full model including the interaction between IQ and negative psychotic symptoms [R^2=.185, F(14,138)=2.244, p=.009, R^2 *95% CI:* .013 to .230] accounted for a statistically significant percentage of variance in BQSS organization score (n=153). However, the previously statistically and practically significant interaction did not significantly contribute to the model [$R^2\Delta$=.006, $F\Delta$ (1,138)=0.979, p=.324]. As such, this interaction will be excluded from the final model and the main effects interpreted.

In addition, only race (b=-0.358, p=.040, sr^2=.026), IQ (b=0.030, p=0.012, sr^2=.038) and head injury/trauma (b=0.729, p=0.011, sr^2=.040) significantly contributed to the model, which is in line with the results of the regressions calculated in the first primary analysis with the exception of the significant contribution of race. However, the statistical significance of race is likely a result of limited variability as 80.6% of the sample identifies as Caucasian. As a result, a multiple regression was conducted to determine the predictive validity of two reduced models compared to the full model: one including only IQ and head injury/trauma, and one including race in addition to these two variables.

E3. RCFT and TMT

For the BQSS copy score, the predictive validity of the best model (IQ and secondary gain) does not statistically differ for RCFT score (Z=1.712, p>.05) or time to completion for TMT Part B (Z=0.565, p>.05). However, the model accounts for a statistically significantly larger percentage of variance in BQSS copy score compared to number of TMT Part A errors (Z=3.726, p<.05), to time to completion for TMT Part A (Z=1.972, p<.05), and number of TMT Part B errors (Z=2.809, p<.05).

For the BQSS organization score, the predictive validity of the best model (IQ and head injury/trauma) does not statistically differ for RCFT score (Z=1.888, p>.05), number of errors on TMT Part A (Z=1.501, p>.05), time to completion on TMT Part A (Z=0.092, p>.05), number of errors on TMT Part B (Z=0.203, p>.05), or time to completion on TMT Part B (Z=-0.860, p>.05).

Given the results of the supplementary analyses, additional multiple regressions were conducted to determine the predictive validity of IQ, secondary gain, and head injury/trauma for RCFT score, number of errors, and time to completion on TMT Parts A and B.

E4. IQ

Though IQ was found to be the strongest predictor in all models as well as a statistically significant predictor of all scores tested with the exception of number of TMT Part A errors, the practical significance of these results must be taken into account. IQ accounts for less than approximately 10% of the variance in BQSS organization score (sr^2=.071), numbers of errors on TMT Part A (sr^2=.017), time to completion on TMT Part A (sr^2=.098), and number of errors on TMT Part B (sr^2=.091). However, the effect of IQ is much larger for BQSS copy score (sr^2=.189), RCFT score (sr^2=.237), and time to completion on TMT Part B (sr^2=.200).

As a result of these analyses, two additional multiple regressions were conducted to determine the predictive validity of overall IQ compared to Performance IQ as assessed by the WAIS for both the BQSS copy and organization scores. Results indicated that Performance IQ (*b*=.218, *p*=.001, sr^2=.055) is a significantly stronger predictor of BQSS copy score compared to overall IQ (*b*=.003, *p*=.963, sr^2<.001). In addition, Performance IQ (*b*=.031, *p*=.135, sr^2=.015) is a stronger, though not statistically significant, predictor of BQSS organization score compared to overall IQ (*b*=.002, *p*=.903, sr^2<.001). However, Performance IQ is highly correlated with overall IQ as would be expected (*r=.885, p*<.001*)*, such that these results are likely a result of multicollinearity. As this indicates that nearly 80% of the variance in Performance IQ can be explained by overall IQ in the current population, inclusion of only overall IQ in each model is statistically appropriate (Field, 2013).

E5. Psychosis

For BQSS copy score, none of the predictors significantly contribute to the full model for individuals without a psychotic diagnosis, though negative symptoms of psychosis (*b*=-14.166, *p*=.140, sr^2=.058) and secondary gain (*b*=4.309, *p*=.106, sr^2=.070) were the strongest predictors. For those with a psychotic diagnosis, only IQ (*b*=.171, *p*<.001, sr^2=.136) and special education (*b*=-2.395, *p*=.043, sr^2=.027) significantly contributed to the full model. As such, two additional multiple regressions were conducted to determine the predictive validity of reduced models compared to the full model for BQSS copy score.

For BQSS organization score, none of the predictors significantly contribute to the full model for individuals without a psychotic diagnosis, though GED was the strongest predictor (*b*=1.560, *p*=.125, sr^2=.081). For those with a psychotic diagnosis, only IQ (*b*=.041, *p*=.006, sr^2=.063), head injury/trauma (*b*=.888, *p*=.010, sr^2=.054), and race (*b*=-0.424, *p*=.043, sr^2=.033) significantly contributed to this model. As such, two additional multiple regressions were conducted to determine the predictive validity of reduced models compared to the full model for BQSS organization score.