DEPARTMENT OF PSYCHOLOGY

SELF-AWARENESS, QUALITY OF LIFE, AND BRAIN VOLUME IN CHRONIC TBI.

DOCTOR OF PHILOSOPHY DISSERTATION

EVA PETTEMERIDOU

2017

DEPARTMENT OF PSYCHOLOGY

SELF-AWARENESS, QUALITY OF LIFE, AND BRAIN VOLUME IN CHRONIC TBI.

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A Dissertation Submitted to the University of Cyprus in Partial Fulfillment

of the Requirements of the Degree of Doctor of Philosophy

December 2017

VALIDATION PAGE

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Dissertation Title: Self-awareness, Quality of Life, and Brain Volume in TBI.

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The present doctoral dissertation was submitted in partial fulfillment of the requirements of the degree of Doctor of Philosophy of the University of Cyprus. It is a product of original work of my own, unless otherwise mentioned through references, notes, or any other statements.

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ΠΕΡΙΛΗΨΗ

Η παρούσα έρευνα εξέτασε εάν μεγαλύτερη ατροφία του εγκεφάλου, όπως αξιολογείται μέσω μαγνητικής τομογραφίας (ΜΤ), σχετίζεται με μεγαλύτερη εξασθένηση των νευροψυχολογικών λειτουργιών και την αυτογνωσία, σε άτομα με χρόνια μέτρια-σοβαρή κρανιοεγκεφαλική κάκωση (ΚΕΚ). Ένα δεύτερο ερευνητικό ερώτημα ήταν κατά πόσον χαμηλότερα επίπεδα αυτογνωσίας σχετίζονται ή μπορούν να προβλέψουν υψηλότερα επίπεδα ποιότητας ζωής (ΠΖ). Ένας τρίτος στόχος ήταν να διερευνηθεί η σχέση μεταξύ της απώλειας εγκεφαλικού όγκου και της ΠΖ, εξετάζοντας έτσι κατά πόσον ο υψηλότερος βαθμός ατροφίας του εγκεφάλου σχετίζεται ή μπορεί να προβλέψει την ΠΖ. Το δείγμα περιελάβανε 57 Ελληνοκύπριους ενήλικες (33 άτομα με ΤΒΙ, 24 υγιή άτομα), ηλικίας 18-60 ετών, ένα έτος μετά τον τραυματισμό τους. Οι συμμετέχοντες με ΚΕΚ ήταν ζευγοποιημένοι με τους υγιείς ενήλικες στην ηλικία, το φύλο και την εκπαίδευση. Η ομάδα ελέγχου χρησιμοποιήθηκε για να εξασφαλιστεί ότι τυχόν μεταβολές στη μορφολογία του εγκεφάλου, τη νευροψυχολογική απόδοση και την ΠΖ οφείλονται στον τραυματισμό. Όλοι οι συμμετέχοντες υποβλήθηκαν σε νευροψυχολογική αξιολόγηση, ΜΤ και σε μια σειρά από ψυχοκοινωνικά εργαλεία, συμπεριλαμβανομένου ερωτηματολογίων για την ΠΖ. Μεγαλύτερη ατροφία στον εγκέφαλο συσχετίστηκε και προέβλεπε μεγαλύτερη έκπτωση στις εκτελεστικές λειτουργίες και ανεπαρκή αυτογνωσία. Η αυτογνωσία έδειξε σημαντικές συσχετίσεις με την ΠΖ. Ωστόσο, τα ευρήματα αυτά μετριάστηκαν από τους μηχανισμούς αντιμετώπισης και την διάθεση. Μόνο οι υγιείς ενήλικες με μεγαλύτερη αυτογνωσία ανέφεραν μεγαλύτερη δυσαρέσκεια με την ΠΖ τους. Τέλος, φαίνεται ότι άτομα με χρόνια ΚΕΚ και μεγαλύτερη ατροφία στον πρωταρχικό αισθητήριο φλοιό και στη κροταφική περιοχή μπορούν να προβλέψουν μεγαλύτερη ικανοποίηση με τη συνολική ΠΖ σχετιζόμενη με την υγεία, και την αντίληψη του εαυτού τους. Επιπλέον, μεγαλύτερος όγκος

στον ιππόκαμπο προβλέπει μειωμένη ικανοποίηση όσον αφορά τις περιβαλλοντικές πτυχές της ζωής, όπως αυτό αξιολογήθηκε σε όλους τους συμμετέχοντες.

ABSTRACT

The current research project examined whether greater brain atrophy, as measured by Magnetic Resonance Imaging (MRI), related to greater impairment in neuropsychological tasks and self-awareness (SA) in individuals with chronic moderate-severe traumatic brain injury (TBI). A second aim of this study was to investigate whether greater impairment in SA related to, or could predict greater levels of quality of life (QOL). A third aim was to explore the relationship between brain volume loss and QOL, thus examining whether greater degree of brain atrophy is related to or is predictive of QOL. The sample consisted of 57 Greek Cypriot adults (33 individuals with TBI, 24 healthy individuals) with an age range of 18–60 years old, at one year post-injury; participants with TBI were be pair-matched to the healthy controls on age, gender, and education. A control group was used to ensure that any changes in brain morphology, neuropsychological performance and QOL resulted from the injury. All participants underwent a neuropsychological assessment, an MRI scan, and a number of QOL and psychosocial measures. Greater brain atrophy in EF and SA –related cortical and sub-cortical regions correlated and predicted greater dysexecutive functioning and SA deficits. Self-awareness showed significant associations with QOL; however, these findings were moderated by coping mechanisms and diathesis, revealing only that healthy controls with greater SA reported greater dissatisfaction with their QOL. Finally, it appears that in chronic TBI greater atrophy in the primary sensory cortex and the temporal area may predict greater satisfaction with overall health-related QOL and the perception of one's self. In addition, greater volume in the hippocampus predicts reduced satisfaction regarding the environmental aspects of one's life, for all participants.

ACKNOWLEDGMENTS

First, and foremost, I would like to express my heartfelt gratitude to my supervisor Dr. Fofi Constantinidou. Her endless support and scientific guidance throughout this long-term process have been the driving force to completing this personal goal. She has been tremendously dedicated to both her student's education and personal growth. It has been a great pleasure and honor to have worked with her.

During my proposal presentation my examiners were Dr. Michael Lombardo and Dr. Mary Kennedy. I am hugely indebted to Dr. Michael Lombardo who has guided me through the MRI data analysis, and provided me with insightful comments and suggestions. I would also wish to express my sincere appreciation to Dr. Mary Kennedy from whom I have received very helpful feedback, which guided several aspects of my research.

Furthermore, I am especially grateful to Marina Charalambous, and Dr. Demetris Demetriou, and the staff of the Melathron Agoniston EOKA, and Michelle Kokkinou from the ICU, for helping me with the recruitment process. Also, a huge thanks to the Ayios Therissos Medical center staff for conducting the MRI scans, and Stephanie Phylactou for helping me with the data collection.

Lastly, I have kept my deepest gratitude and greatest thank you to my family, and especially my father, for their love, support, and constant encouragement because without them, none of this would have been possible; this is dedicated to them.

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CHAPTER 1

INTRODUCTION

Traumatic brain injury (TBI) is a major cause of hospitalization and death worldwide. In the USA alone, 1.7 million people sustain TBI each year and approximately 25% appear to be moderate-severe (CDC, 2015; Chiaravalloti, & Goverover, 2016; Faul, Xu, Wald, & Coronado, 2010). Currently, more than 5 million US citizens live with chronic TBI-related disabilities (Chiaravalloti, & Goverover, 2016; Corrigan, Selassie, & Langlois, 2010). TBI is now considered a long-term condition with chronic and possibly progressive effects rather than a static condition following a short recovery phase (Bigler, 2013; Chiaravalloti, & Goverover, 2016; Green et al., 2014; Masel, & Dewitt, 2010). Research demonstrated that patients with moderate-severe TBI experience significant neuropsychological impairment (Chiaravalloti, & Goverover, 2016; Constantinidou, Thomas, & Robinson, 2008a; Green et al., 2014; Konstantinou et al., 2016), including executive dysfunction and more specifically deficits in self-awareness (Caldwell et al., 2014; Chiaravalloti, & Goverover, 2016). In addition, neuroimaging studies revealed that individuals with TBI present with brain volume loss (Bendlin et al., 2008). Such brain atrophy presents a chronic course along with the neuropsychological impairment (Green et al., 2014; Greenberg, Mikulis, Ng, Desouza, & Green, 2008; Konstantinou et al., 2016). This chronic course may explain the poor quality of life outcome and lingering neuropsychological deficits, such as the lack of self-awareness often observed in moderate-severe TBI (Chiaravalloti, & Goverover, 2016). Hence, it is hypothesized that there is an association between these constructs (neuropsychological deficits, including lack of self-awareness, quality of life, and brain atrophy) in chronic moderate-severe TBI.

Specifically, it is expected that brain volume loss, mainly in the frontal areas, will associate with greater deficits in executive functions and in self-awareness. Also, greater impairment in self-awareness is expected to relate to higher levels of self-reported quality of life. Research has shown that impaired self-awareness affects one's inability to acknowledge injury-related deficits. This difficulty may render brain injury survivors unable to comprehend the effects of these deficits on their quality of life (Sasse et al., 2013). As a result of these two relationships, it is expected that greater brain atrophy will associate to higher levels of subjective QOL.

CHAPTER 2

LITERATURE REVIEW

Neuropsychological Deficits

It has been well-documented that neuropsychological deficits exist following traumatic brain injury (TBI; Bach, & David, 2006; Chiaravalloti, & Goverover, 2016), in both individuals with mild and moderate-severe injury (e.g. Dikmen et al., 2009; Frencham, Fox, & Maybery, 2005; Ord, Greve, Bianchini, & Aguerrevere, 2010). Such deficits include cognitive impairment (i.e. memory, attention, speed of processing), and executive dysfunctions, including deficits in self-awareness (Chiaravalloti, & Goverover, 2016; Felmingham, Baguley, & Green, 2004; Ghajar, & Ivry, 2008; Kinnunen, et al., 2011; Kraus et al., 2007; Lipton, et al., 2009; Lux, 2007; Prigatano, 2005; Rabinowitz, & Levin, 2014).

Theoretical Framework for Executive Functions and Self-awareness

Executive functions (EF) have been defined as "integrative cognitive processes that determine goal-directed and purposeful behavior and are superordinate in the orderly execution of daily life functions, which includes the ability to formulate goals; to initiate behavior; to anticipate the consequences of actions; to plan and organize behavior according to the spatial, temporal, topical or logical sequences; and to monitor and adapt behavior to fit a particular task or context" (Cicerone et al. 2000, p. 1605). Stuss (2011) proposed a hierarchical, interrelated framework where EF are positioned as the middle component of the model, receiving information from both lower (i.e. memory of historical events) and higher level (i.e. beliefs about the "self") processes. The results of this middle component are redistributed to both higher and lower processes (e.g. SA or memory), thus updating them.

Other theoretical perspectives with a more clinical focus consider EF as part of the metacognitive system, with self-regulation skills placed at the core of EF (Hacker, Dunlosky, & Graesser, 1998; Kennedy, & Coelho, 2005; Kennedy et al. 2008; Pressley, & Ghatala, 1990; Reder, 1996; Swanson, 1999). *Metacognition,* or thinking about thinking is conceptualized as the application of self-regulation to cognition, referring to one's ability to observe and assess more basic cognitive processes. This ability includes "self-awareness or metacognitive beliefs as well as self-monitoring and self-control of cognition while performing an activity" (Fitzgerald, Arvaneh, & Dockree, 2017; Kennedy, & Coelho, 2005; Shimamura, 2000). Thus, metacognition is present whilst engaging in highly complex behavior, including goal setting, self-monitoring, self-control, and strategy execution. Metacognition is often applied when engaging in daily routines which despite often being automatic, one may require performing rather differently in some cases (Fitzgerald et al., 2017; Kennedy, & Coelho, 2005; Nelson, & Narens, 1990; Rabinowitz, & Levin, 2014). It is in those cases when one is required to detect the difference, monitor their performance as a result of this change, make a strategy decision, and proceed to its execution (Kennedy, & Coelho, 2005). One's beliefs about their own cognitive abilities may influence their willingness to accept and eventually employ the alternative strategy. Therefore, the ability to solve everyday problems results from a dynamic relationship between a set of skills, such as the byproducts of self-regulation or an executive function system (Kennedy, & Coelho, 2005).

Executive functions have been referred to as a multidimensional construct, with various frameworks providing different definitions, and thus ways in assessing this construct. Constantinidou et al. (2012) suggest a multi-component framework to assess EF based on 3 main domains, equivalent to the ones suggested by the WHO-ICF: (i) Planning and initiation, (ii) Maintenance and flexibility, and (iii) Regulation and effective performance. In this

framework various factors were taken into consideration that could facilitate or impede with recovery. The authors proposed that standardized tests, despite deemed effective in detecting executive dysfunction, may lack predictive ability regarding the effect of EF deficits on daily function, especially in TBI, with impaired EF presenting in a wide spectrum of covert to evident difficulties. Therefore, a comprehensive assessment of EF is required, suggesting that EF skills cannot compartmentalized.

As an effect, it is also worth mentioning that one of the limitations in using EF tasks is that each individual task cannot assess just one specific skill of EF, without tapping into various aspects of EF or other cognitive processes. Specifically, tasks such as the Wisconsin Card Sorting Task (WCST) or the Trail Making Test B (TMT B) can cut across different domains of EF abilities. For example, the TMT B requires the implication of one's working memory, aside from EF skills. Therefore, in cases like TBI, an individual may present with difficulty in remembering the sequence in the TMT B, which will lead to them underperforming in such a task, thus highlighting the necessity of other cognitive processes whilst engaging in EF tasks. Hence, one should not differentiate between the skills present whilst engaging in EF tasks, or attempt to isolate such skills when measuring EF performance.

Therefore, in this study multiple tests assessing executive dysfunction were employed in this study, including the Trails Making Test B, the Symbol Digits Modalities Test, the COWAT, and the Rey Figure copy score, along with contextual behavioral measures such as the DEX-R in order to improve the ecological validity of the EF assessment battery.

Research on the dynamic relationships associated with EF has yielded a number of considerations. One such issue describes a paradoxical phenomenon known as *discounting, or fan effect* (Anderson, & Reder, 1999; Kennedy, & Yorkston, 2004; McGuire, & Maki, 2001).

Discounting specifically refers to instances when individuals having sustained a TBI and greater mnemonic deficits provide higher prediction ratings of future recall, thus being overconfident in their abilities. Additionally, McGuire and Maki (2001) have highlighted that those who can handle more information in their working memory, they will provide lower predictions in their ability for future recall. This phenomenon is often met in individuals with moderate-severe TBI, and more specifically to those presenting with deficits in metacognition and SA (Sherer, Hart, Whyte, Nick, & Yablon, 2005; Kennedy, & Yorkston, 2004). Although not all individuals with moderate-severe TBI will experience SA deficits (Prigatano, & Altman, 1990), for some these difficulties may persist over time (Hoofien, Gilboa, Vakil, & Barak, 2004).

Therefore, the presentation of impaired EF and thus SA as documented in individuals with TBI may "include difficulty starting or initiating, stopping or inhibiting, shifting, and adjusting purposeful behavior; overconfidence or under-confidence in beliefs about their skills; and impaired self-monitoring or self-control during activities" (Kennedy, & Coehlo, 2005, p. 245). As a result, individuals with low SA who appear overconfident about their skills, tend to overestimate their QOL (Sasse et al., 2013). Such discrepancies are mainly recorded using the discrepancy ratings provided by the individual with TBI and an informant's ratings (i.e. clinicians, families, and significant others) of the individual's cognitive, emotional, and mobility abilities following the injury (Sasse et al., 2013).

It has been argued that individuals who are more aware of their strengths and weaknesses are less likely to engage in daily activities that will potentially lead to failure, and thus subsequent disappointment and distress (Chiaravalloti, & Goverover, 2016; Doig, Fleming, & Tooth, 2001). Smith, Magill-Evans and Britnell (1998) further add to this argument by proposing that individuals able to better understand their need for social support

may perceive their life as more gratifying. Thus, impaired SA following TBI could impede the individual's motivation to engage in rehabilitation, hampering rehabilitation efforts and reducing rehabilitation effectiveness (Fischer, Gauggel, & Trexler, 2004; FitzGerald, Carton, O'Keeffe, Coen, & Dockree, 2012; Malec, & Moessner, 2000; Ownsworth, & Fleming, 2005; Tate et al., 2014; Turner, Ownsworth, Turpin, Fleming, & Griffin, 2008). Therefore, SA after TBI has been gaining attention in the past 20 years. Prigatano (2005) argues that individuals with TBI who under-report evident cognitive and behavioral difficulties may present with greater psychiatric symptomatology (Chiaravalloti, & Goverover, 2016; Smeets et al., 2014), whereas individuals with TBI with a more realistic perception of their deficits, were reported to have fewer psychopathological symptoms, better neuropsychological function and greater independence (Noe et al., 2005; Rabinowitz, & Levin, 2014; Smeets et al., 2014;). Individuals with impaired SA may present with poor decision-making, greater difficulty adapting to change or new events, and interpersonal difficulties (Bach & David, 2006; Chiaravalloti, & Goverover, 2016; Fitzgerald et al., 2012; Newman, Garmoe, Beatty, & Ziccardi, 2000; Wood, 2008). These findings allow for a speculation regarding the role of SA and related processes into QOL outcome in TBI.

A number of models have been developed in an attempt to explain the phenomenon of impaired SA following TBI, with no one single model being able to provide a thorough explanation of its neuropsychological underpinnings (Kennedy, & Coehlo, 2005; McGlynn, & Schacter, 1989; Stuss, 1991; Toglia, & Kirk, 2000). According to cognitive neuropsychologists, impaired SA is conceptualized based on the dissociable interaction and conscious experience model (McGlynn, & Schacter, 1989; Schacter, 1990) and the hierarchy of brain function model (Stuss, 1991; Stuss, & Benson, 1986; Stuss, Picton, & Alexander, 2001). This conceptualization supports that impairment to "neural systems responsible for

detection, processing and information control" may be responsible for specific deficits in SA; thus, impaired SA can be detected given that damage occurs to either the EF system itself or to its communication to other cognitive processes (Caldwell et al., 2014, p.54). For example, when individuals with TBI appear to be aware of their deficits, it is hypothesized that the damage is more localized to the executive system rather than the connection of the executive system to other cognitive systems (Caldwell et al., 2014). These models conclude that there is a close relationship between the EF system and SA, highlighting the fact that for SA to be intact the EF should also be unimpaired (Caldwell et al., 2014).

A study measured three aspects of SA (metacognitive knowledge, online emergent awareness, and online anticipatory awareness) in a sample of participants with mild to severe TBI. A number of tests were used including questionnaires of awareness interviews and the discrepancy scores between the patient's and a significant other, and an online error-monitoring task (O'Keeffe, Dockree, Moloney, Carton, & Robertson, 2007). Findings showed that the participants with greater deficits in SA reported greater difficulty in exhibiting disinhibition, interpersonal problems, and more total competency, supporting an association between SA and metacognitive knowledge (Fitzgerald et al., 2017). Such interrelation of executive functioning with emotion, denial, motivation, and metacognition allows for the development of biopsychosocial models of SA (Ownsworth et al. 2007), and is suggestive of a multidimensional approach in capturing any impairment in SA (Fitzgerald et al., 2017).

Cognitive neuropsychology contributes to our understanding of this phenomenon through the identification of typical executive dysfunction correlating with deficits in SA. Individuals with greater impairment in basic problem-solving and concept formation also showed greater deficits in SA (Ownsworth & Fleming, 2005). Furthermore, lower levels of SA have been associated with a decrease in the patient's performance in verbal fluency

(Burgess, Alderman, Evans, Emslie, & Wilson, 1998; Zimmermann, Mograbi, Hermes-Pereira, Fonseca, & Prigatano, 2017), cognitive flexibility, and concept formation tasks, but not in basic attention and memory tasks (Burgess et al., 1998). Finally, Morton and Barker (2010) reported that implicit abilities relate with on-line awareness. Therefore, it has been proposed that impaired SA is supported and maintained via deficits in the executive processes and their interaction with cognitive processes (Hart, Whyte, Kim, & Vaccaro, 2005; Zimmermann et al., 2017).

In summary, according to the aforementioned studies there is a consensus on how executive dysfunction may be predictive of or relate to impaired SA (Giacino, & Cicerone, 1998; Zimmermann et al., 2017). This relationship is of great value in designing treatment programs aiming to improve SA deficits and potentially the patient's QOL, as well as informing cognitive neuropsychological models.

Quality of Life (QOL)

The relationship between SA and QOL has been investigated, indicating that participants with TBI exhibiting lower levels of SA tend to report greater levels in their QOL, mainly regarding their cognitive abilities (Sasse et al., 2013; Sherer et al., 1998). Such findings have been conceptualized by the discounting phenomenon described previously in this document. However, few studies have comprehensively studied this phenomenon using appropriate measures in recording QOL.

In 2001, the WHO published a new classification system of health and health-related domains, known as the International Classification of Functioning, Disability, and Health (ICF). The intention was to create a universal classification system, not just for individuals with TBI or other disabilities, but for the general population as well. The ICF represents a

"biopsychosocial" approach to health, functioning, and disability, to provide "a coherent view of different perspectives of health from a biological, individual and social perspective" (WHO, 2001, p. 28). The WHO is encouraging its application as a framework for social policy, research, education, and clinical practice, e.g. can be used to design one's assessment. There are two *contextual* factors that may impact a person's health state, one describing *environmental* factors such as physical, social, cultural, or institutional in nature that may include the availability, or quality. The second one refers to *personal factors*, such as gender, age, education, and lifestyle. These two contextual components influence other factors of the disease, and are recorded and considered as contributors to the dimensions of *body function/structure, activity, and participation* (WHO, 2001).

Moderate-severe TBI results in significant morphological and neuropsychological impairments and the effects of the deficits are evident years post injury, leading to further long-term impairments and disabilities in functional, emotional, and social domains (Chiaravalloti, & Goverover, 2016; Dikmen, Machamer, Miller, Doctor, & Temkin, 2001; Shukla, Devia, & Agrawal, 2011). Although the mortality rate, following TBI has decreased, no reduction in disability rates has been noted (CDC, 2015; Dikmen et al., 2001; Masel, & Dewitt, 2010). Due to the complexity of the term disability, a number of measures have been created in an attempt to capture this phenomenon in the TBI population, including QOL or functional outcome instruments. Some examples of frequently used functional measures are the Glasgow Outcome Scale (GOS), GOS Extended (GOSe), Disability Rating Scale (DRS), Functional Independence Measure (FIM), and Functional Assessment Measure (FAM) (Shukla et al., 2011). Although such measures allow capturing the functional problems faced by individuals with TBI, they cannot portray the patient's subjective experience of their disability, something that QOL measures do (Shukla et al., 2011).

Quality of life refers to "[a]n individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards, and concerns. It is a broad ranging concept affected in a complex way by the person's physical health, psychological state, level of independence, social relationships, and their relationship to salient features of their environment" (WHOQoL Group, 1993, p. 153). These objective circumstances are experienced in various ways by different people (Dijkers, 2004; Mailhan, Azouvi, & Dazord, 2005). In the past 15 years, QOL has been introduced as an outcome criterion after TBI (Corrigan, Bogner, Mysiw, Clinchot, & Fugate, 2001) with functional outcome, return to work, and productivity already explored. Traumatic brain injury can disrupt one's quality of life (Sasse et al., 2013). More specifically, survivors have reported behavioral changes that include difficulty in maintaining personal relationships, coping with work, and community-related activities (US National Institutes of Health, 1998). The aforementioned definition clearly depicts the key factor in QOL, such as the individual's perception of their functioning. Due to the fact that QOL is a broad concept and may be affected by various aspects, a new term has been recently introduced: the *Health-Related Quality of Life* (HRQL). Health-related quality of life reflects an individual's perception of how their own condition and its treatment affect the physical, mental, and social aspects of his or her life (Nichol et al., 2011).

Studies investigating QOL post TBI have yielded mixed results as individuals either over- or under-report the consequences of TBI on their QOL. For example, individuals with TBI have reported lower levels regarding their QOL compared to healthy controls (Dijkers, 2004; Mailhan et al., 2005; van Baalen, Odding, & Stam, 2008). Specifically, 91% of all patients with severe TBI reported a reduced QOL after 1 year (van Baalen et al., 2008). However, in the study of Engberg and Teasdale (2004), 94-95% of the participants found their

life as good or at least acceptable with only 5-6% reporting finding it hard to bear. Pickelsimer, Selassie, Gu, and Langlois, (2006) reported that one year after TBI, 65% of the recruited patients declared satisfaction with their lives. In a more recent study, only 17% of the participants having sustained severe TBI reported poor QOL, leaving a 73% of the participants with TBI reporting satisfaction with their lives (Anderson, Brown, & Newitt, 2010). As a whole, central tendencies and distributions of life satisfaction seem to be consistent during many years after TBI (Corrigan et al., 2001; Johansson & Bernspång, 2003; Pagulayan, Temkin, Machamer, & Dikmen, 2006). In a cohort study Hawthorne, Gruen, and Kaye (2009) reported that participants with TBI experienced worse general health, social isolation, worse labor force participation rates, as well as worse health status. The most affected areas were social function, role emotion, and mental health, suggesting that TBI has long-term consequences across all aspects of peoples' lives. Thus, the challenge following a brain injury is to comprehensively evaluate and sufficiently provide long-term services targeted at the life areas that individuals with TBI find particularly difficult.

Quality of life has been significantly linked to injury severity (Wood, & Rutterford, 2006). However, these findings are also mixed. TBI severity is most commonly measured by the Glasgow Coma Scale (GCS; Teasdale, & Jennett, 1974). The GCS allows assessing three parameters: eye opening, speech, and motor response. A GCS score of 3–8 indicates a severe injury, 9–13 a moderate injury, and 14–15 a mild injury. Although mild to moderate injuries can lead to serious disability, this outcome may be influenced more by the extent and quality of rehabilitation than by acute medical management. In contrast, patients with a severe head injury more commonly require prolonged intensive and specialist medical treatment. Injury severity has been shown to be predictive of life satisfaction, relationship status, community integration, and employment status (Wood, & Rutterford, 2006; Teasdale, & Engberg, 2005).

Anderson, Brown, and Newitt (2010) have shown that lower levels of QOL more likely occur with greater injury severity and lower levels of perceived independence. Individuals experiencing the after-effects of severe TBI (5-8 years following trauma) reported that their QOL had deteriorated since their injuries (Schalén, Hansson, Nordstrom, & Nordström, 1994). TBI severity was also associated with a significant risk of reporting injury-related problems at survey completion (Brown et al, 2011).

Although studies support a negative relationship between QOL and TBI severity, there is also evidence pointing to no association between these two constructs at all (Dikmen, Machamer, Powell, & Temkin, 2003; Lin et al., 2010; van Delft-Schreurs et al., 2013). In a longitudinal study, although patients reported an improvement on all domains of QOL as measured by the WHOQOL-BREF (WHOQoL group, 1993), except for social relationships, over the first six and 12 months post injury, none of these domains indicated any significant association with injury severity (Lin et al., 2010). Additionally, evidence indicates an association among TBI severity, functional outcome, and employment status, rather than QOL (Andelic et al., 2009; Corrigan et al., 2001). According to the findings of van Delft-Schreurs and colleagues (2013), QOL depends on two more factors such as pre-accidental comorbidity and living alone, rather than injury severity. Dikmen and colleagues (2003) measured the outcome at three to five years following the injury of individuals with moderate-severe TBI. Their results also indicated that brain injury is related to neuropsychological and functional outcomes post injury, but not to injury severity.

Furthermore, there is evidence suggesting an anomalous relationship between injury severity and QOL, as patients with the most severe injuries reported higher levels of life satisfaction one or more years after injury (Brown, & Vandergoot, 1998; Corrigan et al., 2001). In the study of Corrigan and colleagues (2001) subjects noting the lowest GCS score

reported slightly higher life satisfaction than other groups a year post injury. This adverse event may be explained by a lack of insight, or impaired SA, of the patients' own disabilities. As discussed above, participants with TBI of greater severity may be less aware of their disabilities and thus rate their QOL as being higher.

In conclusion, evidence suggests that further investigation is required in order to better comprehend the association among neuropsychological performance, SA, and QOL. Mixed findings may result from: 1) the discounting phenomenon (i.e. impaired SA), 2) the lack of measures specific to TBI when measuring QOL, in individuals with TBI, 3) the time since injury, as it remains unclear whether SA deficits improve or persist in chronic TBI and, 4) the severity of the injury, with some individuals with a more severe injury presenting with greater SA impairment, whereas others do not (see following subsection). The present study will account for these prior pitfalls by measuring SA, and by incorporating TBI-related measures of QOL, in chronic moderate-severe TBI.

QOL & Neuropsychological Deficits

Mixed findings exist on the predictive ability of neuropsychological tests on QOL. Klonoff and colleagues reported that individuals with TBI, who scored higher on tasks of motor functioning, memory, and constructional ability, also reported higher levels of QOL, at two to four years following the injury (Klonoff, Costa, & Snow, 1986). In addition, Wood and Rutterford (2006) reported that only working memory could predict satisfaction with life, at one year post injury. It seems that individuals with working memory deficits, also experience low perception of their ability to handle daily situations effectively which was further associated with lower levels in life satisfaction. They also revealed that a sense of self-efficacy mediated the relationship between working memory and QOL (Wood, & Rutterford, 2006). Esbjörnsson, Skoglund and Sunnerhagen (2013) reported a correlation among cognitive tasks

and attention deficits, SA of executive impairment, lack of motivation, mood disturbances, and HRQOL. Health-related quality of life was associated with the perception of general consequences of the trauma, with the inability to manage one's own hygiene and major activities being considered as the greater disability. Individuals with TBI reporting higher levels of QOL also indicated better cognitive function and attention, perceived themselves as more motivated, reported less difficulty in planning, as well as less depressive symptomatology and social isolation, than those with lower levels of QOL. In addition, participants with TBI who depended upon others to manage their personal hygiene and daily activities were more likely to report greater consequences as a result of trauma. Those with fewer mobility issues had better cognition than the more disabled patients.

On the contrary, Hanks and colleagues (2008) investigated the predictive validity of a brief neuropsychological test battery on functional outcome in individuals with TBI one year following the injury. All neuropsychological measures predicted handicap, functional outcome, supervision needs, and vocational outcome, but not satisfaction with life, as measured by Satisfaction With Life Scale (SWLS).

However, this incongruence between one's performance in neuropsychological tasks and QOL may be accounted by the ICF model and the need for contextual assessment. For example, a mild word finding problem may not be important in a computer programmer's QOL, but may significantly interfere with the work of an attorney.

QOL, SA, and Injury Severity

Although injury severity and functional status at time of hospital admission are generally considered good predictors across most of the domains of outcome assessed, neuropsychological measures may provide us with greater predictive validity with measures of sensorimotor responsiveness such as the GOSe than injury severity, during the post-acute

phase (Hanks et al., 2008). In addition, the predictive validity of injury severity on impaired SA remains unclear (Sasse et al., 2013), with some studies demonstrating that participants who had sustained a more severe injury overestimated their abilities (Holm, Schonberger, Poulsen, & Caetano, 2009; Prigatano, 2005; Prigatano, Borgaro, Baker, & Wethe, 2005); whereas other research failed to find such association (Sawchyn, Mateer, & Suffield, 2005).

Despite the complexity of this relationship between SA deficits and injury severity, it seems that individuals who have sustained a more severe injury and present with greater SA impairment, may overestimate their abilities (Seel, Macciocchi, & Kreutzer, 2010); therefore, further complicating the relationship between SA and HRQOL (Sasse et al., 2013). Poor SA has been described as a significant barrier to social integration, and as a result it is considered a significant predictor of poor psychosocial outcome and employment (Chiaravalloti, & Goverover, 2016; Sherer et al., 2005). Social integration requires the implication of intact EF, and thus intact SA (Chiaravalloti, & Goverover, 2016). Sasse and colleagues (2013) explored the association between SA and HRQOL in participants with severe, moderate, mild, and complicated mild TBI. Findings revealed that individuals experiencing greater SA deficits reported greater HRQOL in the cognitive and leisure activities domains. Such evidence is further supported by Mathias and Wheaton (2007) reporting that participants with TBI are less likely to be aware of changes in behavior and EF than changes in more concrete domains, such as motor function. Goverover and Chiaravalloti (2014) have also reached to a similar conclusion for individuals with higher SA who reported lower levels of HRQOL, but these results were mediated by higher levels of depressive symptoms and memory deficits. On the other hand, there is evidence suggesting that SA has no implication in reporting QOL. Specifically, evidence has shown that individuals with TBI who presented with low SA,

reported less satisfaction with their HRQOL, than participants with TBI with adequate SA (Formisano et al., 2017).

It may be concluded that no clear evidence exists to describe how injury severity and its association to QOL may be affected by impairment in SA. It has been reported that recovery of SA can also increase the risk for depression, due to a realization that expectations about recovery are not being met (Fleminger, Oliver, Williams, & Evans, 2003; Zimmermann et al., 2017).

Brain Morphology & TBI

Pathophysiologic sequelae exists in individuals with TBI, including the location and severity of the damage, diffuse effects, and secondary mechanisms of injury, leading to focal and diffuse brain injury. Contusions can directly disrupt function in both cortical and sub-cortical regions. “Upon contact, the individual may sustain a focal injury at the site of lesion (coup) or at a site distant to the lesion (contrecoup) and inertial loading caused by acceleration-deceleration forces, resulting in multifocal and diffuse lesions” (Constantinidou, & Kennedy, 2012b, p.366). Certain brain regions may be more vulnerable to contusion following trauma, such as the frontal and anterior temporal cortices, due to their position within the skull (Blennow, Hardy, & Zetterberg, 2012; McAllister, 2011; Povlishock, & Katz, 2005).

Disruption of function can also result from more diffuse damage to white matter tracts that are particularly susceptible to the shearing forces that often occur with TBI (Graham, Gennarelli, & McIntosh, 2002). Such diffuse axonal injury (DAI) can disrupt critical cortical-subcortical pathways and lead to widespread cognitive dysfunction, as seen in the review study of Levine and colleagues (2006) reporting both white and gray matter loss in individuals with moderate-severe TBI. Diffuse axonal injury can result directly from the trauma, or as a

secondary effect due to ischemia. Brain edema and shift can compromise blood supply and lead to secondary infarction in the corpus callosum and deep grey matter, while elevated intracranial pressure can cause damage to the brainstem in TBI (Graham et al., 2002).

The brain area described as more sensitive to such morphological changes is the hippocampus (Blennow et al., 2012; Katz, 1992; Povlishock, & Katz, 2005). In addition, greater injury severity has been associated to an increase in pathophysiology, as identified on MRI scans. For example, chronic moderate-severe TBI has been related to atrophy in the corpus callosum (Mathias et al., 2004; Tomaiulo et al., 2005). Also, patients with chronic severe TBI presented with an increased atrophy in the fornix, anterior limb of the internal capsule, superior frontal gyrus, parahippocampal gyrus, optic radiations and optic chiasma (Tomaiulo et al., 2005).

The relationship between severity of injury and impaired SA is not a linear one. Damage may develop over time after the initial injury, or indeed large, seemingly acute, hematomas may have little impact on SA. Sherer, Hart, Whyte, Nick, and Yablon (2005) found that the number of brain lesions rather than the volume or location of the lesions was predictive of the degree of impaired SA in a sample of 91 participants with acquired brain injury. Furthermore, diffuse axonal injury affecting broadly distributed networks may have a greater effect on SA than focal lesions.

Brain Abnormalities, Executive Functions, and SA

Literature has come to a consensus concerning the implication of the frontal lobes in the EF, SA, and thus one's ability to function adaptively. However, Prigatano (1991) also highlights the involvement of other brain areas and circuits in effectively employing behaviors such as planning, coordinating and monitoring of behavior. These neurocircuits have been

described in detail by Constantinidou, Wertheimer, Evans, Tsanadis and Brown (2012c), arguing their implication in the relationship of the frontal lobes and EF, through transferring information generated by the cortex and moving this towards subcortical regions, such as the globus pallidus, and the thalamus, and back to the cortex. The first two circuits involve mainly motor functions; whereas the rest are implicated in cognitive and behavioral functions: the dorsolateral prefrontal circuit (DLPFc), the orbitofrontal circuit (OFc), and the anterior cingulate circuit (ACc). Similar behavioral patterns may be produced as a result of damage to these circuits, as the basal ganglia have been linked to the cortex through these circuits (Constantinidou et al., 2012c).

Specifically, damage to the DLPFc is accompanied by greater deficits in organizational skills, greater difficulty in shifting attention, and environmental dependency. Such symptoms have been linked to executive dysfunction, and thus the DLPFc has been described as the circuit relating mostly to EF. Personality changes, including irritability, lack of empathy, and inappropriate social behavior, following a TBI have been attributed to impairment within the OFc. Despite such deficits neural damage to the OFc, individuals may still perform adequately on EF tasks. Finally, damage to the Acc has been reported to result in lack of motivation and apathy, including akinetic mutism, poor response inhibition, minimal creative thinking, and difficulty in producing spontaneous speech (see Constantinidou et al., 2012c).

In a study investigating the relationships between regional variation in grey matter volume and cognitive impairment in individuals with mild to severe TBI, evidence supported the association between the two, whereby the participants with most severe TBI displayed the most significant impairment (Spitz, Bigler, Abildskov, Maller, O'Sullivan, & Ponsford, 2013). Specifically, patients with lower scores on executive control performance indicated lower cortical volume in temporal, parietal, and occipital regions. In an fMRI study employing the

use of the single-trial Stroop and Auditory Consonant Trigrams in measuring selective attention and cognitive flexibility in severe TBI, it was suggested that atrophy of the right cACC may contribute to reduced performance on EF tasks (Merkley, Larson, Bigler, Good, & Perlstein, 2013).

Therefore, it may be argued that behavioral deficits relating to damage to the EF system may result from damage to the frontal and prefrontal regions, as well as to the cortical and subcortical regions connected to this system (Constantinidou et al., 2012c). As an effect, researchers have focused on further exploring the underlying networks guiding such behavior.

Similar brain areas and connections have been found to relate to impaired SA (Prigatano, 1991; Taylor, Stern, & Gehring, 2007). In a review article, the authors posit for the implication of two circuits in a number of processes depicting SA: 1) “the dorsolateral prefrontal circuit (responsible for self-regulation, self-monitoring, and other EF) includes links to the basal ganglia, thalamus and prefrontal cortex”, and 2) “the lateral orbitofrontal circuit (responsible for empathic and socially appropriate responses) includes links to the basal ganglia, thalamus, and orbitofrontal cortex” (see review FitzGerald, Carton, O'Keeffe, Coen, & Dockree, 2012). Evidence has shown the implication of the anterior cingulate cortex (ACC) in procedural learning and behavior modification through transferring reinforcing stimuli to diffuse areas of cortical and subcortical regions. In addition, O’Connell and colleagues (2007) have reported the implication of the ACC in error diagnosis and detection, and conflict processes. Such processes are enabled when one is called to respond correctly, at the presence of competitive stimuli. The aforementioned processes signal self-monitoring and -regulation abilities, which directly relate to emergent awareness. Taylor and colleagues (2007) support this evidence and highlight the involvement of a number of neural regions in error awareness, such as the dorsal ACC, rostral ACC, posterior medial frontal cortex, anterior medial frontal

cortex, and prefrontal cortex, which allows for the development of adaptive behaviors, that in turn further strengthen global awareness (O'Keeffe et al., 2007).

In conclusion, these findings clearly support the complexity of the relationships among the different areas and neural circuits underlying impaired SA. In spite all the evidence supporting the existence of an association among neuropathology, EF, and SA and the relationship between SA and QOL, no evidence exists to describe the relationship between neuropathology and QOL. Such information will further enhance rehabilitation experts in designing the best possible treatment for individuals having sustained a brain injury.

Project Innovation & Originality

Evidence revealed mixed findings concerning the relationship between neuropsychological deficit and QOL in individuals with TBI, with studies showing either an increase or decrease in cognitive performance relating to lower level of QOL. One plausible explanation for inconsistencies in previous research may be attributed to the notion stating that survivors of moderate-severe TBI often do not acknowledge the extent of their injury in their daily life functioning, due to cognitive difficulties in recognizing the full range of their deficits resulting from the injury. However, the relationship between neuropsychological performance and QOL may be influenced by the underlying brain morphology. The lack of research associating brain morphology with QOL does not allow for any direct inferences to be made. Therefore, the current study attempts to associate brain morphology with QOL measures and provide insight in this gap met in current TBI literature.

Primary Research Hypotheses

It is expected that 1) greater brain atrophy in the frontal lobes and associated structures such as the prefrontal and temporal cortex, the ACC, the thalamus, the putamen, the insula, and the

caudate as measured by MRI, will positively correlate to EF deficits, including impaired SA; 2) participants with moderate-severe TBI will present with greater executive dysfunction, including SA, as compared to non-injured controls; 3) QOL is expected to correlate negatively to SA deficits as measured by neuropsychological performance (moderate-severe deficits at time of study participation). Finally, it is expected that 4) greater degree of brain atrophy in cortical and sub-cortical brain areas relating to EF and SA will correlate with higher levels in QOL in participants with TBI.

CHAPTER 3

METHODOLOGY & PROCEDURE

Participants

Power analysis and sample size estimation. In Konstantinou et al. (2016) a sample of 32 participants yielded an effect size of Cohen's d = 0.85, for group differences, and an effect size of r = 0.32 and greater, for correlational analyses. The G*Power software was used to further validate this argument. According to G*Power, 31 participants per group was proposed for group comparisons for a Cohen's d of greater than 0.85; and for correlation analyses, and an effect size of r = 0.80 and higher, the sample size should consist of 34 participants minimum.

Group with TBI

The group of participants with TBI consisted of 33 Greek-speaking individuals with an age range of 18–51 years old with a primary diagnosis of moderate-severe closed head injury (CHI), as opposed to an open or penetrating head injury at least two years post injury. Moderate-severe brain injury was determined by three or more of the following indices: (i) initial Glasgow Coma Scale score of less than 12, (ii) abnormal initial computed tomography (CT) or MRI findings indicating acute central nervous system pathology, (iii) length of impaired consciousness greater than 20 min as specified by the emergency records, (iv) length of acute hospital stay longer than three days, (v) length of posttraumatic amnesia (PTA) greater than 24 hours as specified in the acute emergency records, (vi) positive neurological examination on hospital admission and discharge indicating focal sensory and motor neurological deficits, or changes in the mental status attributed to brain injury, and (viii) head injury severity classifications according to hospital records. In addition, individuals with TBI

should have scored a Level VI or higher on the Rancho Los Amigos Scale, indicating appropriate, goal-oriented behavior, and post-traumatic amnesia (PTA) resolution. No aphasia was present with the exception of mild to moderate word finding problems due to cognitive deficits.

Exclusion criteria included (i) penetrating head injuries, (ii) a diagnosis of stroke at the time of injury, (iii) uncorrected visual deficit or hearing impairment affecting speech comprehension, (iv) a premorbid central nervous system disorder or learning disability, (v) a premorbid psychiatric disorder as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (commonly referred to as DSM-V), that resulted in hospitalization, incapacity to work, or to perform activities of daily living, and (vi) an active or current alcohol, drug, or other controlled substance abuse that interfered with participation. The above inclusion/exclusion criteria for participants with TBI are consistent with the Constantinidou and colleagues, 2005 and 2008a criteria.

Control group

The non-TBI group consisted of 24 healthy Greek-speaking volunteers who were pair-matched to the individuals with TBI on gender, age, and education, with a variance of plus or minus 2 years. The non-injured individuals had no history of TBI or any other neurological condition. Participants with uncorrected vision or hearing, color blindness, psychiatric disorder, substance abuse, and learning disability were excluded from the study.

Procedure

Participants with TBI were recruited from collaborating physicians, the Intensive Care Unit database, and the Melathron Agoniston EOKA database, using the rolling admission process for this study. Patient identification and screening process occurred; with the patients'

referring physicians identifying potential study participants on the basis of the inclusion/exclusion criteria. The control group consisted of volunteers recruited and matched on critical variables. Participants meeting the inclusion/exclusion criteria for this project were referred to the investigator. The project manager contacted and informed the participants about the study procedure. The study was divided into three phases: neuroimaging, neuropsychological assessment, and questionnaires assessing quality of life. All study procedures were approved by the Cyprus Bioethics Committee and a consent form was obtained from each participant.

Funding for this project was received from the Cyprus Research Promotion Foundation through a grant co-funded by the Cyprus Government and the European Regional Development Fund (FC, PI; NEW INFRASTRUCTURE/STRATEGIC/0309/37). The data collection project consisted of three phases and the doctoral researcher and a research assistant had conducted the 2 phases of the experiment (i.e., the neuropsychological and psychosocial assessment). Imaging data were obtained by the Medical Diagnostic Center personnel and a copy of two CDs and a clinical report were provided to the doctoral researcher. A CD and the clinical report were given to the participants; whereas the second CD was used for the data analyses. Also, a copy of the report was kept in each participant's file. The study adhered to data protection and privacy procedures as approved by the National Bioethics Committee and the Commissioner for Protection of Personalized Data, Republic of Cyprus.

All data collection procedures were completed in eight months. The data was collected in a laboratory setting, where both the neuropsychological and QOL measures were administered individually. The neuropsychological testing lasted for 1.5-2 hours per participant with TBI, and 1-1.5 hours for the control group participants. The QOL measures

took about 45 minutes for the TBI group and 10 minutes for the control group. The MRI data acquisition took about one hour per participant.

Materials

Neuropsychological Performance

This study was part of an ongoing research program which incorporates a comprehensive assessment of cognitive functions. Therefore, all participants underwent a battery of pen-and-paper neuropsychological tests sensitive to cognitive deficits associated with TBI. The test battery lasted for approximately 90 minutes and was completed in one session. During testing participants were provided with scheduled breaks in order to avoid mental fatigue. All tests were adjusted to Greek-native speakers. The cognitive domains of interest were investigated by using the following measures: (i) EF were assessed using the Rey Complex Figure Test (copy) (Rey, 1993), the Trail Making Tests A and B (also processing speed; Zalonis et al., 2008), the Symbol Digits Modalities Test (SDMT; Smith, 1982), and the phonological (letter F) and category recall (Animal recall) from the Control Oral Word Association Test (COWAT; Kosmidis, Vlahou, Panagiotaki, & Kiosseoglou, 2004); (ii) Verbal and visual memory was examined using the Digit Span Forward and Backwards and Visual Span Forward and Backwards (adapted Wechsler Memory Scale-III, WMS-III; Wechsler, 1997), the Greek adaptation of the Auditory Verbal Learning Test (AVLT; Constantinidou, & Evripidou, 2012a), the Rey Complex Figure Test immediate and delayed recall (Rey, & Osterrieth, 1993), the Greek Passage Memory test (which is based on the Wechsler Memory Scale Logical Memory subtest; Constantinidou, & Ioannou, 2008b); (iii) and Cognitive Reserve was assessed using the Peabody Picture Vocabulary Test (PPVT; Simos, Kasselimis, & Mouzaki, 2011) and a reading measure assessing the total number of

pseudowords correctly read in 45 s as measured by a test of pseudowords in Greek (Simos, Sideridis, Kasselimis, & Mouzaki, 2013).

The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005) was used as a cognitive screening tool with a cut off score of 26 and lower. The MoCA places more emphasis on tasks of frontal executive functioning and attention than the MMSE, which may make it more sensitive in detecting non-AD dementia. The original MoCA is a 30-point scale with 7 cognitive subtests including the alternating trail making, visuo-constructional skills, naming, memory, attention, vigilance, serial 7s, sentence repetition, verbal fluency, abstraction, delayed recall, and orientation tasks. The Greek version of the MoCA developed by Kounti and Tsolaki (2006) was used in this study.

Self-awareness

The **Dysexecutive Questionnaire** (DEX; Burgess, Alderman, Wilson, Evans, & Emslie, 1996) was used in this study to assess deficits in EF and SA. It is a 37-item inventory assessing cognitive, behavioral, emotional, and motivational problems associated with executive dysfunction in neuropathological groups, including individuals with TBI. Items are scored on a 5-point Likert scale (*0*–"*Never*" and *4*–"*Very often*"), with higher scores indicating more problems. According to Dimitriadou (2016), three factors can be extracted from this measure: (i) the Social and Self-Regulation subscale relating to the Orbitofrontal Circuit, assessing symptoms such as anger, knowing-doing dissociation, and social disinhibition; (ii) the Flexibility Fluency and Working Memory index measuring behaviors relating to the Dorsolateral Circuit, e.g. working memory and information processing deficits; and (iii) the Motivation and Attention factor associated with the Anterior Cingulate Circuit, depicting symptoms of apathy, poor planning and decision making. This measure was

completed by both the participants (DEX-R-S) and an informant (DEX-R-I), i.e., a significant other or a family member. Reliability analyses were conducted for each factor and version (self or informant) of the questionnaire, separately, and were very high (Cronbach's $\alpha > 0.90$; see Table 1, Appendix A).

The **Self-Regulation Skills Interview** (SRSI; Ownsworth, McFarland, & Young, 2000) was also conducted to further grasp the SA phenomenon. SRSI is a five-item semi-structured interview measuring emergent awareness, anticipatory awareness, strategy generation, strategy-use, and strategy effectiveness. The five items are scored on a 10-point scale. The scale was translated in Greek using the forward and backward method upon permission by the author. Three dimensions have been extracted from this interview (Ownsworth et al., 2000). Good inter-rater (r = 0.81-0.92) and test-retest reliability (r = 0.69-0.91) has been reported for this measure (Ownsworth et al., 2000). The same factors were produced using the data of this study. Two item scores were summed to provide an Awareness Index score (range 0-20) measuring emergent/anticipatory awareness of a behavioral problem identified by the participant (for example, memory or anger problems) and scored according to standard prompts. The remaining three items were combined to generate a Strategy Index score (range 0-30) measuring participant's awareness of any behavioral strategies they used with the identified problem(s). Again, high scores represented low levels of awareness. Reliability analyses, for this sample, for two of the three factors revealed a Cronbach's α of 0.93, for each scale. The third dimension, i.e. readiness for change, was not tested as it comprised of one item, only.

All of the QOL and the SA scales were completed by the TBI group, and a family member or a significant other, where appropriate. The MPAI-4 was completed by a family

member or a significant other, only. The control group was only administered the WHOQOL-BREF, and the DEX.

Quality of Life

According to Cieza and Stucki (2005), HRQOL and the ICF represent two different perspectives of functioning and health. Cieza and Stucki (2005) investigated the relationship between the ICF and 6 HRQOL instruments, including the SF-36, the WHOQOL-BREF, and the EQ-5D. Findings indicated that the ICF correlated with all 6 measures of HRQOL. All but 12 concepts (out of 226 concepts) linked to the ICF with kappa coefficients rising up to 0.98. The concepts derived from the items of the HRQOL measures were associated with categories of the component environmental factors, the component activities and participation, and the component body functions. For the component describing activities and mobility, only three of the measures covered mobility aspects. In addition, not all measures, with only of four out of six, attempt to examine environmental factors. Finally, for the body functions component only one function is universally captured, the "emotional functions". Therefore, they propose that both HRQOL and ICF related measures are used simultaneously for a thorough assessment.

The choice of instruments employed in this study attempted to follow the ICF concepts in order to thoroughly describe the HRQOL phenomenon. One of the measures has been examined in the aforementioned study, the World Health Organization Quality of Life assessment instrument-BREF (WHOQOL-BREF) and covers many of the areas of the ICF. However, because this measure is generic and not specific to the population examined in this study, a second HRQOL questionnaire has been selected: the Quality of Life after Brain Injury (QOLIBRI) which is specific to individuals with TBI. Finally, since HRQOL questionnaires are not considered outcome measures, and according to Cieza and Stucki (2005) they do not

capture all areas of the ICF, the current study also included two outcome measures: the MPAI-4 (Malec, 2004b) and the GOSe (Wilson et al., 2007).

Therefore, two measures were used to assess the participants' QOL, and two as outcome measures:

The **Greek** version of the **World Health Organization Quality of Life assessment instrument-BREF** (WHOQOL-BREF; WHOQOL group, 1993) consists of 26 items measuring a person's subjective perceptions about their life with respect to their goals, concerns, and satisfaction. The items fell into 4 main domains: physical health, psychological health, social relationships, the environment; and two general questions on quality of life and general health. Each question has a 5-point response scale, with 1 being *"Very poor"* and *5 being "Very good"*. The scores were scaled in a positive direction with higher scores indicating higher QOL. The WHOQOL-BREF has shown good reliability and validity (Trompenaars et al., 2005). Reliability testing was conducted for this sample revealing a Cronbach's α ranging from 0.62 to 0.82.

The **Quality of Life after Brain Injury** (QOLIBRI; von Steinbuechel et al., 2012) is a 37–item inventory consisting of six domains regarding the QOL of groups with TBI. The six dimensions measure: cognition, self, daily life and autonomy, social relationships, emotions and physical problems. Each item is measured on a 5-point likert scale (*1 – None* and *5 – Very much*). The scores are scaled in a positive direction with higher scores indicating higher QOL. Due to its recent development, there is a continuing evaluation for its psychometric properties, with promising findings (internal consistency of each scale Cronbach's alpha ranges from 0.75 to 0.89), and a good test-retest reliability with intra-class correlations ranging from 0.78 to

0.85) (von Steinbuechel et al., 2012). Reliability analyses were also conducted for each scale using this sample (see Table 2, Appendix A).

Functional Outcome

The **Mayo-Portland Adaptability Inventory** (MPAI-4; Malec, 2004b) (and the PI, a component of the MPAI-4) is an outcome measure for individuals with TBI during the post–acute stage of recovery. The MPAI–4 consists of 35 items. Each item is measured on a 5–point Likert scale (*0 – None* and *4 – Severe Disability*). These items fall into one total score and 3 subscale scores measuring: Ability, Adjustment, and Participation. This measure can be filled in by both the patient and a significant other or clinical staff. In this study, only an informant completed this measure. The MPAI-4 has established concurrent, construct, and predictive validity. Reliability has also been established with Cronbach's α index ranging from .76 to .83 (Malec, 2004a). For the purposes of this study, MPAI-4 was translated in Greek through forward and backward procedures, and factors were constructed based on the original manual (Malec, & Lezak, 2008). Therefore, reliability tests were conducted for each scale revealing a Cronbach's α of 0.71 and larger (Table 3, Appendix A).

The **Glasgow Outcome Scale extended** (GOSe; Wilson et al., 2007) is a 9–item scale assessing the patient's status on an 8–point scale: dead, vegetative state, lower severe disability, upper severe disability, lower moderate disability, upper moderate disability, lower good recovery, and upper good recovery. The GOSe ratings were based on a structured interview with the participants with TBI that were easily recoded to GOS ratings. This scale is one of the most commonly used global outcome measure with individuals with TBI, allowing for the comparison with world literature on TBI outcome.

Coping

A coping measure was also incorporated into the testing procedure, as literature has shown that coping mechanisms may affect one's self-awareness and executive dysfunction (Brands, Köhler, Stapert, Wade, & van Heugten, 2014a; Toglia, & Golisz, 2017, pp. 117-143). Specifically, Brands et al. (2014a) reported that greater impairment in EF may guide the use of ineffective coping strategies. In addition, it has been suggested that QOL may be also hampered by the use of coping mechanisms, including substance abuse, avoidance, passive coping, self-blame (Anson, & Ponsford, 2006; Brands, Köhler, Stapert, Wade, & van Heugten, 2014b; Wolters, Stapert, Brands & van Heugten, 2011). Therefore, coping strategies may further complicate the SA and QOL relationship, and should be controlled for. This measure was administered to both groups.

The **Brief Cope** (Carver, 1997) is a 28-item tool assessing the strategies employed by individuals in order to cope with problems and stress. The original Brief Cope produces 14 coping strategies including acceptance, active coping, positive reframing, planning, use of instrumental support, use of emotional support, behavioral disengagement, self-distraction, self-blame, humor, denial, religion, venting, and substance use (Carver, 1997; Muller, & Spitz, 2003); each loaded by 2 items. Responses are provided on a 4-point Likert scale ranging from "not at all" to "very much", with higher scores indicating higher use of coping strategies. The Greek adaptation of the Brief COPE yielded 14 factors: Active Coping, Planning, Positive Reframing, Acceptance, Humor, Using Emotional Support, Religion, Instrumental Support, Denial, Self-distraction, Venting, Self-blame, Substance use, and Behavioral Disengagement (Kapsou, Panayiotou, Kokkinos, & Demetriou, 2010). However, for the present project three interpretable higher-order indexes were extracted, as suggested by a more recent study (Michaelides, Christodoulou, Karekla, & Panayiotou, 2016). This study tested for the reliability levels of each factor (Table 4, Appendix A).

Mood & Anxiety

Finally, the **Greek** adaptation of the **Symptoms Rating Scale for Depression and Anxiety** (SRSDA; Fountoulakis et al., 2003) was employed in order to assess the presence of depressive or anxious symptomatology as it has been suggested that mood and anxiety – related disorders are more common in the TBI population (Whelan-Goodinson, Ponsford, Schonberger, & Johnston, 2009), and may hamper EF, SA, and as an effect QOL (see Toglia, & Golisz, 2017, pp. 117-143). It has been reported that individuals with TBI who experience mood and anxiety disorders are more likely to regard themselves as less capable and address greater physical and cognitive deficits, compared to those recorded on the actual evaluation. Despite this differentiating from the individuals with TBI with low SA overestimating their abilities (Seel, Macciocchi, & Kreutzer, 2010), it may affect findings regarding SA, and QOL. Hence, SRSDA was used in this study and completed by both groups in order to eliminate the confounding effects of psychological disorders on the neuropsychological and psychosocial responses.

The SRSDA is a 42-item scale that investigates a number of mood related disorders. It has been based on the Beck Depression Inventory-I (BDI-I) and therefore contains the BDI as a subscale along with several other subscales. These include the Asthenia subscale, the Melancholia Inventory, the Anxiety Inventory, and the Mania subscale. This measure also covers the BDI-I-13 and BDI-I-21 scores. According to Fountoulakis et al. (2003) reliability levels ranged between 0.86 and 0.92 for individual scales, with only the Mania subscale revealing a low alpha (0.12). Test-retest reliability for this measure ranged between 0.79 and 0.91.

Magnetic Resonance Imaging Protocol

Image acquisition. MR images were acquired with a 3.0 Tesla scanner (Achieva, Philips Medical Systems, Best, The Netherlands). The built-in quadrature RF body coil and a phased array 8-channel head coil was used for proton excitation and signal detection, respectively. An isotropic, three-dimensional (3D), T1-weighted rapid acquisition gradient-echo sequence (fast field echo; repetition time = 25ms; echo time =1.85 ms; flip angle = 30^{o}) allowed for acquiring whole brain, transverse MR images with an acquisition/reconstruction voxel of 1.0×1.0 ×1.0 mm (data interpolation was not implemented in any direction to improve resolution and reduce partial volume effects). The scanning session included other standard pulse sequences (e.g., T2-weighted turbo spin echo, diffusion weighted imaging and fluid-attenuated inversion recovery) to exclude significant brain pathology of a different etiology.

Ethics

The sample of the current study consisted of adults. Therefore, despite participants experiencing cognitive deficits no parental/legal guardian consent was required, unless there was a court appointed guardian for the participants with TBI. This phenomenon did not occur with all participants providing informed consent, themselves. Participants (i) were provided with a consent form in order to obtain their own personal consent before participating in the study; (ii) were informed of their right to skip any questions if they wished not to respond, and (iii) were informed of their right to withdraw from the study at any point, if had they felt uncomfortable. It was made explicit that if one refuses participation or wishes discontinuation of the study, no consequences were to follow. The information sheet and consent form described the research purpose, process and duration of the project. Participants were informed that the study had obtained all necessary legal permission (from the Cyprus

Bioethics Committee). In addition, they were informed that the study would not cause any more stress than one might experience on a daily basis. Privacy issues were also managed according to privacy regulations and approved by the Commissioner for Data Protection and the National Bioethics Committee. All personal information was kept confidential, identification data were encrypted using an acronym; and all information were kept on a hard disk available to the researcher only. Participants were informed that the proposed study may involve voice recording and were provided with the researcher's contact details to contact her had they any questions, required additional information, or wished to withdraw from the study. Finally, information was provided regarding the dissemination of the results and the implication of the project. All participants received a copy of their MRI CD and a neuropsychological report stating their current cognitive status.

Data Analyses

In order to examine the proposed hypotheses the following statistical analyses using the Statistical Package for the Social Sciences (SPSS) were conducted: (i) Mixed models Multivariate Analysis of Variance (MANOVA) and (ii) independent samples t-tests were conducted to investigate whether participants with moderate-severe TBI differed in their neuropsychological performance, including EF, SA and QOL, as compared to the controls. (iii) Two-tailed Pearson correlations were conducted to test an association between EF and SA, and QOL. (iv) Two-tailed Pearson correlations were also performed to investigate whether negative associations between QOL and SA deficits, existed. (v) Additionally, the relationships between the low performances in EF tasks, the SA deficits, the QOL, and the functional outcome, in TBI, were examined. (vi) Finally, regression analyses were conducted to examine the predictive validity of SA on QOL.

Volumetry was used to detect group differences in overall GM, WM, and CSF volume with the individuals with TBI expected to present with greater volume loss as compared to the non-injured participants. Individual brain volume calculation was performed using the Individual Brain Atlases Statistical Parametric Mapping toolbox (IBASPM; Alemán-Gómez, Melie-García, & Valdés-Hernandez, 2006) under MATLAB 8.1 (MathWorks, Natick, MA). IBASPM uses the segmentation routines of SPM12 (Wellcome Department of Cognitive Neurology, Institute of Neurology, University College London, London, UK). The MR images were segmented into grey matter, white matter, and cerebral spinal fluid and individual volumes for each tissue type were then extracted. Percent volume change between the control group and the TBI group was calculated using the formula, (mean control group volume – mean TBI group volume) / (mean control group volume) * 100. This index allowed for the quantification of tissue volumetric changes between the two groups.

Preprocessing of the MR images was conducted using SPM 12, prior to performing Voxel-Based Morphometry (VBM) analysis. Preprocessing steps included segmentation of the MR images into GM and WM, followed by a Diffeomorphic Anatomical Registration through Exponentiated Lie Algebra (DARTEL) for inter-subject registration of the GM and WM images. Local GM and WM volumes were conserved by modulating the image intensity of each voxel by the Jacobian determinants of the deformation fields computed by DARTEL. The registered images were, then, smoothed with a Gaussian kernel (Full Width at Half Maximum = 8 mm) and were further transformed to Montreal Neurological Institute (MNI) stereotactic space using affine and nonlinear spatial normalization implemented in SPM12 for statistical comparisons.

Following the pre-processing steps, voxel-based-morphometry analyses were conducted to investigate whether significant volume reduction in specific brain regions was

evident between the group with TBI and the control group. These hypotheses were tested through conducting two samples t-test models in SPM12. In order to detect significant statistical variations in GM and WM in the brain regions between the two groups, a statistical threshold of $p < 0.05$ was set and corrected for the whole brain volume at a cluster level using the "Non-Stationary Cluster Extent Correction" toolbox for SPM12 (http://fmri.wfubmc.edu/cms/ NS-General; (Hayasaka et al. 2004). For this analysis, the design matrix consisted of the group that is, group with TBI vs. the control group. Also, the age and years of education were also entered as covariates of no interest.

Further analyses were conducted to investigate the associations between the volume in EF and SA –related brain regions and neuropsychological performance, and psychosocial measures. Specifically, regression analyses were performed to investigate the predictive validity of brain volume in regions-of-interest (ROIs) in executive dysfunction, impaired SA, and QOL. Therefore, masks of brain regions relating specifically to EF, and SA were downloaded from the database of Neurosynth.org (see Figure 1). These maps were then entered into MRICRON and individual masks of each ROI were hand-drawn. Each mask was then used to extract the volume from each ROI, using MATLAB. Data were then entered into SPSS, to compare differences in these volumes (ROIs), using MANCOVA, with age, education and overall brain volume entered as covariates of no interest. Finally, stepwise regression analyses were performed to investigate the predictive validity of brain volume in ROIs in executive dysfunction, impaired SA, and QOL.

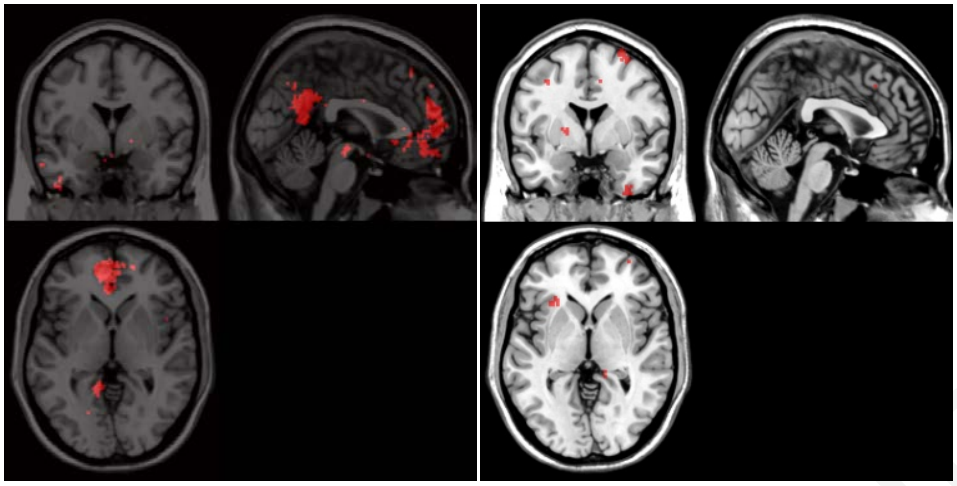

Figure 1. *Masks extracted from Neurosynth.org.*

CHAPTER 4

Experiment 1

Rationale

Traumatic brain injury is a major cause of hospitalization, chronic disability and death worldwide (Chiaravalloti, & Goverover, 2016; Corrigan et al., 2010). It has been described as a chronic and progressive condition accompanied by numerous effects, including neuropsychological impairment, such as deficits in memory, executive functions (EF), and self-awareness (SA), further affecting quality of life (QOL) and functional outcome (Chiaravalloti, & Goverover, 2016; Konstantinou et al., 2016). Although an association has been shown between these chronic impairments, literature has not reached a consensus on the directionality of these relationships (Hanks et al., 2008; Sawchyn et al., 2005; Seel et al., 2010; Skoglund, & Sunnerhagen, 2013).

Despite evidence supporting that EF impairment is evident as recorded several years following a TBI (Caldwell et al., 2014; Konstantinou et al., 2016), mixed findings exist regarding the levels of SA deficits associated with moderate to severe TBI. Specifically, Sasse et al. (2013) showed a significant decline in SA persisting up to 15 years post-injury; whereas Curtiss (2007) indicated that SA improved as time post-injury increased. This perplexity may be assigned to injury severity, as it appears that people with a moderate-severe injury present with greater SA impairment (Seel et al., 2010).

Damage in SA may lead individuals to overestimating their cognitive abilities (Sasse et al., 2013; Seel et al., 2010). This finding may explain the heterogeneity regarding QOL following an injury, with individuals with TBI either over- or under-reporting the consequences of TBI on their QOL (Anderson et al., 2010; van Baalen et al., 2008). Studies

have shown that individuals with TBI experiencing greater SA deficits tend to report greater HRQOL in cognitive, behavioral and leisure aspects, than for more concrete domains, such as motor disability (Mathias, & Wheaton, 2007; Sasse et al., 2013). However, contradicting evidence exists showing that SA deficits may not interfere with how individuals with TBI perceive their QOL (Formisano et al., 2017).

Further complicating this association is evidence regarding the predictive ability of neuropsychological tests, and EF, on QOL. A relationship between these constructs has been found suggesting that people with TBI reporting higher levels of QOL also show better overall cognitive function, perceive themselves as more motivated, report less difficulty in planning, present with fewer mobility issues, are independent in managing their personal hygiene and daily activities, and report less depressive symptomatology and social isolation, than those with lower levels of QOL (Esbjörnsson et al., 2013). However, opposing findings show that neuropsychological performance may be predictive of handicap, functional outcome, supervision needs, and vocational outcome, but not satisfaction with life (Hanks et al., 2008).

It may be concluded that no clear evidence exists to describe how executive dysfunction and SA deficits could affect QOL. Hence, in Experiment 1 it was hypothesized that there is an association between these constructs (neuropsychological deficits, including lack of self-awareness, and quality of life) in chronic moderate-severe TBI.

Statement of Purpose

The incongruency in the association between EF, SA and QOL may be accounted by the ICF model and the need for contextual assessment. For example, a mild word finding problem may not be important in a computer programmer's QOL, but may significantly interfere with the work of an attorney. Therefore, Experiment 1 attempted to capture these

relationships with a more comprehensive assessment package, and examine the predictive ability of EF and SA on each domain of QOL and HRQOL, separately. Additionally, various outcome measures were used to differentiate QOL from HRQOL and functional outcome. Finally, the sample consisted of individuals with moderate-severe TBI to avoid contaminating potential findings with changes assigned to injury severity.

As a result of previous findings, it was expected that the group with TBI would exhibit differences in neuropsychological performance, including SA deficits as compared to the control group. In addition, it was expected that no differences would be detected regarding QOL domains, except for physical aspects. Another aim of this study was to examine if low levels of SA relate to higher levels of quality of life.

Significance

Findings from Experiment 1 shed light to the enigmatic relationships between EF, SA, and QOL. Poor SA has been described as a significant barrier to social integration, and as a result it is considered a significant predictor of poor psychosocial outcome and employment post TBI (Chiaravalloti, & Goverover, 2016). Therefore, highlighting the chronic and persistent course of executive dysfunction, and thus SA deficits, and how these may further affect one's QOL will assist health-care professionals in designing more comprehensive rehabilitation programs by focusing on the impact of impairments on daily participation that would further improve patients' recovery. In addition, results from this experiment would contribute towards the further development of biopsychosocial models that include the interrelationship between EF, QOL, and metacognition in TBI recovery.

Hypotheses

Experiment 1 examined differences in neuropsychological performance, self-awareness (SA), and quality of life between a group with moderate-severe TBI and non-injured controls, as well as the association between these constructs.

Hypothesis 1

It was hypothesized that the participants with moderate-severe TBI would present with greater cognitive impairment, including executive dysfunction and reduced SA, as compared to the non-injured controls. TBI has been associated with severe and persistent neuropsychological impairment for several years post injury (e.g. Ord et al., 2010; Rabinowitz, & Levin, 2014). Such deficits include impairment in memory, attention, and speed of processing, as well as executive dysfunctions (Barak, O., Vakil, & Levy, 2013; Prigatano, 2005; Rabinowitz, & Levin, 2014; see Vakil, 2005; Vakil, 2013). Persistent deficits in SA have also been reported in individuals with moderate-severe TBI up to 15 years post injury (Prigratano, 2005; Sasse et al., 2013).

Hypothesis 2

Secondly, it was expected that the TBI and control groups would not differ on their reported levels of overall QOL, as measured by the WHOQOL-BREF, neither on QOL specific domains relating to their social relationships, and psychological and environmental areas of their lives, except for the physical aspects. Literature focusing on QOL post TBI supports that individuals with TBI either tend to report greater levels of satisfaction concerning their cognitive abilities or report lower QOL levels when focusing on their mobility issues (Sasse et al., 2013). However, few studies have extensively examined this phenomenon by employing appropriate QOL measures, with most mainly focusing on the

overall QOL. Therefore, this study aimed at using the WHOQOL-BREF as a measure to record these different areas, independently, between the two groups.

Hypothesis 3

Finally, it was hypothesized that QOL, as measured by the WHOQOL-BREF and the QOLIBRI would negatively correlate to and be predicted by SA deficits in individuals with chronic TBI. It has been reported that individuals with TBI, presenting with lower levels of SA tend to report greater levels of satisfaction with their QOL, mainly regarding their cognitive abilities, but not aspects relating to mobility problems (Sasse et al., 2013). Previous studies investigating these associations have not reached a consensus since findings have been mixed, suggesting that injury severity may be further complicating the association between EF, SA and QOL; or that this incongruence between one's performance in neuropsychological tasks and QOL may be accounted by the ICF model and the need for contextual assessment. Therefore, this study recruited only individuals with moderate-severe TBI to avoid potential implications of injury severity. In addition, a comprehensive assessment was conducted including both a QOL and HRQOL measure, as well as measures of functional outcome in order to differentiate QOL from HRQOL and functional outcome.

Methods

Participants

The sample consisted of 57 native Greek speaking adults: 33 individuals with TBI, and 24 healthy individuals; with an age range of 18–51 years old. Participants with TBI were pair-matched to the non-injured controls on age, gender, and education. The patient group consisted of individuals who had sustained moderate-severe TBI at least one year post injury. All participants underwent a neuropsychological assessment, and a number of QOL and

psychosocial measures. A control group was used to ensure that any changes in brain morphology, neuropsychological performance and QOL resulted from the injury. The types of questionnaires measuring QOL were selected to provide information specific to the effects of TBI. For a full description of the sample see Table 5, in Appendix A.

Procedure

Data collection procedures, including the neuropsychological and psychosocial assessment, were completed in eight months. The data was collected in a laboratory setting, where both the neuropsychological and QOL measures were administered individually. The neuropsychological testing lasted for 1.5-2 hours per participant with TBI, and 1-1.5 hours for the control group participants. During testing participants were provided with scheduled breaks in order to avoid mental fatigue. The QOL measures took about 45 minutes for the TBI group and 10 minutes for the control group. Participants with TBI completed additional TBI-specific measures (see Methods in Chapter 3).

Materials

All participants underwent a battery of pen-and-paper neuropsychological assessment tools sensitive to cognitive deficits associated with TBI, and psychosocial questionnaires. All testing material was adjusted to Greek-native speakers. For a detailed description of the materials, see Chapter 3, Materials.

Neuropsychological Performance

Neurocognitive performance, i.e. memory, EF, attention, and cognitive reserve, was assessed using the following measures: (i) EF were assessed using the Rey Complex Figure Test (copy) (Rey, 1993), the Trail Making Tests A and B (also processing speed; Zalonis et al., 2008), the Symbol Digits Modalities Test (SDMT; Smith, 1982), and the phonological

(letter F) and category recall (Animal recall) from the Control Oral Word Association Test (COWAT; Kosmidis et al., 2004); (ii) Verbal and visual memory was examined using the Digit Span Forward and Backwards and Visual Span Forward and Backwards (adapted Wechsler Memory Scale-III, WMS-III; Wechsler, 1997), the Greek adaptation of the Auditory Verbal Learning Test (AVLT; Constantinidou, & Evripidou, 2012a), the Rey Complex Figure Test immediate and delayed recall (Rey, & Osterrieth, 1993), the Greek Passage Memory test (which is based on the Wechsler Memory Scale Logical Memory subtest; Constantinidou, & Ioannou, 2008b); (iii) and Cognitive Reserve was assessed using the Peabody Picture Vocabulary Test (PPVT; Simos, Kasselimis, & Mouzaki, 2011) and a reading measure assessing the total number of pseudowords correctly read in 45 s as measured by a test of pseudowords in Greek (Simos et al., 2013). The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005; Greek version Kounti, & Tsolaki, 2006) was also conducted as a screening tool to detect mild cognitive impairment, with a cut off score of 26 and lower.

Self-awareness

An additional two questionnaires were employed to detect SA deficits: (i) the Dysexecutive Questionnaire (DEX; Burgess et al., 1996), which was completed by both the participants themselves (group with TBI and control group; DEX-R-S), and an informant (both groups; DEX-R-I), i.e., a significant other or a family member; and (ii) the Self-Regulation Skills Interview (SRSI; Ownsworth et al., 2000) conducted in a semi-structured interview format with the individuals with TBI, only.

Quality of Life

Quality of life was assessed using a number of questionnaires that attempted to follow the ICF concepts in order to thoroughly describe the HRQOL phenomenon. Therefore, both a

generic and a TBI-specific measure were used: the World Health Organization Quality of Life assessment instrument-BREF (WHOQOL-BREF; WHOQOL group, 1993), which was completed by all participants, covering many of the areas of the ICF; and the Quality of Life after Brain Injury (QOLIBRI; von Steinbuechel et al., 2012), specific to individuals with TBI. In addition, two functional outcome measures were employed: the *fourth edition* of the Mayo-Portland Adaptability Inventory 4 (MPAI-4; Malec, 2004b; completed by an informant) and the Galveston Outcome Scale Extended (GOSe; Wilson et al., 2007; completed by the participants with TBI, only), as HRQOL measures do not capture all areas of the ICF (Cieza, & Stucki, 2005).

Coping and Mood & Anxiety

Finally, a coping measure and a questionnaire concerning mood and anxiety –related issues were added to the testing package. Literature has shown that both coping mechanisms and depressive or anxious symptomatology, often experienced by individuals with TBI, may also affect one's self-awareness and executive dysfunction, and further affect QOL (Toglia, & Golisz, 2017, pp. 117-143). Thus, the Brief Cope (Carver, 1997) was conducted to assess coping strategies employed; whereas the Symptoms Rating Scale for Depression and Anxiety (SRSDA; Fountoulakis et al., 2003) was used to detect potential depressive or anxious symptomatology.

Statistical analysis

For Experiment 1, four main statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS): (i) Mixed models Multivariate Analysis of Variance (MANOVA) and (ii) independent samples t-tests were employed to investigate whether participants with moderate-severe TBI differed in their neuropsychological performance,

including EF, SA and QOL, as compared to the non-injured group. (iii) One-tailed Pearson correlations were performed to examine the relationships between EF, SA, QOL, and functional outcome. (iv) Lastly, regression analyses were conducted to examine the predictive validity of EF, and thus SA, on QOL. Corrections for multiple statistical tests were implemented where warranted to reduce the probability for Type I error.

Results

Demographics

Group with TBI

For Experiment 1, the group with TBI consisted of 33 male adults that met the study criteria and were included in Experiment 1. The participants with TBI had a mean age of 31.06 (range = 18–51; SD = 8.51), and a mean level of education of 12.42 (range = 6–19; SD = 2.98). All participants with moderate-severe TBI were recruited at a mean time since injury of 5.24 years (range = 1–19; SD = 5.63). Functional outcome was assessed using the GOSe which indicated that six participants presented with lower severe disability (18.2%), four with upper severe disability (12.1%), three with lower moderate disability (9.1%), 14 were rated as upper moderate disability (42.4%), five had achieved lower good disability (15.2%), and one with upper good disability (3%).

Causes of TBI included motor vehicle collisions with 33.3% being involved in a car accident, 39.4% in a motor cycle injury, and 12.1% in a pedestrian-vehicle collision, thus a total of 84.8%. A percentage of 6.1 were injured in a non-sports-related fall, another 6.1% in an assault, and a 3% by object falling. These causes are consistent with the primary causes of injury reported in the literature (Roozenbeek, Maas, & Menon, 2013). Almost all participants had received acute inpatient rehabilitation up to 360 days, except for three individuals who

had received fragmented individualized outpatient treatment. However, no participant had received systematic and comprehensive post-acute rehabilitation services. A full description of each participant with TBI is provided in Table 5, in Appendix A.

Control Group

The control group consisted of 24 male participants, with a mean age of 31.92 (range = 21–49; SD = 8.18), and a mean educational level of 13.63 (range = 8–17; SD = 2.48). This group comprised of volunteers from the greater areas of Nicosia, Limassol, Paphos and Larnaca. None of the controls exhibited any neurological, learning disability, documented psychological disorder, or substance abuse.

Group Comparisons

The two groups did not differ in terms of age and education: age, $t(55) = -0.38$, $p = 0.705$; education, $t(55) = -1.61$, $p = 0.113$. Therefore, any differences detected in subsequent analyses may not be attributed to differences in age or education between the two samples.

Neuropsychological Performance

Executive Functioning

1. Trails Making Tests A & B

A repeated measures MANOVA was conducted to compare the two groups on the Trail Making Tests (TMT) A and B. The two groups differed significantly on their performance on TMT A & B, with $F(1, 55) = 13.74$, $p = 0.0001$, $\eta^2 = 0.20$, observed power = 0.95. As expected, there was a significant main effect for the TMT, Pillai's Trace = 0.65, $F(1, 55) = 101.98$, $p = 0.0001$, $\eta^2 = 0.65$, observed power = 1.00, as subjects required more time to complete TMT B, than TMT A. An interaction between the group and the TMT was also

evident, Pillai's Trace = 0.07, $F(1, 55) = 4.31$, p = 0.043, η^2=0.07, observed power = 0.53, with the participants with TBI taking almost twice as much time to complete TMT B than TMT A, as compared to the control group participants (Table 6, Appendix A).

2. Verbal Fluency

Note. To account for multiple statistical tests, the a level was reduced to .01 (Bonferroni α' = a/k where k is number of tests; α' = .05/4 = 0.01).

Animal Naming

Independent samples t-test revealed a significant difference between the two groups in animal naming, $t(55) = -3.22$, p = 0.002, with the group with TBI retrieving fewer items than the control group (M = 13.58, SD = 4.87 and M = 17.50, SD = 4.03, respectively; Table 7, Appendix A).

Letter F

Similar results were evident for the ability to retrieve words starting with the letter F, $t(55) = -3.44$, p = 0.001, with the group with TBI producing a significantly fewer words (M = 8.70, SD = 3.27) compared to the control group (M = 12.13, SD = 4.25; Table 7, Appendix A).

3. Rey Complex Figure Test: Copy Trial

Independent samples t-test revealed a significant difference between the two groups for the Rey Copy trial, $t(37.92) = -4.28$, p = 0.0001, with the group with TBI showing reduced performance in their ability to copy a complex design (M = 26.40, SD = 6.28), than the control group (M = 31.29, SD = 1.66; Table 7, Appendix A).

4. Symbol Digits Modalities Test

The two groups differed significantly on the SDMT, $t(51.45) = -6.62$, $p = 0.0001$, with the control group performing significantly higher ($M = 51.25$, $SD = 7.26$) compared to the group with TBI ($M = 33.00$, $SD = 13.37$; Table 7, Appendix A).

Verbal Memory

1. AVLT

Learning Trials

A repeated measures MANOVA was conducted to compare the two groups on the AVLT learning trials (i.e., Trials 1 to 5). The two groups differed significantly on their overall performance across the five learning trials, $F(1, 55) = 30.36$, $p = 0.0001$, $\eta^2 = 0.36$, observed power = 0.10, with the group with TBI demonstrating lower scores than the control group. In addition, all participants showed an increase in their learning, for each trial; Pillai's Trace = 0.82, $F(4, 52) = 60.30$, $p = 0.0001$, $\eta^2 = 0.82$, observed power = 1.00. An interaction between the group and the AVLT was also found, Pillai's Trace = 0.19, $F(4, 52) = 2.97$, $p = 0.028$, $\eta^2 = 0.19$, observed power = 0.76, with the group with TBI showing a smaller increase for each learning trial, and thus a lower learning curve, as compared to the non-injured individuals (Table 6, Appendix A).

Recall Trials

Mixed model repeated measures MANOVA was also conducted to compare the two groups on the AVLT immediate (trial 5 and 6) and delayed (trial 7) recall trials. The two groups differed significantly on their performance, $F(1, 54) = 20.73$, $p = 0.0001$, $\eta^2 = 0.28$, observed power = 0.99, with the control group performing significantly higher than the group with TBI. There was a significant main effect of the AVLT Recall trials, Pillai's Trace = 0.63, $F(2, 53) = 44.80$, $p = 0.0001$, $\eta^2 = 0.63$, observed power = 1.00, where differences between the

immediate and delayed trials were shown. An interaction between the group and the AVLT Recall trials was also evident, Pillai's Trace = 0.11, $F(2, 53)=3.36$, p = 0.042, $\eta^2 = 0.11$, observed power = 0.61, with the control group participants showing a decline in retaining information for trial 6, but not for trial 7; whereas the group with TBI showed a constant decline in maintaining information across all three trials, i.e. a decline between trial 5 to 6, and trial 6 to 7 (Table 6, Appendix A).

2. Logical Memory

Mixed model repeated measures MANOVA was used to compare the two groups on the Logical Memory Test, for the story type (Story A & B) and the time of recall (Immediate, Delayed). The two groups differed significantly on their performance on this test, $F(1, 55) = 32.82$, p = 0.0001, $\eta^2 = 0.37$, observed power = 1.00, with the control group demonstrating superior performance across stories and recall conditions. There was a significant main effect for the Recall condition (Immediate, Delayed) factor, Pillai's Trace = 0.52, $F(1, 55) = 59.32$, p = 0.0001, $\eta^2 = 0.52$, observed power = 1.00, indicating that participants across groups recalled more items during the immediate as compared to the delayed condition. No main effect was evident for the Story type (Story A, Story B), Pillai's Trace = 0.05, $F(1, 55) = 2.62$, p = 0.111, $\eta^2 = 0.05$, observed power = 0.36. An interaction between Group and Recall conditions was found, Pillai's Trace = 0.08, $F(1, 55) = 4.90$, p = 0.031, $\eta^2 = 0.08$, observed power = 0.59, with the group with TBI showing greater decline from immediate to delayed recall than the control group (Table 6, Appendix A). There was no Story type by group interaction, Pillai's Trace = 0.00, $F(1, 55) = 0.07$, p = 0.790, $\eta^2 = 0.01$, observed power = 0.06. However, a Recall by Story type interaction was evident, Pillai's Trace = 0.09, $F(1, 55) = 5.63$, p = 0.021, $\eta^2 = 0.09$, observed power = 0.65, demonstrating that the participants remembered significantly more information for story A, than B, during the immediate recall, but their performance during the

delayed condition was similar for both stories. Finally, no 3-way interaction (Recall by Story type by group interaction) was found, Pillai's Trace = 0.04, $F(1, 55) = 2.33$, $p = 0.133$, $\eta^2 = 0.04$, observed power = 0.32 (Table 6, Appendix A).

3. Digit Span Forward and Backwards

Note. To account for multiple statistical tests, the a level was reduced to .01 (Bonferroni α' = a/k where k is number of tests; α' = .05/4 = 0.01, including Digit Span (Forward and Backward) and Visual Span (Forward and Backward)).

An independent samples t-test revealed a significant difference between the two groups on the Digit Span Forward, $t(55) = -2.86$, $p = 0.006$, with the control group showing greater performance on this task ($M = 7.83$, $SD = 1.88$), than the group with TBI ($M = 6.21$, $SD = 2.26$). Similar findings were evident for the Digit Span Backward, $t(55) = -3.51$, $p = 0.001$, with the group with TBI demonstrating greater difficulty in recalling the reverse sequence of numbers presented to them ($M = 5.00$, $SD = 2.24$), compared to the control group ($M = 7.04$, $SD = 2.07$).

For a full description of the statistics see Table 7 in Appendix A.

Non-Verbal Memory

1. Rey Complex Figure Test

Immediate and Delayed Recall

A mixed model repeated measures MANOVA revealed a significant difference between the two groups, $F(1, 55) = 12.04$, $p = 0.001$, $\eta^2 = 0.18$, observed power = 0.93, with the control group recalling more items during the two conditions. There was a significant Condition effect indicating that participants across groups performed better at the immediate

recall condition than the delayed condition, Hotelling's T = 0.15, $F(1, 55) = 8.15$, p = 0.006, $\eta^2 = 0.13$, observed power = 0.80. No interaction was shown between the Recall conditions and Group; Hotelling's T = 0.01, $F(1, 55) = 0.46$, p = 0.50, $\eta^2 = 0.13$, observed power = 0.10 (Table 6, Appendix A).

2. Visual Span Forward and Backward

The two groups did not differ on the Visual Span Forward, $t(54) = -1.75$, p = 0.087, with both groups performing equally well (see Table 7 in Appendix A for statistics). Furthermore, no significant differences were found between the two groups on the Visual Span Backward, $t(54) = -1.57$, p = 0.123, suggesting that both groups performed similarly on tasks of visual working memory.

Language Tests

Note. To account for multiple statistical tests, the a level was reduced to .0025 (Bonferroni α' = a/k where k is number of tests; α' = .05/2 = 0.025).

1. Pseudowords

An independent samples t-test revealed a significant difference between the two groups on this test, $t(55) = -4.37$, p = 0.0001, with the control group demonstrating higher reading ability of pseudowords ($M = 38.63$, $SD = 11.30$), than the group with TBI ($M = 23.94$, $SD = 13.35$).

2. Peabody Picture Vocabulary Test

A significant difference was found on the PPVT, $t(43.98) = -4.72$, p = 0.0001, with the group with TBI showing a significantly reduced performance ($M = 20.94$, $SD = 6.86$), compared to the control group ($M = 27.13$, $SD = 2.66$).

For a full descriptive of the Language tests, see Table 7 in Appendix A.

Despite the fact that the two groups were matched on years of education, it appears that they differed on tasks measuring crystallized intelligence. Therefore, further statistical analysis was conducted using a median split on the TSI (Median = 3 years) for the group with TBI and further comparisons were conducted on these measures. Results show significant differences within the group with TBI on the PPVT, $t(31) = -2.40$, $p = 0.023$, indicative that hold intelligence is negatively affected by TSI.

Summary

Findings revealed significant differences between the two groups on their neuropsychological performance, with the control group demonstrating superior performance across most of the tests, except for two tasks measuring non-verbal attention and non-verbal short term memory capacity (i.e., the Visual Span Forward & Backward).

Psychosocial Measures

Multivariate ANOVAs were conducted to investigate differences between the two groups (group with TBI vs. controls) regarding SA and QOL levels, the coping mechanisms used, and the participants' mood and anxiety levels.

Self-Awareness Measure

DEX-R

Three factors were extracted for the DEX-R-Self (DEX-R-S) and the DEX-R-Informant (DEX-R-I) ratings, separately, as suggested by Dimitriadou (2016). To fully capture the degree of SA the discrepancy between the indexes of the informants were subtracted from the participants factors, i.e. (DEX-R-Self – DEX-R-Informant = DEX-R-Discrepancy$_i$),

resulting in a continuum of negative and positive differences. A positive score depicts no SA deficits, whereas negative scores are indicative of greater impairment in SA. The two groups were compared on the new indexes, Pillai's Trace = 0.29, $F(3, 53) = 7.12$, p = 0.001, η^2 = 0.29, $\eta^2 = 0.08$, observed power = 0.95. The group with TBI indicating greater SA deficits on two of the constructs, (Fluency, Flexibility and Working Memory, $F(1, 55) = 3.22$, p = 0.078, $\eta^2 = 0.06$, observed power = 0.42, Social and Self-Regulation, $F(1, 55) = 13.99$, p = 0.0001, η^2 = 0.20, observed power = 0.96, Motivation and Attention, $F(1, 55) = 11.72$, p = 0.001, η^2 = 0.18, observed power = 0.92, compared to the control group. For a full description of the means and standard deviations of the discrepancy indexes, see Table 8 in Appendix A.

Quality of Life Measures

WHOQOL-BREF

Multivariate ANOVA revealed a significant difference between the two groups; Pillai's Trace = 0.32, $F(4, 52) = 6.19$, p = 0.0001, $\eta^2 = 0.99$, observed power = 1.00. Specifically, the group with TBI reported lower levels on the quality of their Physical Health, $F(1, 55) = 5.18$, p = 0.027, $\eta^2 = 0.09$, observed power = 0.61; whereas the control group reported lower levels on the environmental aspects of their lives (e.g., financial resources, physical safety and security, home environment, health and social care, participation in and opportunities for leisure activities), $F(1, 55) = 4.68$, p = 0.035, $\eta^2 = 0.08$, observed power = 0.57. No significant differences were detected for any of the remaining subscales of the WHOQOL-BREF (Psychological, $F(1, 55) = 0.00$, p = 0.994, observed power = 0.05; Social Relationships, $F(1, 55) = 1.39$, p = 0.244, observed power = 0.21; Total, $F(1, 55) = 0.49$, p = 0.826, observed power = 0.06).

Coping Measure

COPE-Brief

No group effect was found (Pillai's Trace = 0.11, $F(3, 53) = 2.13$, $p = 0.107$, $\eta^2 = 0.11$, observed power = 0.52), using the three factors extracted from the COPE Brief (Michaelides et al., 2016; see Table 9, Appendix A).

Mood and Anxiety Scale

SRSDA

The two groups differed significantly on the SRSDA, Pillai's Trace = 0.24, $F(6, 48) = 2.48$, $p = 0.036$, $\eta^2 = 2.37$, observed power = 0.78. Specifically, the two groups showed a significant difference on the Melancholia subscale ($F(1, 53) = 4.35$, $p = 0.042$, $\eta^2 = 0.08$, observed power = 0.54), only, with the group with TBI expressing more symptoms of melancholia. No significant differences were found for any of the other scales of the SRSDA (Long Depression, $F(1, 53) = 2.22$, $p = 0.143$, $\eta^2 = 0.04$, observed power = 0.31; Short Depression, $F(1, 53) = 3.43$, $p = 0.070$, $\eta^2 = 0.06$, observed power = 0.44; Asthenia, $F(1, 53) = 0.00$, $p = 0.961$, $\eta^2 = 0.00$, observed power = 0.05; Anxiety, $F(1, 53) = 0.04$, $p = 0.835$, $\eta^2 = 0.00$, observed power = 0.06; Mania, $F(1, 53) = 0.11$, $p = 0.738$, $\eta^2 = 0.00$, observed power = 0.06). For descriptive statistics refer to Table 10 (Appendix A).

Summary

Given these findings, it appears that the group with TBI presented with lower levels of SA as indicated by their informants' ratings, who reported more dysfunctional behavioral patterns regarding executive cognition and motivation, than the significant others of the control group. Also, individuals with TBI reported lower levels of satisfaction regarding their somatic health, than the non-injured group. The control group appeared less satisfied with their financial resources, health and social care, and other aspects included in the environment

dimension of the WHOQOL-BREF. No significant differences were detected between the two groups regarding the coping techniques employed. Finally, the group with TBI expressed higher levels of melancholic mood, as compared to the non-injured individuals.

Correlations

One-tailed Pearson's correlations were conducted to examine the relationships between EF, SA, and QOL. Specifically, it was expected that QOL, as measured by the WHOQOL-BREF and HRQOL by the QOLIBRI, in individuals with chronic TBI, would negatively correlate to SA deficits. These analyses were performed using either the group with TBI, the controls, or both groups.

Prior to examining these associations, it was sought to investigate whether the coping strategies and emotional state of the participants associated with EF, SA, and QOL, as it has been proposed that such variables may hinder the correlations under investigation.

Due to the number of correlations conducted corrections for multiple comparisons were applied for each analysis by lowering α' to 0.01.

Relationships Coping Mechanisms & EF, SA & QOL

Executive Functions

Whole Group

Two significant negative associations were detected for the entire group, with the participants who performed better on the TMT A, $r(57) = -0.34$, $p = 0.005$ and the SDMT, $r(57) = -0.47$, $p = 0.0001$, also reported lower use of emotion-focused coping techniques. For all other correlations see Table 11, in the Appendix section A.

Independent Group

For the group with TBI, results showed a significant association between emotion-focused coping techniques and the SDMT, $r(33) = -0.43$, $p = 0.006$, supporting that greater performance on the SDMT related to lower use of emotion-related coping (Table 12, Appendix A). No significant associations were evident for the control group (Table 13, Appendix A).

Self-awareness

Whole-Group

No significant associations were evident for the whole-group analysis between the coping mechanisms employed and SA, as measured by the DEX-R (Table 14, Appendix A).

Independent-Group

Again, no significant associations were revealed between the coping measure and the DEX-R (Table 15, Appendix A), nor the SRSI (Table 17, Appendix A), both measuring SA, for the group with TBI. However, for the control group, results showed that greater SA in Flexibility, Fluency and Working Memory associated with more avoidance-related mechanisms, $r(24) = 0.59$, $p = 0.001$, and less problem-task coping, $r(24) = -0.48$, $p = 0.009$. Also, non-injured individuals with greater awareness for their motivational and attentional skills reported fewer problem-task coping techniques, $r(24) = -0.71$, $p = 0.0001$. In addition, greater emotion focused coping was associated with less SA of social and self –regulation, $r(24) = -0.56$, $p = 0.002$, and motivational and attentional, $r(24) = -0.68$, $p = 0.0001$, abilities, as well as overall SA, $r(24) = -0.67$, $p = 0.0001$. For all other correlations see Table 16 in Appendix A.

Quality of Life

Whole-group

QOL, as measured by the WHOQOL-BREF, did not related to the coping indexes extracted from the COPE-Brief (Table 18, Appendix A).

Independent-group

No associations were evident for the WHOQOL-BREF and the COPE-Brief for the individuals with TBI (Table 19, Appendix A). However, the QOLIBRI, measuring HRQOL, significantly associated with one dimension, with the individuals with TBI who reported more use emotional coping also reported greater satisfaction with their social relationships, $r(33) = 0.46$, $p = 0.003$ (Table 21, Appendix A).

The control group reporting greater dissatisfaction with their psychological aspect of their QOL employed more avoidance-related techniques, $r(24) = -0.54$, $p = 0.003$. Finally, individuals who reported more satisfaction with their social relationships used less problem-task oriented skills, $r(24) = -0.69$, $p = 0.0001$. For all other correlations see Table 20 in Appendix A.

Summary

Brought together, these findings suggest that EF, self-awareness, and quality of life significantly relate to coping mechanisms employed by the participants. Specifically, all individuals over performing in EF tasks reported less emotion-focused coping techniques. Greater self-awareness was also related to more avoidant coping, and fewer problem-task and emotion-focused coping mechanisms, within the control group. In addition, the control group reporting fewer avoidance and problem-task related techniques also stated greater satisfaction with their psychological health and social relationships, respectively. Finally, participants with

TBI who declared greater use of emotional coping reported greater satisfaction with their social relationships, as this was measured by a TBI-specific HRQOL measure. Therefore, it appears that coping techniques do relate to EF, SA, and QOL, thus further complicating these associations.

Relationships between Mood & EF, SA & QOL

Executive Functions

Whole-group

Executive functions did not relate to any of the mood and anxiety dimensions measured by the SRSDA (Table 22, Appendix A).

Independent-group

Similarly, no associations were evident for the group with TBI (Table 23, Appendix A). Still significant associations were evident for the control group, with the individuals scoring lower on the TMT B also reporting greater asthenia, $r(24) = -0.56$, $p = 0.002$, and anxiety, $r(24) = -0.53$, $p = 0.004$, –related symptomatology. In addition, non-injured controls reporting more symptoms of melancholia, $r(24) = -0.49$, $p = 0.008$, and asthenia, $r(24) = -0.49$, $p = 0.008$, underperformed on the SDMT (Table 24, Appendix A).

Self-awareness

Whole-group

For the whole-group, anxiety, $r(57) = 0.42$, $p = 0.001$, significantly correlated with the fluency, flexibility and working memory index, and the overall awareness index, $r(57) = 0.33$, $p = 0.006$, indicating that individuals experiencing more anxiety also exhibited greater SA of their executive cognition abilities and overall SA, on the DEX-R. Also, individuals

reporting more symptoms of asthenia presented with greater SA regarding their fluency, flexibility, and working memory abilities, $r(57) = 0.34$, $p = 0.005$ (Table 25, Appendix A).

Independent-group

Additional findings were evident for the group with TBI, supporting that greater asthenia, $r(33) = 0.52$, $p = 0.001$, and anxiety scores, $r(33) = 0.59$, $p = 0.0001$, significantly related to greater SA on executive cognition. Also, greater anxiety related to greater overall SA, $r(33) = 0.48$, $p = 0.002$, and SA relating to motivation and attention –related abilities, $r(33) = 0.44$, $p = 0.006$ (Table 26, Appendix A). No significant associations were found for the control group on DEX-R (see Table 27, Appendix A), or the group with TBI on the SRSI (see Table 28, Appendix A).

Summary

Analysis relating SA and mood and anxiety disorders yielded similar results for both the entire group and the group with TBI alone, indicating that more symptoms of asthenia and anxiety associated with greater cognition, and motivation and attention –related SA and overall SA, supporting the implication of mood and anxiety conditions in SA, and vice versa.

Quality of Life

Whole-group

For the whole-group analyses, all participants reporting more mood and anxiety –related issues also stated less satisfaction with life, but for those with more manic traits who reported greater life satisfaction. Greater long-term and short-term depressive symptoms were associated with less satisfaction on all indexes of the WHOQOL-BREF (see Table 1). In addition, reporting of greater melancholic mood was related to less satisfaction with physical,

$r(57) = -0.45$, $p = 0.0001$, and psychological health, $r(57) = -0.44$, $p = 0.0001$, social relationships, $r(57) = -0.32$, $p = 0.00$, and overall well-being, $r(57) = -0.47$, $p = 0.0001$. Greater symptomatology regarding asthenia was significantly correlated less satisfaction with one's physical health, $r(57) = -0.32$, $p = 0.008$. Similar results were found between greater anxiety and overall wellbeing, $r(57) = -0.31$, $p = 0.009$. However, individuals reporting more manic-related behaviors also stated greater satisfaction with their physical, $r(57) = 0.41$, $p = 0.001$, and psychological health, $r(57) = 0.47$, $p = 0.0001$, the environmental aspect, $r(57) = -0.32$, $p = 0.009$, and their overall QOL, $r(57) = 0.45$, $p = 0.0001$.

Table 1. *Correlations between WHOQOL-BREF and Mood & Anxiety –related symptoms*

	WHOQOL-BREF				
	Physical Health	**Psychological**	**Social Relationships**	**Environment**	**Total**
Long Depression	-0.46*	-0.53*	-0.38*	-0.37*	-0.56*
Short Depression	-0.49*	-0.55*	-0.43*	-0.37*	-0.58*
Melancholia	-0.45*	-0.44*	-0.32	-0.28	-0.47*
Asthenia	-0.32*	-0.16	-0.04	-0.23	-0.26
Anxiety	-0.31	-0.24	-0.12	-0.26	-0.31*
Mania	0.41*	0.47*	0.14	0.32*	0.45*

** p<0.01; Whole-group N=57.*

Independent-group

WHOQOL-BREF

Similar findings to the whole-group were evident for the group with TBI (see Table 2), alone, greater symptomatology of long and short –term depression associated with more dissatisfaction with physical health ($r(33) = -0.44$, $p = 0.005$; $r(33) = -0.47$, $p = 0.003$, respectively), psychological health ($r(33) = -0.62$, $p = 0.0001$; $r(33) = -0.62$, $p = 0.0001$, respectively), social relationships ($r(33) = -0.46$, $p = 0.004$; $r(33) = -0.50$, $p = 0.002$, respectively), environmental aspects ($r(33) = -0.51$, $p = 0.001$; $r(33) = -0.52$, $p = 0.001$, respectively), and overall QOL ($r(33) = -0.62$, $p = 0.0001$; $r(33) = -0.64$, $p = 0.0001$, respectively). In addition, participants with TBI reporting more melancholic mood also stated greater dissatisfaction with their physical ($r(33) = -0.42$, $p = 0.008$) and psychological health ($r(33) = -0.55$, $p = 0.001$), the environmental aspects of their life ($r(33) = -0.47$, $p = 0.003$) and their overall QOL ($r(33) = -0.56$, $p = 0.0001$). Less satisfaction with one's psychological ($r(33) = -0.45$, $p = 0.004$), environmental ($r(33) = -0.45$, $p = 0.004$), and overall well-being ($r(33) = -0.48$, $p = 0.002$) was correlated to greater anxiety levels. Finally, more mania-related issues were associated with greater satisfaction with their physical health ($r(33) = 0.49$, $p = 0.002$).

For the control group, only three significant positive associations were found, indicating that non-injured individuals reporting more manic characteristics also stated greater satisfaction with their psychological ($r(24) = 0.84$, $p = 0.0001$), environmental ($r(24) = 0.60$, $p = 0.001$) and overall well-being ($r(24) = 0.66$, $p = 0.0001$). Also, greater satisfaction with

one's overall QOL reported fewer depressive symptomatology ($r(33) = -0.48$, $p = 0.008$; see Table 2).

Table 2. *Correlations between the WHOQOL-BREF and Mood & Anxiety –related symptoms*

	WHOQOL-BREF									
	Physical Health		**Psychological**		**Social Relationships**		**Environment**		**Total**	
	Group with TBI	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**
Long Depression	-0.44*	-0.42	-0.62*	-0.38	-0.46*	-0.09	-0.51*	-0.42	-0.62*	-0.44
Short Depression	-0.47*	-0.37	-0.62*	-0.46	-0.49*	-0.13	-0.52*	-0.47	-0.64*	-0.48*
Melancholia	-0.42*	-0.37	-0.55*	-0.27	-0.38	-0.03	-0.47*	-0.23	-0.56*	-0.32
Asthenia	-0.36	-0.32	-0.33	0.07	-0.13	0.12	-0.43	-0.06	-0.40	-0.06
Anxiety	-0.36	-0.30	-0.45*	0.06	-0.29	0.15	-0.45*	-0.06	-0.48*	-0.06
Mania	0.49*	0.19	0.29	0.84*	0.03	0.39	0.18	0.60*	0.34	0.66*

** p<0.01; Group with TBI N=33; Control group=24.*

QOLIBRI

Significant associations were detected between the HRQOL-related indexes and the SRSDA, in participants with TBI. Specifically, greater long-term depression was associated with lower satisfaction rates with one's cognitive abilities ($r(33) = -0.45$, $p = 0.004$), self-perception ($r(33) = -0.65$, $p = 0.0001$), social relationships ($r(33) = -0.44$, $p = 0.005$), and

overall HRQOL ($r(33) = -0.62$, $p = 0.0001$). Higher scores in short-term depression was correlated with lower satisfaction with cognitive abilities ($r(33) = -0.45$, $p = 0.004$), self-perception ($r(33) = -0.63$, $p = 0.0001$), daily life and autonomy ($r(33) = -0.46$, $p = 0.004$), social relationships ($r(33) = -0.45$, $p = 0.004$), and overall HRQOL ($r(33) = -0.62$, $p = 0.0001$). Additionally, greater dissatisfaction with one's perception of themselves ($r(33) = -0.59$, $p = 0.0001$), and overall HRQOL ($r(33) = -0.55$, $p = 0.0001$) were associated with greater melancholic mood ($r(33) = -0.59$, $p = 0.0001$, $r(33) = -0.55$, $p = 0.0001$, respectively) and greater levels of anxiety ($r(33) = -0.46$, $p = 0.003$, $r(33) = -0.51$, $p = 0.001$, respectively).

Table 3. *Correlations between the QOLIBRI and Mood & Anxiety –related symptoms*

	QOLIBRI						
	Cognition Scale	**Self Scale**	**Daily Life & Autonomy Scale**	**Social Relationships Scale**	**Emotions Scale**	**Physical Problems Scale**	**Total Scale**
Long Depression	-0.45*	-0.65*	-0.40	-0.44*	-0.34	-0.24	-0.62*
Short Depression	-0.45*	-0.63*	-0.46*	-0.45*	-0.33	-0.23	-0.62*
Melancholia	-0.33	-0.59*	-0.34	-0.40	-0.34	-0.23	-0.55*
Asthenia	-0.26	-0.38	-0.19	-0.23	-0.28	-0.34	-0.43
Anxiety	-0.40	-0.46*	-0.28	-0.32	-0.29	-0.28	-0.51*
Mania	0.26	0.19	0.42	0.23	0.09	0.06	0.30

** p<0.01; Group with TBI N=33.*

Summary

In summary, findings clearly depict associations between mood and anxiety –related symptoms and EF, SA, and QOL. Better performance in cognitive flexibility was associated with less asthenia and anxiety symptomatology, for the non-injured participants. In addition, findings from the whole-group revealed that greater SA regarding executive cognition and overall SA was associated with greater anxiety and asthenia symptoms. Finally, it appears that participants reporting more mood and anxiety –related issues, including depression and melancholy, also stated less satisfaction with their QOL, except for those with more manic traits who reported greater life satisfaction. The latter findings were replicated for the group with TBI, alone, regarding both their QOL and HRQOL. Brought together, these findings clearly indicate that mood and anxiety –related issues may further perplex the association between EF, SA, and QOL

As a result of the aforementioned findings, coping skills and mood and anxiety –related symptoms were partialled-out of the associations under investigation.

Executive Functions

Relationship between EF tasks and SA measures

DEX-R

Initially, the relationships between all EF tasks and SA, as measured by the DEX-R, were investigated using either groups or each group separately. Corrections for multiple comparisons were applied by lowering the α' level to 0.01.

Whole-group

Results revealed significant positive associations between all EF tasks and all four indexes of the DEX-R. Specifically, over performing on the COWAT Animal Naming, (r(44) = 0.39, p = 0.003), and the SDMT (r(44) = 0.38, p = 0.005) correlated with greater SA regarding executive cognition. Also, higher scores in SDMT (r(44) = 0.45, p = 0.001), COWAT Animal Naming, (r(44) = 0.48, p = 0.0001) and words from Letter F (r(44) = 0.40, p = 0.003) associated with greater SA of motivation and attention –related abilities. Higher social and self –regulation SA was associated only with greater performance on the COWAT Animal naming task (r(44) = 0.38, p = 0.005). Finally, one's higher scores in the SDMT (r(44) = 0.40, p = 0.003), and the COWAT Animal Naming, (r(44) = 0.44, p = 0.001) associated with better overall SA. Taken together, these correlations indicated that participants who performed better on EF tasks also presented with greater SA regarding fluency, flexibility and working memory, social and self -regulation, and motivation and attention -related behaviors (see Table 4).

Table 4. Partial correlations between DEX-R and EF tasks

	DEX-R			
	Fluency, Flexibility, Working Memory	**Social & Self - Regulation**	**Motivation & Attention**	**Overall SA**
	Bivariate			
Rey Figure Copy	0.31	0.33*	0.36*	0.35*
TMT A (Valenced)	0.11	0.20	0.33*	0.21
TMT B (Valenced)	0.33*	0.31	0.36*	0.35*

SDMT	0.37*	0.38*	0.46*	0.42*
COWAT Animal Naming	0.43*	0.40*	0.50*	0.46*
COWAT Letter F	0.37*	0.34*	0.45*	0.40*
Partial Controlling for Coping and Diathesis				
Rey Figure Copy	0.26	0.30	0.32	0.31
TMT A (Valenced)	-0.05	0.10	0.26	0.10
TMT B (Valenced)	0.28	0.16	0.26	0.24
SDMT	0.38*	0.32	0.45*	0.40*
COWAT Animal Naming	0.40*	0.38*	0.48*	0.44*
COWAT Letter F	0.25	0.26	0.40*	0.31

** p<0.01; Whole-group N=57.*

Independent-Group

These analyses were repeated for both groups separately, with no significant associations found between the DEX-R discrepancy indexes and the various EF tasks. For the results for both groups see Table 5 below.

Table 5. *Partial correlations between DEX-R and EF tasks*

	DEX-R			
	Fluency, Flexibility,	**Social & Self - Regulation**	**Motivation & Attention**	**Overall SA**

	Working Memory							
	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group
	Bivariate							
Rey Figure Copy	0.25	-0.05	0.17	0.04	0.23	-0.11	0.22	-0.02
TMT A (Valenced)	0.01	-0.01	0.02	0.11	0.19	0.38	0.06	0.16
TMT B (Valenced)	0.29	-0.02	0.15	0.09	0.22	0.25	0.22	0.11
SDMT	0.30	0.14	0.11	0.29	0.29	0.30	0.22	0.30
COWAT Animal Naming	0.42*	0.31	0.33	0.04	0.45*	0.26	0.41	0.20
COWAT Letter F	0.40	0.22	0.30	-0.20	0.47*	0.02	0.40	-0.04
	Partial Controlling for Coping and Diathesis							
Rey Figure Copy	0.05	0.25	0.10	-0.03	0.14	-0.10	0.10	0.04
TMT A (Valenced)	-0.36	-0.24	-0.11	0.05	0.01	0.40	-0.17	0.04
TMT B (Valenced)	-0.02	-0.23	-0.15	-0.39	-0.10	-0.09	-0.11	-0.35
SDMT	0.18	-0.04	0.11	0.05	0.21	0.06	0.17	0.03

COWAT Animal Naming	0.34	-0.27	0.29	-0.20	0.45	-0.08	0.37	-0.23
COWAT Letter F	0.21	0.32	0.26	0.09	0.41	0.01	0.30	0.16

** p<0.01; Group with TBI N=33; Control group=24.*

SRSI

Significant associations were found between the TMT B and both the Strategy ($r(44) = -0.49$, $p = 0.009$) and Online/emergent ($r(44) = -0.50$, $p = 0.008$) Awareness indexes of the SRSI, showing that individuals with TBI underperforming in the TMT B tasks also presented with greater SA deficits regarding strategic and emergent –related SA (Table 6).

Table 6. *Partial correlations between SRSI and EF tasks*

	Strategy Awareness	**Emergent/Online Awareness**	**Readiness To Change**
	Bivariate		
Rey Figure Copy	-0.34	-0.36	-0.07
TMT A (Valenced)	-0.13	-0.11	-0.12
TMT B (Valenced)	-0.38	-0.39	-0.13
SDMT	-0.37	-0.37	-0.23
COWAT Animal Naming	-0.40	-0.36	-0.06

COWAT Letter F	-0.40	-0.40	0.38
	Partial Controlling for Coping and Diathesis		
Rey Figure Copy	-0.06	-0.01	-0.03
TMT A (Valenced)	-0.25	-0.31	-0.08
TMT B (Valenced)	-0.49*	-0.50*	-0.20
SDMT	-0.25	-0.24	-0.11
COWAT Animal Naming	-0.35	-0.37	0.46
COWAT Letter F	0.27	0.26	0.15

** p<0.01; Group with TBI N=33.*

Brought together, findings showed that individuals who performed better on EF tasks also presented with greater SA for behaviors relating to executive cognition, social and self-regulation, and motivation and attention, as well as overall SA. Given that the effects of coping and mood and anxiety –related issued were partialled-out these associations were preserved, thus strongly supporting that greater executive functioning relates to better SA.

Relationship between EF tasks & QOL

One-tailed Pearson's correlational analyses were conducted to examine relationships between EF tasks, and quality of life, as measured by the WHOQOL-BREF, and the QOLIBRI.

WHOQOL-BREF

Whole-group

One significant correlation was evident between the COWAT Animal naming task and physical health ($r(44) = 0.40$, $p = 0.003$), suggesting that greater performance on EF tasks associates to greater satisfaction with one's physical health. All correlations are presented in Table 7.

Table 7. *Partial correlations between WHOQOL-BREF and EF tasks*

	WHOQOL-BREF				
	Physical Health	**Psychological**	**Social Relationships**	**Environment**	**Total**
			Bivariate		

Rey Figure Copy	0.28	-0.10	-0.20	-0.15	-0.03
TMT A (Valenced)	0.12	-0.06	-0.01	-0.21	-0.06
TMT B (Valenced)	0.12	-0.17	-0.08	-0.18	-0.10
SDMT	0.18	-0.17	-0.01	-0.36*	-0.13
COWAT Animal Naming	0.26	-0.07	-0.02	-0.22	-0.02
COWAT Letter F	0.21	0.15	0.05	0.02	0.14
	Partial Controlling for Coping and Diathesis				
Rey Figure Copy	0.22	-0.21	-0.20	-0.17	-0.09
TMT A (Valenced)	0.19	0.05	0.11	-0.11	0.06
TMT B (Valenced)	0.01	-0.15	0.05	-0.11	-0.08
SDMT	0.25	-0.15	0.08	-0.28	-0.05
COWAT Animal	0.40*	0.02	0.11	-0.16	0.11

Naming					
COWAT Letter F	0.23	0.15	0.00	0.07	0.16

** p<0.01; Whole-group N=57.*

Independent-Group

The aforementioned findings did not replicate for neither group, once the analyses were repeated independently. For a full description of these correlations see Table 8.

Table 8. *Partial correlations between WHOQOL-BREF and EF tasks*

	WHOQOL-BREF									
	Physical Health		**Psychological**		**Social Relationships**		**Environment**		**Total**	
	Group with TBI	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**	**Group with TBI**	**Control Group**
	Bivariate									
Rey Figure Copy	0.18	0.12	-0.14	-0.06	-0.33	-0.30	-0.03	0.02	-0.05	-0.03
TMT A	0.02	-0.16	-0.13	0.27	-0.15	0.36	-0.18	0.04	-0.12	0.13

TMT B	-0.03	0.07	-0.23	-0.07	-0.20	0.00	-0.09	-0.03	-0.15	-0.02
SDMT	-0.03	0.04	-0.27	-0.11	-0.21	0.07	-0.33	-0.08	-0.24	-0.05
COWAT Animal Naming	0.20	0.05	-0.04	-0.15	-0.22	0.19	-0.17	-0.05	-0.04	-0.02
COWAT Letter F	0.12	0.05	0.20	0.13	-0.01	-0.03	0.14	0.21	0.15	0.15
	Partial Controlling for Coping and Diathesis									
Rey Figure Copy	0.12	0.05	-0.15	0.21	-0.31	0.18	0.04	0.39	-0.04	0.29
TMT A	0.08	-0.06	0.05	0.39	0.02	0.33	-0.08	-0.07	0.02	0.12
TMT B	-0.16	0.09	-0.15	0.17	-0.11	0.11	-0.07	0.08	-0.16	0.13
SDMT	0.14	-0.18	-0.15	0.26	-0.02	0.01	-0.21	-0.15	-0.06	-0.05
COWAT Animal Naming	0.36	0.10	0.20	0.14	0.08	-0.03	-0.02	-0.13	0.22	0.00
COWAT Letter F	0.24	0.20	0.47	-0.37	0.15	-0.27	0.42	0.14	0.42	-0.03

** p<0.01; Group with TBI N=33; Control group=24.*

QOLIBRI

It was further sought to investigate possible associations with a specific measure of TBI HRQOL, completed by the group with TBI, only. No significant correlations were found between the EF tasks and the QOLIBRI (Table 9).

Table 9. *Partial correlations between QOLIBRI and EF tasks*

	QOLIBRI						
	Cognition	**Self**	**Daily Life & Autonomy**	**Social Relationships**	**Emotions**	**Physical Problems**	**Total**
	Bivariate						
Rey Figure Copy	-0.01	0.00	0.07	-0.30	-0.06	0.15	-0.02
TMT A	-0.15	-0.12	0.06	-0.31	-0.08	-0.02	-0.15
TMT B	-0.21	-0.09	0.07	-0.25	-0.01	-0.21	-0.18
SDMT	-0.16	-0.22	-0.11	-0.19	-0.23	-0.01	-0.22
COWAT Animal Naming	0.08	-0.01	-0.06	-0.15	-0.22	0.17	-0.04

COWA T Letter F	0.02	0.07	0.18	-0.05	-0.26	-0.12	-0.06
Partial Controlling for Coping and Diathesis							
Rey Figure Copy	-0.07	0.19	-0.04	-0.27	-0.11	0.19	-0.02
TMT A	-0.17	0.23	0.08	-0.22	-0.07	0.14	0.01
TMT B	-0.13	0.27	0.03	-0.15	0.02	-0.23	-0.08
SDMT	-0.16	0.08	-0.10	0.09	-0.30	0.16	-0.07
COWA T Animal Naming	0.30	0.28	0.08	0.13	-0.29	0.22	0.16
COWA T Letter F	0.16	0.34	0.25	0.07	-0.25	-0.02	0.09

** p<0.01; Group with TBI N=33.*

According to these findings, only one EF task associated with QOL suggesting that suggesting that individuals over performing on EF report greater satisfaction with their physical health. However, this being the only finding supporting the association between EF and QOL, it should be interpreted with caution.

Relationship between EF and Functional Outcome

Pearson's one-tailed correlations were conducted to investigate the association between the EF tasks and functional outcome, for the participants with TBI, revealing no significant associations (MPAI-4, Table 29, Appendix A; GOSe, see Table 30, Appendix A).

Summary

In summary, results examining the association between EF and SA in the entire sample support that greater performance in EF relates to greater SA on all three aspects of SA, as these were measured by the DEX-R (fluency, flexibility and working memory, social and self –regulation, and motivation and attention), as well as overall SA. These findings were not replicable for either group when the analyses were repeated separately. However, despite partialling out the associations of the COPE-Brief and the SRSDA, relationships between EF and SA were maintained, thus clearly depicting the true relationship between EF and SA, thus supporting that potential impairment in EF could related to deficits in SA.

Finally, it appears that individuals over performing on an EF task reported greater satisfaction with their physical health. It may be further argued that the fact that this finding was evident only after coping and diathesis were partialled-out, allows for a direct association between the two constructs to come forward. However, this being only one association

connecting EF and QOL, it cannot be generalized but rather interpreted as a possible finding in need for further investigation.

Self-awareness & Quality of Life

Further correlational analyses were conducted to investigate the relationship between SA and QOL for the entire sample (TBI and controls), and for each group, separately. However, due to the significant correlations with coping and mood –related concepts with SA and QOL, the latter variables were partialled-out, in order to grasp a clear understanding of the relationship between SA and QOL.

DEX-R & WHOQOL-BREF

Whole-Group

No significant partial associations were evident between the WHOQOL-BREF and the DEX-R for the whole-group, instigating that perhaps the associations between these constructs may be mediated by coping mechanisms and mood–related disorders (see Table 10).

Independent-Group

No significant findings were evident for the group with TBI, either (Table 10). However, for the control group significant associations were found showing that less satisfaction with physical health related to greater social and self –regulation SA, $r(12) = -0.73$, $p = 0.001$, and overall SA, $r(12) = -0.65$, $p = 0.005$. In addition greater social and self –regulation SA correlated with greater dissatisfaction with their psychological health, $r(12) = -0.65$, $p = 0.005$, and their social relationships, $r(12) = -0.63$, $p = 0.008$ (see Table 10).

Table 10. *Partial correlations between WHOQOL-BREF and DEX-R*

	DEX-R

	Fluency, Flexibility, Working Memory	Social & Self - Regulation	Motivation & Attention	Overall SA
		Bivariate		
Physical Health	0.02	0.11	0.20	0.11
Psychological	-0.27	-0.34*	-0.21	-0.31
Social Relationships	-0.16	-0.17	-0.05	-0.15
Environment	-0.27	-0.26	-0.23	-0.27
Total	-0.21	-0.20	-0.09	-0.19
		Partial Controlling for Coping and Diathesis		
Physical Health	0.11	0.10	0.31	0.17
Psychological	-0.26	-0.27	-0.08	-0.24
Social Relationships	-0.08	-0.06	0.09	-0.04
Environment	-0.20	-0.16	-0.12	-0.18
Total	-0.14	-0.13	0.06	-0.10

p<0.01; Whole-group, N = 57

	DEX-R							
	Fluency, Flexibility, Working Memory		Social & Self - Regulation		Motivation & Attention		Overall SA	
	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group
				Bivariate				
Physical Health	-0.03	-0.37	0.05	-0.73*	0.14	-0.53*	0.04	-0.72*
Psychological	-0.33	-0.25	-0.36*	-0.77*	-0.28	-0.15	-0.36*	-0.61*
Social Relationships	-0.28	0.32	-0.31	-0.18	-0.21	0.47	-0.30	0.11

Environment	-0.28	-0.15	-0.06	-0.70*	-0.13	-0.28	-0.16	-0.56*
Total	-0.26	-0.18	-0.18	-0.78*	-0.11	-0.22	-0.20	-0.61*
Partial Controlling for Coping and Diathesis								
Physical Health	0.11	-0.31	0.05	-0.73*	0.34	-0.27	0.14	-0.65*
Psychological	-0.16	-0.54	-0.22	-0.65*	-0.02	0.04	-0.17	-0.60
Social Relationships	-0.14	-0.56	-0.15	-0.63*	-0.02	0.14	-0.13	-0.57
Environment	-0.09	0.40	0.02	-0.18	0.02	-0.16	-0.01	-0.03
Total	-0.06	-0.15	-0.07	-0.59	0.14	-0.11	-0.02	-0.47

p<0.01; Group with TBI, N = 33; Control group, N = 24.

DEX-R & QOLIBRI

For this analysis only the group with TBI was used, as this is a measure of HRQOL specific to TBI population. For the DEX-R, three associations were evident with greater satisfaction with one's cognitive abilities correlating with greater SA deficits regarding social and self –regulation, $r(21) = -0.51$, $p = 0.001$, motivation and attention, $r(21) = -0.45$, $p = 0.005$, and overall SA impairment, $r(21) = -0.48$, $p = 0.003$. However, none of these associations survived once partialling-out the effects of coping and mood and anxiety –related problems (Table 11).

Table 11. *Partial correlations between QOLIBRI and DEX-R*

	DEX-R			
	Fluency, Flexibility, Working Memory	**Social & Self - Regulation**	**Motivation & Attention**	**Overall SA**

	Bivariate			
Cognition	-0.36	-0.51*	-0.45*	-0.48*
Self	-0.25	-0.28	-0.34	-0.30
Daily Life & Autonomy	-0.13	-0.21	-0.06	-0.16
Social Relationships	-0.12	-0.14	-0.07	-0.13
Emotions	-0.21	-0.06	-0.26	-0.16
Physical Problems	-0.26	-0.16	-0.11	-0.20
Total	-0.34	-0.33	-0.32	-0.36
	Partial Controlling for Coping and Diathesis			
Cognition	-0.13	-0.27	-0.23	-0.24
Self	0.06	-0.08	-0.08	-0.04
Daily Life & Autonomy	-0.07	-0.17	0.06	-0.10
Social Relationships	0.06	-0.06	0.09	0.01
Emotions	-0.14	-0.04	-0.19	-0.11
Physical Problems	-0.12	-0.03	0.09	-0.03
Total	-0.12	-0.16	-0.07	-0.14

p<0.01; Group with TBI, N = 33.

SRSI & WHOQOL-BREF & QOLIBRI

No significant correlations were evident between the SRSI indexes, and neither the WHOQOL-BREF, nor the QOLIBRI, for the group with TBI (see Table 31 for WHOQOL-BREF, and Table 32 for QOLIBRI, Appendix A).

Summary

Brought together, these findings support that greater SA and overall SA regarding social and self –regulation related to greater dissatisfaction with physical health. In addition greater social and self –regulation SA correlated with greater dissatisfaction with their psychological health, and their social relationships. Although these findings were evident for the control group, alone, they may allow inferences to be made regarding the group with TBI, i.e. that lower SA may relate to greater QOL.

Relationships between SA and QOL with Functional Outcome

The relationships between SA and functional outcome, and QOL and functional outcome were also investigated, for the group with TBI alone. No associations were found between the SRSI and the GOSe (Table 33, Appendix A). However, one significant correlation was detected between the SRSI and the MPAI-4, showing that greater levels of strategic SA related to greater ability outcome, $r(21) = 0.55$, $p = 0.003$ (Table 34, Appendix A).

The DEX-R showed significant associations with the MPAI-4. Specifically, findings showed that greater overall SA, and SA regarding executive cognition and social and self –

regulation related to better adjustment following a TBI (MPAI-4, Table 36, Appendix A). No correlations were shown between the DEX-R and the GOSe (Table 35, Appendix A).

For the QOL measures, findings supported that greater quality in one's physical health directly related to better ability, $r(21) = -0.55$, $p = 0.003$, adjustment, $r(21) = -0.56$, $p = 0.003$, and overall functional outcome, $r(21) = -0.58$, $p = 0.002$, as measured by the MPAI-4 Table 37, Appendix A), but not the GOSe (Table 38, Appendix A).

When comparing HRQOL to the GOSe, findings supported that greater quality in one's physical health relates to better functional outcome, $r(21) = 0.57$, $p = 0.002$ (Table 39, Appendix A); whereas no findings reached significance for the MPAI-4 (Table 40, Appendix A).

Regressions

Based on the previous findings, multiple stepwise regression analyses were conducted to identify which of these variables may best predict the perceived level of life satisfaction on the various domains independently, as measured by each scale obtained on the WHOQOL-BREF, and the QOLIBRI. The predictors of interest used were variables upon which the two groups showed significant differences or correlations: (i) SA, as measured by DEX-R (discrepancy scores); (ii) the COPE-Brief; (iii) the SRSDA; (iv) and neuropsychological performance on Verbal and Visual Memory, EF, and Attention (see below how these constructs were computed).

Standard score transformations were conducted to construct the four composite scores measuring neuropsychological performance. The scores from the neuropsychological battery conducted were combined into the conceptually derived four composite scores of Verbal, and Visual Memory, Executive Functions, and Attention/Organization. Initially, all scores were

valenced so that a lower score indicated worst performance. Individual test scores obtained from each participant were transformed into z-scores (standard score) based on the mean of all the participants. Finally, the calculated standard scores from each test were averaged to derive a score for each construct.

WHOQOL-BREF

Results from the stepwise regression analysis are displayed in Table 10 (unstandardized regression coefficients (B) and intercept, the standardized regression coefficients (β), the semi-partial correlations (sr^2), the adjusted R^2). The overall well-being of all participants, as measured by the Total scale of the WHOQOL-BREF, was predicted by short-term depression, mania, and visual memory, $F(2, 51)=13.10$, $p = 0.0001$, explaining 40% of the variability. Greater satisfaction with one's overall QOL may be predicted by lower levels of depression, $B = -1.22$, $t(55) = -4.13$, $p = 0.0001$, and more manic-like behaviors, $B = 2.23$, $t(55) = 2.40$, $p = 0.020$.

Physical health was predicted by problem-task coping and short-term depression, $F(2, 51) = 11.57$, $p = 0.0001$, Adjusted $R^2 = 0.31$. Specifically, more problem-task coping, $B = 0.27$, $t(55) = 2.06$, $p=0.045$, and less depressive symptomatology, $B = -0.44$, $t(55) = -4.09$, $p=0.0001$, predicted greater satisfaction with one's physical health.

Psychological well-being, as expressed through the WHOQOL-BREF, was significantly predicted by the DEX-R Social and Self –Regulation, avoidant coping, short depression, and mania, explaining 45.2% of the variability, $F(4, 49) = 11.93$, $p = 0.0001$. Specifically, more social and self –regulation SA deficits ($B = -0.95$, $t(55) = -3.16$, $p = 0.003$) and depression ($B = -0.29$, $t(55) = -3.21$, $p = 0.002$), as well as more manic traits ($B = 0.84$, $t(55) = 2.89$, $p = 0.006$) lead to greater satisfaction with one's psychological well-being.

Again depression, and asthenia predicted 0.36% of the Social Relationships aspect of one's life, $F(2, 53)=9.40$, p = 0.0001, with fewer depressive, B = -0.34, $t(55)$ = -4.64, p = 0.014, but more asthenia related symptoms, B = 0.18, $t(55)$ = 2.53, p = 0.005, predicting greater satisfaction with social relationships.

Finally, the environmental aspect of the WHOQOL-BREF was predicted by the DEX-R Social and Self-Regulation scale and mania, significantly contributing a 39% variability, $F(2, 51)$ = 15.95, p = 0.0001. Greater SA deficits in social and self-regulation, B = -0.17, $t(55)$ = -4.81, p = 0.0001, and more manic-related traits, B = 0.77, $t(55)$ = 2.52, p = 0.029, lead to more satisfaction with one's environmental aspect of their lives, as measured by the WHOQOL-BREF.

Table 12. *Stepwise regression of DEX-Discrepancy scores, Coping, Mood and Anxiety, Neuropsychological Constructs on each factor of WHOQOL–BREF.*

	Total						
	B	**β**	**sr2**	**F**	**p**	**R**	**AdjR²**
				13.10	0.0001	0.66	0.41
Short Depression	-1.22	-0.48	-0.44				
Mania	2.23	-0.23	-0.25				
	Physical Health						
	B	**β**	**sr2**	**F**	**p**	**R**	**AdjR²**
				12.00	0.0001	0.56	0.29
Problem-Task Coping	0.27	-0.24	0.24				
Short Depression	-0.44	-0.48	0.48				

Psychological Health							
	B	**β**	**sr2**	**F**	**p**	**R**	**AdjR²**
				12.00	0.0001	0.70	0.45
DEX-R Social & Self - Regulation	-0.10	-0.33	-0.32				
Avoidance Coping	-0.11	-0.09	-0.08				
Short Depression	-0.29	-0.36	-0.33				
Mania	0.84	0.34	-0.29				
Social Relationships	**B**	**β**	**sr2**	**F**	**p**	**R**	**AdjR²**
				9.40	0.0001	0.60	0.32
Short Depression	-0.34	-0.66	-0.53				
Asthenia	0.18	0.36	0.29				
Environment	**B**	**B**	**sr2**	**F**	**p**	**R**	**AdjR²**
				16.01	0.0001	0.62	0.36
DEX-R Social & Self - Regulation	-0.17	-0.53	-0.53				
Mania	0.77	0.25	0.24				

QOLIBRI

Given that the group with TBI completed one more questionnaire on HRQOL, a second sequential stepwise regression analysis was conducted to explore whether the same factors similarly loaded to the HRQOL dimensions extracted from the QOLIBRI. All analyses were repeated for every factor (Total, Cognition, Self, Daily Life and Autonomy, Social Relationships, Emotions, Physical) and all findings are reported in detail in Table 11.

The Total score of the QOLIBRI was significantly predicted by short depression, with 41% of the variability explained, $F(1, 30) = 20.87$, $p = 0.0001$, showing that fewer depressive symptoms lead to greater satisfaction with one's overall health-related quality of life, $B = -1.52$, $t(55) = -4.57$, $p = 0.0001$.

Subsequent analysis yielded revealed that the DEX-R social and self –regulation index and the mania significantly contributed to the Cognition scale with a 40.1% of the variability, $F(2, 29) = 9.71$, $p = 0.001$. Greater SA deficits regarding social and self –regulation, $B = -0.75$, $t(55) = -4.03$, $p = 0.0001$, accompanied by manic traits flexibility, $B = 1.17$, $t(55) = 2.60$, $p = 0.014$ predicted more satisfaction with cognitive abilities.

Depression predicted both the Self and the Daily Life and Autonomy scales of the QOLIBRI, significantly contributing a 40.2% of the variability of the former index and 19.7% to the latter ($F(1, 30) = 21.80$, $p = 0.0001$, and $F(1, 30) = 8.60$, $p = 0.006$, respectively). Individuals with more depressive symptomatology, were more likely to be less satisfied with their perception of their self ($B = -1.41$, $t(55) = -4.67$, $p = 0.0001$, long depression) and daily life and autonomy ($B = -1.80$, $t(55) = -2.93$, $p = 0.006$, short depression).

Better social relationships were predicted by greater emotion-focused coping, $B = 2.56$, $t(55) = 2.46$, p = 0.020, and fewer short depression symptoms, $B = -1.19$, $t(55) = -2.36$, p = 0.025, thus explaining 33.7% of the variability, $F(2, 29) = 7.37$, p = 0.003.

Finally, lower levels of asthenia were predictive of greater satisfaction with physical health, thus explaining 13% of the variance, $B = -2.02$, $t(55) = -2.12$, p = 0.043. However, this findings did not survive corrections, $F(1, 30) = 4.47$, p = 0.043.

Table 13. *Stepwise regression of DEX-Discrepancy scores, Coping, Mood and Anxiety, and Neuropsychological Constructs on each factor of QOLIBRI.*

Total							
	B	β	sr2	F	p	R	AdjR^2
Short Depression	-1.52	-0.64	-0.64	20.87	0.0001	0.64	0.39
Cognition							
	B	β	sr2	F	p	R	AdjR^2
				10	0.001	0.63	0.36
DEX-R Social & Self - Regulation	-0.75	-0.59	-0.58				
Mania	4.38	0.38	0.37				
Self							
	B	β	sr2	F	p	R	AdjR^2
Long Depression	-1.41	-0.30	-0.65	21.80	0.0001	0.65	0.40
Daily Life & Autonomy							
	B	β	sr2	F	p	R	AdjR^2
Short Depression	-1.80	-0.47	-0.47	8.60	0.006	0.47	0.20

Social Relationships							
	B	β	sr2	F	p	R	AdjR^2
				7	0.003	0.58	0.29
Emotion-Focused Coping	2.56	0.38	0.37				
Short Depression	-1.12	-0.37	-0.36				

Physical							
	B	β	sr2	F	p	R	AdjR^2
Asthenia	-2.02	-0.36	-0.36	4.47	0.043	0.36	0.10

Summary

In summary, stepwise regressions were performed to investigate for predictors of QOL and HRQOL dimensions. Greater overall QOL of all participants, as measured by the WHOQOL-BREF, was predicted by fewer short-term depressive and manic symptoms, explaining 40% of the variability; whereas for the group with TBI alone, greater satisfaction for one's overall well-being, measured by the QOLIBRI, was predicted by short depression alone, explaining a 41%.

All participants reporting greater satisfaction with their physical health were more likely to report more problem-task coping and less depressive symptomatology. For the group with TBI fewer symptoms of asthenia were predictive of greater satisfaction with physical health; however, this finding did not survive corrections.

Individuals' psychological well-being, as expressed through the WHOQOL-BREF was predicted by greater social and self –regulation SA deficits and depression, as well as more manic traits. Additionally, for the group with TBI, more SA deficits regarding social and self – regulation with more by manic traits were predictive of greater satisfaction with their

cognitive abilities. In addition, individuals with TBI who stated greater satisfaction with themselves were more likely to experience less depressive symptomatology.

Individuals with TBI were more likely to report more dissatisfaction with their social relationships given they had stated fewer emotion-focused coping and depressive symptomatology. These findings were somewhat similar for the entire sample, with individuals reporting less satisfaction with their QOL more likely experiencing more depression and asthenia –related symptoms.

Finally, greater SA deficits in social and self-regulation, and more manic-related traits lead to more satisfaction with one's environmental aspect of their lives, as measured by the WHOQOL-BREF. For the group with TBI, a similar scale of the QOLIBRI measuring greater daily life and autonomy was predicted by fewer depressive symptoms.

Discussion

Experiment 1 sought to investigate differences between EF, SA and QOL, and then further explore for possible associations between these constructs. As expected, individuals with TBI displayed significant executive dysfunction and neuropsychological impairment, as they underperformed on all neuropsychological tasks but visual memory tasks, compared to the non-injured controls.

Self-awareness was also assessed in both groups, not only using self-reports but also through collecting data from their family members/significant other. Three factors were extracted from the DEX-R, and the discrepancy scores for each index were further calculated. The group with TBI differed from the control group regarding SA on social and self – regulation and motivation and attention. Such discrepancy between the participants with TBI

and the controls on such behavioral patterns is indicative of lower SA, as reported in TBI literature (May et al., 2017; O'Keeffe et al., 2007).

It has also been hypothesized that low SA would promote similar response pattern regarding the participants' QOL, except for the physical aspects of health, as the individuals with TBI are expected to suffer certain physical trauma and related difficulties (Sasse et al., 2013; Sherer et al., 1998). Findings do, somewhat, support this hypothesis with participants with TBI having stated lower levels of satisfaction with their physical health, than the control group. Despite the posed predictions, the group with TBI reported greater satisfaction regarding their financial resources, their physical safety, their home environment, and other aspects included in the environment scale of the WHOQOL-BREF. This finding may be due to the lack of involvement of individuals with TBI with such aspects, as these are attended to mainly by the family members or main care provider. Findings with this Greek Cypriot sample are consistent with previous literature with US samples that investigated the burden of the care provider (Abrahamson, Jensen, Springett, & Sakel, 2017) and could provide support to the universal aspects of caregiver burden post TBI. Also, the lack of differentiation between the two groups on the social relationships dimension, and the psychological and overall well-being may be attributed to the lack of SA in individuals with TBI, who are often unaware of their cognitive disabilities and how these may impede their QOL (Sasse et al., 2013).

Coping mechanisms and mood disorders were also examined, as literature supports that these factors appear to influence neuropsychological performance, SA, and QOL (Brands et al., 2014a; Brands et al., 2014b). There were no significant differences in reported coping mechanisms between the two groups, contradicting past findings (Anson, & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011).

The group with TBI expressed greater levels of melancholic diathesis on average at 5.24post injury. Depression symptoms have been reported in moderate-severe TBI (Whelan-Goodinson et al., 2009) and could interfere with EF, SA, and QOL (see Toglia, & Golisz, 2017, pp. 117-143; Whelan-Goodinson et al., 2009).

Based on these findings, it was sought to examine the relationship between the aforementioned variables. However, despite not detecting differences in the coping mechanisms employed by the participants, this measure was examined whether it associates with EF, SA, and QOL, as it was previously reported in literature to relate to all three (Anson, & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011). Findings support that coping mechanisms employed significantly correlated with EF, SA, and QOL. Specifically, individuals performing higher on EF tasks use less emotion-focused coping techniques. Also, the non-injured participants with greater SA reported more ineffective coping strategies, i.e. avoidant coping, and fewer problem-task and emotion-focused coping mechanisms. In addition, coping mechanisms practiced by the control group, and specifically more avoidant and problem-task -related techniques were related to greater dissatisfaction with their psychological health and social relationships, respectively. It appears that the healthy adults using more ineffective coping techniques show greater SA and more dissatisfaction with specific aspects of their QOL.

Finally, participants with TBI declaring greater use of emotional coping reported greater satisfaction with their social relationships, as this was measured by a TBI-specific HRQOL measure. Following TBI, it appears that social relationships, mainly with their family members, play a significant role in managing one's condition (Anson, & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011). Therefore, it appears that coping techniques do relate to EF, SA, and QOL (Anson, & Ponsford, 2006; Brands et al., 2014b; Wolters et al.,

2011), despite the fact that the two groups do not differentiate on this measure, thus further complicating the associations between EF, SA, and QOL.

Mood and anxiety –related factors were also examined to detect whether they relate to the main variables under investigation. For EF, non-injured individuals over performing in a cognitive flexibility task reported less asthenia and anxiety -related symptomatology. In addition, whole-group analysis showed that greater SA regarding executive cognition and overall SA was associated with greater anxiety and asthenia reported difficulties. Also, greater dissatisfaction with one's QOL related to more depressive and melancholic issues. However, individuals expressing more manic-like behavior reported greater life satisfaction. These findings were replicated for the group with TBI regarding both their QOL and HRQOL. Brought together, these findings clearly indicate that mood and anxiety –related issues appear to implicate and thus complicate the associations between the constructs under investigation (Chiaravalloti, & Goverover, 2016).

As a result of the aforementioned findings, coping skills and mood and anxiety –related symptoms were partialled-out of the associations of interest. Findings support the association between EF and SA within the whole group, clearly showing that greater performance in EF relates to greater SA on fluency, flexibility and working memory, social and self –regulation, and motivation and attention, as well as overall SA. These findings were not replicable for either group when the analyses were repeated separately. However, it is worth mentioning that despite partialling-out the associations of the COPE-Brief and the SRSDA, relationships between EF and SA were preserved enhancing existing literature associating these constructs that allows informing existing biopsychosocial models linking the two (Fitzgerald et al., 2017; Ownsworth et al. 2007). Such evidence may be informative of the event that people with fewer EF deficits are more aware of their own disabilities (Hart et al.,

2005; Zimmermann et al., 2017), and enhance literature showing a direct association between impaired SA and executive dysfunction (Caldwell et al., 2014; Hart et al., 2005; Zimmermann et al., 2017), and that impaired SA may persist for years following an injury (Hoofien et al., 2004).

In this study, no solid evidence linking EF and functional outcome, were evident. Correlational analysis revealed that individuals who score higher on EF reported greater satisfaction with their physical health. Though this finding may be encouraging and could tempt the researcher to connect EF and QOL, the reader should be cautioned that the aforementioned finding was the result of one association alone and thus should be interpreted with caution until follow up studies provide more robust evidence. According to the literature, EF dysfunction associates with functional outcome, and QOL (see Rabinowitz, & Levin, 2014; Kelley et al., 2014).

One major inquiry of this study was to examine whether lower levels of SA would relate to QOL. Findings from this study, regarding the healthy controls alone, show that greater SA and overall SA regarding social and self –regulation related to greater dissatisfaction with physical health. In addition greater social and self –regulation SA correlated with greater dissatisfaction with their psychological health, and their social relationships. These results may somewhat support this hypothesis, as the negative association between the two constructs is depicted. It appears that healthy individuals with intact SA perceive their life as less satisfying, therefore it may be inferred that individuals with SA deficits could potentially report greater satisfaction with their QOL (Sasse et al., 2013; Mathias, & Wheaton, 2007).

Self-awareness was found to relate to functional outcome, with lower levels of SA, as described by the individuals with TBI themselves, correlating to poorer functional outcome. Also, associations between the informants' responses on the DEX-R and the functional outcome measures further enhanced the argument that SA and related behavioral deficits relate to worst functional outcome. This evidence has been reported by past literature (Chiaravalloti, & Goverover, 2016; Sherer et al., 2005).

Finally, the association between the HRQOL measure and functional outcome was examined. Findings indicated that when one deals with fewer physical challenges, it is more likely to report better adjustment and abilities, participate more, and present with better overall functional outcome. Additionally, if one maintains higher levels of daily life independence and autonomy, and overall QOL and HRQOL, they are more likely to have better adjustment, participation and overall functional outcome. This evidence is in coherence with Johnson et al. (2010), suggesting that greater satisfaction with one's QOL relates to better functional outcome in chronic TBI (Johnson et al., 2010).

One final hypothesis was to investigate whether QOL and HRQOL could be predicted by EF, SA, mood, and coping mechanisms. Findings revealed that all QOL and TBI-HRQOL specific dimensions extracted by the WHOQOL-BREF, and the QOLIBRI, respectively, were mainly predicted mainly by coping mechanisms, mood, and SA. Specifically, satisfaction with one's overall well-being was predicted by fewer short-term depressive and manic symptoms; whereas for the group with TBI alone, greater satisfaction for one's HRQOL was predicted by short depression alone. In addition, participants were more likely to report greater satisfaction with their physical health given the use of more problem-task coping and fewer depressive symptoms. For the group with TBI, however, satisfaction with physical health showed a predictive trend of asthenia; however, not reaching significance.

Psychological health, for the WHOQOL-BREF, was predicted by greater social and self –regulation SA deficits and depression, as well as more manic traits. The latter two predictors were significant for the group with TBI predicting greater satisfaction with their cognitive abilities. In addition, individuals with TBI who stated greater satisfaction with themselves were more likely to experience less depressive symptomatology.

Satisfaction with social relationships was predicted by greater use of emotion-focused coping and depressive symptomatology for people with TBI. These findings were somewhat similar for the entire sample, with individuals reporting less satisfaction with their QOL more likely experiencing more depression and asthenia –related symptoms.

Finally, greater SA deficits in social and self-regulation, and more manic-related traits lead to more satisfaction with one's environmental aspect of their lives, as measured by the WHOQOL-BREF. For the group with TBI, a similar scale of the QOLIBRI measuring greater daily life and autonomy was predicted by fewer depressive symptoms. Overall, these findings are of significance as no evidence has been reported to clearly depict predictors involve on QOL and HRQOL dimensions, including EF, SA, QOL (see Sasse et al., 2013).

CHAPTER 5

Experiment 2

Rationale

Traumatic brain injury (TBI) is a major cause of hospitalization, death, and chronic disability (CDC, 2015; Chiaravalloti, & Goverover, 2016; Faul et al., 2010). Chronic TBI-related disabilities constitute TBI as a chronic condition rather than a static one (Bigler, 2013; Chiaravalloti, & Goverover, 2016; Green et al., 2014; Masel, & Dewitt, 2010). Such disabilities include significant neuropsychological impairment (Constantinidou et al., 2008a; Green et al., 2014; Vakil & Lev-Ran Galon, 2014; see Vakil, 2013), including executive dysfunction, as well as deficits in self-awareness (SA; Caldwell et al., 2014; Chiaravalloti, & Goverover, 2016). Neuropsychological impairment is accompanied by chronic and progressive brain volume loss (Green et al., 2014; Konstantinou et al., 2016), as TBI may result in a pathophysiologic sequelae. This sequelae can be analogous to the location and severity of the damage, with cortical and sub-cortical areas such as frontal and anterior temporal, and the hippocampus, respectively, being more vulnerable to the trauma, due to their position within the skull (Blennow et al., 2012; McAllister, 2011; Povlishock, & Katz, 2005).

Greater injury severity leading to an increase in pathophysiology in both gray matter (GM) and white matter (WM) within the frontal lobes and related brain areas and circuits, has been found to affect the executive functions (EF), and SA, and thus one's ability to function adaptively (Kinnunen et al., 2011; Prigatano, 1991). Executive dysfunction and impaired SA have been linked to greater gray and white matter pathology in numerous frontal, temporal, parietal, and occipital cortical and sub-cortical regions, such as the cingulate cortex, medial frontal cortex, dorsolateral prefrontal cortices, the superior frontal gyri, ventrolateral prefrontal

cortex, the hippocampus, thalamus, insula and caudate (Merkley et al., 2013; O'Keeffe et al., 2007; Spitz et al., 2013; Taylor et al., 2007).

This chronic degenerative course in brain volume and lingering neuropsychological deficits, including low SA may explain the quality of life (QOL) outcome, with individuals with moderate-severe TBI over reporting their QOL levels. However, no evidence exists to attempt associating brain morphology with QOL. Therefore, the current study attempts to associate brain morphology with QOL measures and provide insight in this gap met in current TBI literature.

Statement of Purpose

Literature has identified associations between the various brain regions and neural circuits underlying impaired EF, and SA. Additionally, associations among EF, SA and QOL, have been reported. However, no evidence has been presented to describe the direct relationship between neuropathology and QOL.

Significance

Findings from Experiment 2 are expected to shed light to the enigmatic relationship of brain volume and QOL. Therefore, highlighting the chronic and persistent course of brain volume loss, EF, and thus SA deficits, and how these may further affect one's QOL will assist with the development of related biopsychosocial model and the design of more comprehensive rehabilitation programs by focusing on the impact of impairments on daily participation that would further improve patients' recovery.

Hypotheses

Hypothesis 1

It was hypothesized that participants with chronic TBI will sustain significant brain atrophy as demonstrated by significantly less GM and WM volume and greater CSF volume as compared to their non-injured counterparts.

Hypothesis 2

It was also examined whether the group with TBI will present with significant brain atrophy as expressed by less volume in cortical and subcortical areas, relating to EF, and SA, as compared to the control group. Past findings have shown that such areas include the frontal and temporal lobes and related structures such as the medial prefrontal cortex, the cingulate cortex, the frontal gyrus, the thalamus, the insula, and the caudate (Konstantinou et al., 2016; Prigatano, 1991).

Hypothesis 3

Given hypothesis 2, it was further hypothesized that greater brain volume atrophy in the frontal lobes and the associated structures, including the medial prefrontal cortex, the cingulate cortex, the thalamus, the caudate, and the cerebellum (Konstantinou et al., 2016; Prigatano, 1991) will positively correlate to and predict EF deficits, including impaired SA.

Hypothesis 4

Finally, it was expected that less brain volume in the frontal and temporal cortical and sub-cortical areas would negatively relate to QOL, in participants with TBI. No evidence has been reported exploring this association. However, it has been shown that people with TBI with greater brain volume loss display significant deficits regarding their neuropsychological performance, and SA deficits. Additionally, individuals with TBI who present with greater EF and SA deficits tend to report greater satisfaction with their QOL, mainly regarding their

cognitive abilities (Sasse et al., 2013). Therefore, it has been sought to investigate if less brain volume loss extends to over-reporting one's QOL levels.

Methods

Participants

The sample consisted of 57 native Greek speaking adults: 33 individuals with TBI, and 24 healthy individuals. However, two of the participants with TBI were excluded as they did not undergo MRI procedures. One participant was wearing braces, and the other had a hydrocephalus valve. Therefore, the finalized sample consisted of 31 individuals with TBI, with an age range of 18–51 years old. Participants with TBI were pair-matched to the non-injured controls on age, gender, and education. The patient group consisted of individuals who had sustained moderate-severe TBI at least one year post injury. All participants underwent a neuropsychological assessment, and a number of QOL and psychosocial measures. A control group was used to ensure that any changes in brain morphology, neuropsychological performance and QOL resulted from the injury. The types of questionnaires measuring QOL were selected to provide information specific to the effects of TBI. For a full description of the sample see Table 5 in Appendix A.

Procedure

Data collection procedures, including the neuropsychological and psychosocial assessment, were completed in eight months. The data was collected in a laboratory setting, where both the neuropsychological and QOL measures were administered individually. The neuropsychological testing lasted for 1.5-2 hours per participant with TBI, and 1-1.5 hours for the control group participants. During testing participants were provided with scheduled breaks in order to avoid mental fatigue. The QOL measures took about 45 minutes for the TBI

group and 10 minutes for the control group. Participants with TBI completed additional TBI-specific measures. The MRI data acquisition took about one hour per participant (see Methods in Chapter 3). No incidental findings were evident from the MRI images.

Materials

All participants underwent a battery of pen-and-paper neuropsychological assessment tools sensitive to cognitive deficits associated with TBI, and psychosocial questionnaires. All testing material was adjusted to Greek-native speakers. For a detailed description of the materials, see Chapter 3, Materials.

Neuropsychological Performance

Neurocognitive performance, i.e. memory, EF, attention, and cognitive reserve, was assessed using the following measures: (i) EF were assessed using the Rey Complex Figure Test (copy) (Rey, 1993), the Trail Making Tests A and B (also processing speed; Zalonis et al., 2008), the Symbol Digits Modalities Test (SDMT; Smith, 1982), and the phonological (letter F) and category recall (Animal recall) from the Control Oral Word Association Test (COWAT; Kosmidis et al., 2004); (ii) Verbal and visual memory was examined using the Digit Span Forward and Backwards and Visual Span Forward and Backwards (adapted Wechsler Memory Scale-III, WMS-III; Wechsler, 1997), the Greek adaptation of the Auditory Verbal Learning Test (AVLT; Constantinidou, & Evripidou, 2012a), the Rey Complex Figure Test immediate and delayed recall (Rey, & Osterrieth, 1993), the Greek Passage Memory test (which is based on the Wechsler Memory Scale Logical Memory subtest; Constantinidou, & Ioannou, 2008b); (iii) and Cognitive Reserve was assessed using the Peabody Picture Vocabulary Test (PPVT; Simos et al., 2011) and a reading measure assessing the total number of pseudowords correctly read in 45 s as measured by a test of pseudowords in Greek (Simos

et al., 2013). The Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005; Greek version Kounti, & Tsolaki (2006)) was also conducted as a screening tool to detect mild cognitive impairment, with a cut off score of 26 and lower.

Self-awareness

An additional two questionnaires were employed to detect SA deficits: (i) the Dysexecutive Questionnaire (DEX; Burgess et al., 1996), which was completed by both the participants themselves (group with TBI and control group; DEX-R-S), and an informant (both groups; DEX-R-I), i.e., a significant other or a family member; and (ii) the Self-Regulation Skills Interview (SRSI; Ownsworth et al., 2000) conducted in a semi-structured interview format with the individuals with TBI, only.

Quality of Life

Quality of life was assessed using a number of questionnaires that attempted to follow the ICF concepts in order to thoroughly describe the HRQOL phenomenon. Therefore, both a generic and a TBI-specific measure were used: the World Health Organization Quality of Life assessment instrument-BREF (WHOQOL-BREF; WHOQOL group, 1993), which was completed by all participants, covering many of the areas of the ICF; and the Quality of Life after Brain Injury (QOLIBRI; von Steinbuechel et al., 2012), specific to individuals with TBI. In addition, two functional outcome measures were employed: the *fourth edition* of the Mayo-Portland Adaptability Inventory 4 (MPAI-4; Malec, 2004b; completed by an informant) and the Galveston Outcome Scale Extended (GOSe; Wilson et al., 2007; completed by the participants with TBI, only), as HRQOL measures do not capture all areas of the ICF (Cieza, & Stucki, 2005).

Coping and Mood & Anxiety

Finally, a coping measure and a questionnaire concerning mood and anxiety –related issues were added to the testing package. Literature has shown that both coping mechanisms and depressive or anxious symptomatology, often experienced by individuals with TBI, may also affect one's self-awareness and executive dysfunction, and further affect QOL (Toglia, & Golisz, 2017, pp. 117-143). Thus, the Brief Cope (Carver, 1997) was conducted to assess coping strategies employed; whereas the Symptoms Rating Scale for Depression and Anxiety (SRSDA; Fountoulakis et al., 2003) was used to detect potential depressive or anxious symptomatology.

Magnetic Resonance Imaging Protocol

Image acquisition. MR images were acquired with a 3.0 Tesla scanner (Achieva, Philips Medical Systems, Best, The Netherlands). The built-in quadrature RF body coil and a phased array 8-channel head coil was used for proton excitation and signal detection, respectively. An isotropic, three-dimensional (3D), T1-weighted rapid acquisition gradient-echo sequence (fast field echo; repetition time = 25ms; echo time =1.85 ms; flip angle = 30^{o}) allowed for acquiring whole brain, transverse MR images with an acquisition/reconstruction voxel of 1.0×1.0 ×1.0 mm (data interpolation was not implemented in any direction to improve resolution and reduce partial volume effects). The scanning session included other standard pulse sequences (e.g., T2-weighted turbo spin echo, diffusion weighted imaging and fluid-attenuated inversion recovery) to exclude significant brain pathology of a different etiology.

Statistical analysis

Prior to performing any analyses, preprocessing of the MR images was conducted using SPM 12. Preprocessing of the MR images was conducted using SPM 12, prior to

performing Voxel-Based Morphometry (VBM) analysis. Preprocessing steps included segmentation of the MR images into GM and WM, followed by a Diffeomorphic Anatomical Registration through Exponentiated Lie Algebra (DARTEL) for inter-subject registration of the GM and WM images. Local GM and WM volumes were conserved by modulating the image intensity of each voxel by the Jacobian determinants of the deformation fields computed by DARTEL. The registered images were, then, smoothed with a Gaussian kernel (Full Width at Half Maximum = 8 mm) and were further transformed to Montreal Neurological Institute (MNI) stereotactic space using affine and nonlinear spatial normalization implemented in SPM12 for statistical comparisons.

Volumetry was used to detect group differences in overall GM, WM, and CSF volume, using IBASPM to calculate individual brain volume. These indexes allowed for the quantification of tissue volumetric changes between the two groups. Indexes were entered into SPSS and MANOVA was conducted compare the individuals with TBI to the non-injured participants.

Voxel-based-morphometry (VBM) analyses were conducted to investigate whether significant volume reduction in whole-brain regions was evident between the group with TBI and the control group. These hypotheses were tested through conducting two samples t-test models in SPM12, with age, education and overall brain volume entered as covariates of no interest.

Further analyses were conducted to investigate the associations between the volume in EF and SA –related brain regions and neuropsychological performance, and psychosocial measures. Specifically, regression analyses were performed to investigate the predictive validity of brain volume in regions-of-interest (ROIs) in executive dysfunction, impaired SA, and QOL. Therefore, masks of brain regions relating specifically to EF, and SA were

downloaded from the database of Neurosynth.org (see Figure 1 & Figure 2). These maps were then entered into MRICRON and individual masks of each ROI were hand-drawn. Each mask was then used to extract the volume from each ROI, using MATLAB (see an example of this process in Figure 3). Data were then entered into SPSS, to compare differences in these volumes (ROIs), using MANCOVA, with age, education and overall brain volume entered as covariates of no interest. Finally, stepwise regression analyses were performed to investigate the predictive validity of brain volume in ROIs in executive dysfunction, impaired SA, and QOL.

Results

Demographics

For Experiment 2, the group with TBI consisted of 31 male adults, with a mean age of 31.48 (SD = 8.54), and a mean level of education of 12.55 (SD = 2.97). A full description of each participant with TBI is provided in Table 5 (Appendix A). The control group consisted of 24 male participants, with a mean age of 31.92 (SD = 8.18), and a mean educational level of 13.63 (SD = 2.48).

Group Comparisons

The two groups were very similar in terms of age and education (age, $t(53) = -0.190$, $p = 0.850$; education, $t(53) = -1.432$, $p = 0.158$; two-tailed two-samples t-tests). Therefore, any significant differences in subsequent analyses cannot be attributed to sample differences.

Volumetry

As hypothesized the two groups differed significantly in mean GM volume, $t(53) = -3.20$, $p = 0.002$, Cohen's $d = 0.85$, with the group with TBI presenting with less GM volume ($M = 635.62$ cm^3, $SD = 63.22$ cm^3) as compared to the control group ($M = 701.26$ cm^3, $SD =$

88.94 cm^3). Similar findings were also shown for mean WM volume with the group with TBI showing significantly less volume (M = 392.51 cm^3, SD = 74.56 cm^3) than the non-injured individuals (M = 495.72 cm^3, SD = 59.98 cm^3), $t(53) = -5.53$, p = 0.0001, Cohen's d = 1.53.

As an effect of the reduction in volume in both gray and white matter, the CSF volume was significantly larger in the group with TBI (M = 367.53 cm^3, SD = 76.19 cm^3), than the control group (M = 273.93 cm^3, SD = 60.16 cm^3), $t(53) = 4.94$, p = 0.0001, Cohen's d = 1.36. See Figure 1 for a graphic representation of the gray, white, and CSF volumes.

Voxel-Based Morphometry

Whole-brain Analysis

Whole-brain analysis was further conducted in order to distinguish between brain areas that significantly differed in GM and WM, between the two groups. The group with TBI showed significantly less volume in GM in the left Medial Frontal Cortex, and the left Middle Frontal Gyrus, than the non-injured group (Table 14). Reduced volume was also observed in the right cerebral WM for the group with TBI, compared to the controls (Table 14). No brain regions showed significantly greater GM or WM volume in the group with TBI, as compared to the control group.

Table 14. *Whole-brain VBM*

Anatomical Region	Tissue Type	Side	MNI Coordinates			Peak-z	p-value
			x	y	z		
Medial Frontal Cortex	GM	L	-2	33	-18	7.67	0.002

Medial Frontal Gyrus	GM	L	45	-22	0	7.62	0.001
Cerebral	WM	R	30	57	3	7.36	0.002

ROIs

Neural systems involved with the concepts of EF and 'Self' have been detected in healthy adults and are presented in the Neurosynth Database. A meta-analyses brain map of 97 studies for EF and one of 903 studies for the 'Self' concept were extracted from the NeuroSynth database, revealing neural substrates involving these two concepts, separately (see Figure X; http://www.neurosynth.org; Yarkoni et al., 2011). The map relating to EF identifies clusters mainly in the bilateral frontal and temporal cortical and subcortical regions. Similar areas, including the parietal cortex, seem to be involved with the term "self". Therefore, specific Regions of Interest (ROIs) were investigated for group differences in EF and Self -related structures, using the meta-analyses masks extracted from Neurosynth.org (see Figure 1).

Executive Functions

Note. To account for multiple statistical tests, the a level was reduced to .01 (Bonferroni α' = a/k where k is number of tests; α' = .05/20 = 0.0025).

Multivariate ANCOVA was conducted with age, educational level, and global volume entered as covariates. A group effect was found on the EF brain regions, $F(20, 31) = 3.05$, $p<0.01$, $\eta^2 = 0.66$, with the group with TBI showing significantly less volume in a great number of cortical and sub-cortical areas involved in EF, as compared to the non-injured controls, including the left globus pallidus, the orbitofrontal cortex (OFC), the left primary sensory cortex, the putamen, the temporal cortex, the right temporal pole, the left thalamus,

the insula, the caudate, the right cingulate cortex, and the medial prefrontal cortex (MPFC) (see Table 15 below and Figure 2). For a detailed description of the areas extracted see Table 41 (Appendix A).

Executive Functions									
		Group with TBI		Control Group		Statistics			
Areas	Side	M	SD	M	SD	*F*	P	η^2	Observed Power
Brodmann's 8	L & R	.33	.04	.36	.05	11.63	.001	.19	.92
Globus Pallidus	L	.08	.01	.09	.01	29.48	.000	.37	1.00
OFC	L & R	.35	.05	.43	.06	33.31	.000	.40	1.00
Primary Sensory Cortex	L	.46	.07	.53	.11	10.35	.002	.17	.88
Putamen	L & R	.27	.03	.31	.05	17.02	.000	.25	.98
Temporal Cortex	L & R	.45	.06	.51	.06	24.68	.000	.33	1.00
Temporal Pole	R	.40	.08	.47	.06	17.32	.000	.26	.98
Thalamus	L	.40	.08	.49	.10	15.34	.000	.23	.97
Insula	L & R	.45	.06	.54	.07	32.15	.000	.39	1.00
Caudate	L & R	.20	.04	.24	.04	21.36	.000	.30	.99
Cingulate Cortex	R	.44	.07	.51	.09	13.39	.001	.21	.95
MPFC	L & R	.33	.04	.36	.05	11.56	.001	.19	.92

Table 15. *Group differences in EF-related brain regions*

p < 0.0025; Group with TBI, N = 33; Control group, N=24.

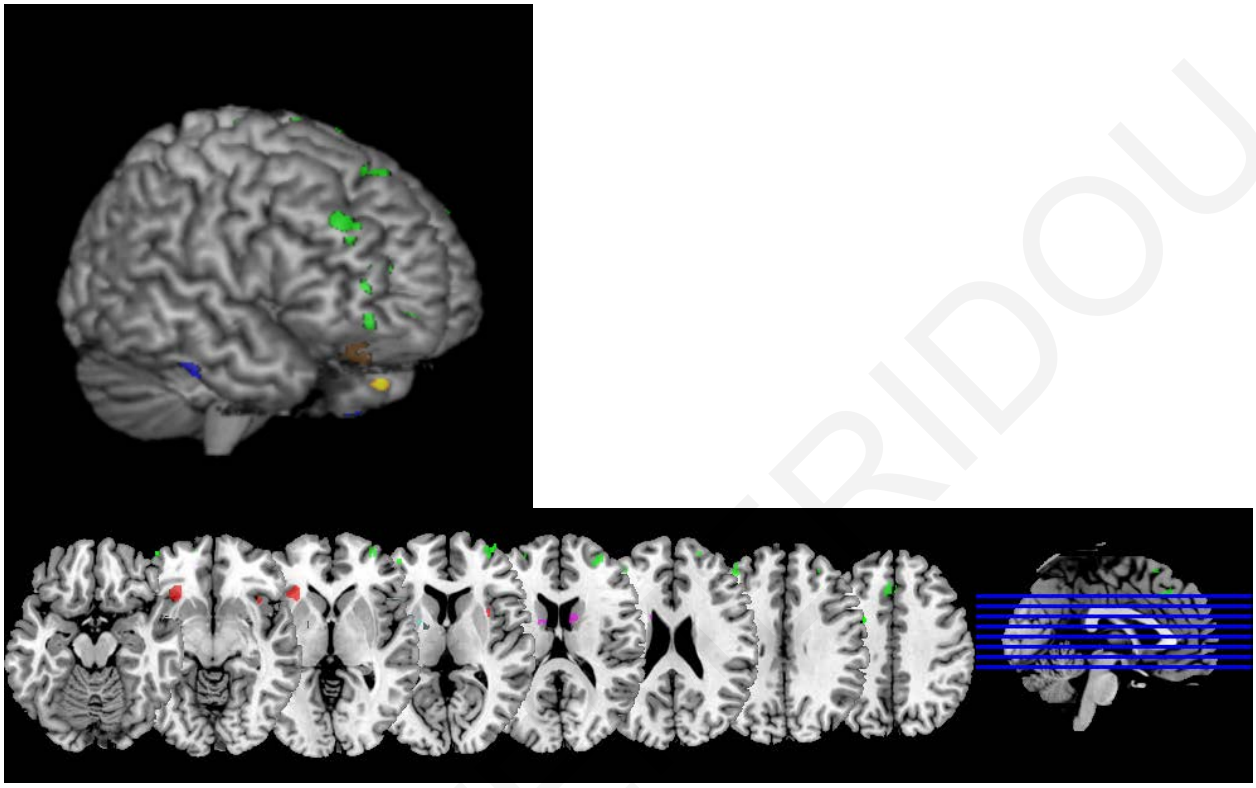

Figure 2. *Differences in EF-related brain regions*

Self-Awareness

Note. To account for multiple statistical tests, the a level was reduced to .01 (Bonferroni α' = a/k where k is number of tests; α' = .05/20 = 0.0023).

A similar analysis was used to investigate differences in regions relating to the concept of 'Self', between the two groups. When controlling for age, educational level, and overall brain volume, differences in areas relating to the Self were detected between the two groups, $F(25, 26) = 2.33$, $p<0.05$, $\eta^2 = 0.69$. Specifically, the group with TBI displayed significantly less brain volume in numerous brain regions, such as the OFC, the cingulate cortex, the temporal cortex and pole, the inferior frontal gyrus, the MPFC, and the hippocampus bilaterally, as well as the left insula, the left putamen, the pons, and the right globus pallidus,

compared to the control group (Table 16 or for full results see Table 42, Appendix A; see Figure 3 for graphic dipslay).

Table 16. *Group differences in SA-related brain regions*

	Self-awareness								
		Group with TBI		Control Group		Statistics			
Areas	Side	M	SD	M	SD	*F*	P	η^2	Observed Power
Basal Ganglia	L & R	.23	.03	.27	.04	30.61	.000	.38	1.00
Brodmann's 8	L & R	.35	.04	.43	.07	31.74	.000	.39	1.00
Corpus Callosum	L & R	.27	.04	.33	.05	34.00	.000	.40	1.00
Globus Pallidus	R	.10	.01	.11	.01	15.65	.000	.24	.97
Hippocampus	L & R	.51	.06	.58	.05	30.27	.000	.38	1.00
Inferior Frontal Gyrus	L & R	.48	.06	.53	.06	10.77	.002	.18	.90
Insula	L	.50	.05	.57	.08	22.35	.000	.31	1.00
MPFC	L & R	.35	.04	.41	.06	28.71	.000	.36	1.00
OFC	L & R	.41	.06	.52	.09	35.87	.000	.42	1.00
Putamen	L	.39	.04	.45	.05	31.86	.000	.39	1.00
Cingulate Cortex	L & R	.44	.05	.53	.08	29.62	.000	.37	1.00
Temporal Area	L & R	.19	.04	.23	.06	15.22	.000	.23	.97

Temporal Cortex	L & R	.42	.06	.49	.06	28.15	.000	.36	1.00
Temporal Pole	L & R	.41	.07	.47	.06	18.06	.000	.27	.99

p < 0.0023; Group with TBI, N = 33; Control group, N=24.

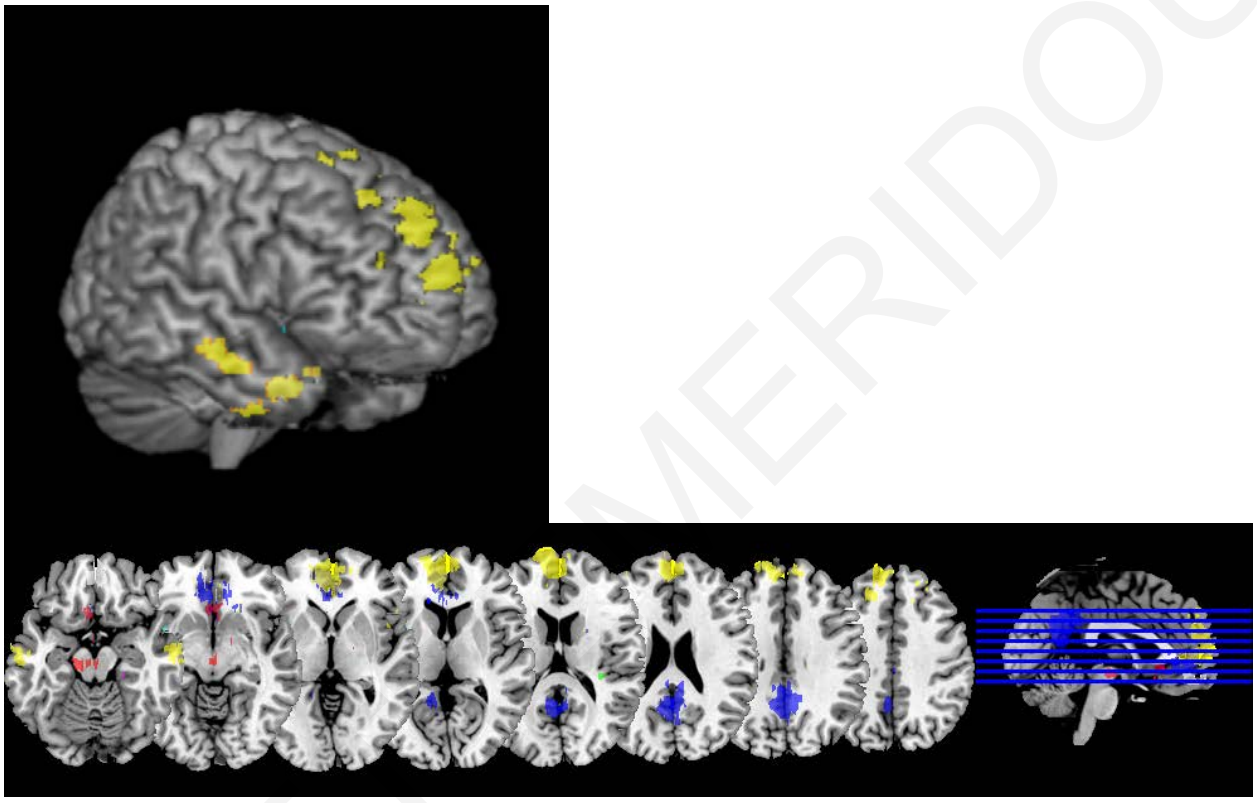

Figure 3. *Differences in SA-related brain regions*

In summary, MANCOVA revealed significant differences between the two groups, with the control group presenting with greater volume in EF and SA –related cortical and sub-cortical regions, as compared to the group with TBI; with EF and SA-related brain areas sharing common neuropathology OR neurological sequelae.

Taken together, the results of the whole-brain analysis revealed specific areas, such as the left MFC, and middle frontal gyrus, and the right cerebral WM, that are also detectable in the ROIs analyses; whereas additional areas were detected using the ROIs extracted from the Neurosynth masking process, including the OFC, the putamen, the caudate, the globus

pallidus, the insula, the thalamus, the hippocampus, the parahippocampus, the temporal cortex and pole, the primary sensory cortex, the fusiform gyrus, the cingulate cortex, the angular area, the frontal cortex, the visual association area, and the cerebellum.

Individual Differences Analyses

Given the findings in Experiment 1, i.e. differences in the psychosocial measures between the two groups, it was sought to examine whether these differences were reflected on the neural substrates involving these behaviors, for the areas where the group with TBI signified lower brain volume in comparison to the non-injured individuals.

For this reason, one-tailed Pearson correlational analyses were conducted to investigate the relationship between the regional volume and executive dysfunction and the psychosocial measures, separately. For the variables presenting with significant correlations ($r >= \pm 0.4$), multiple regression analyses were further conducted to examine the predictive validity of the regional volumes on EF, SA, and QOL. Corrections were applied to the correlation and regression analyses due to the numerous comparisons per ROI, thus the α level was lowered to 0.01.

EF-related regions & SA-related regions

Initially, it was sought to examine whether the EF-related regions correlated with the SA-related regions, for the entire sample. As expected, EF and SA –related brain areas showed significant associations, despite corrections for multiple comparisons, supporting that executive dysfunction and impaired SA may share a similar morphological sequelae (see Table 43, Appendix A).

Executive Functions

EF-related regions & EF construct

Whole-Group

One-tailed Pearson correlational analyses revealed significant positive associations between EF-related brain regions and the EF construct, with participants with higher scores in EF tasks presenting with greater volume in the globus pallidus, $r(50) = 0.35$, $p = 0.005$, the OFC, $r(50) = 0.33$, $p = 0.008$, the temporal cortex, $r(50) = 0.40$, $p = 0.002$, the thalamus, $r(50) = 0.33$, $p = 0.008$, the insula, $r(50) = 0.38$, $p = 0.003$, the cingulate cortex, $r(50) = 0.37$, $p = 0.003$, and the caudate, $r(33) = 0.46$, $p = 0.0001$ (Table 44, Appendix A). However, when stepwise regression was conducted, only the caudate was predictive of EF, $F(1, 53) = 14.22$, $p = 0.0001$, explaining 33% of the variance. Specifically, greater volume in the caudate could predict better performance in EF tasks, $B = 8.39$, $t(54) = 4.10$, $p = 0.0001$.

Independent-Group

No significant correlations were found between the EF-related brain regions and the EF construct, for neither group (Table 44, Appendix A). Therefore, no regression analysis was conducted.

EF-related regions & SA measures

DEX-R

Whole-Group

Significant associations were found between each of the DEX-R discrepancy ratings measured and brain regions relating to EF (Table 45, Appendix A). Specifically, significant associations were detected between the orbitofrontal cortex (OFC) and SA cognition ($r(50) = 0.34$, $p = 0.006$), social and self –regulation SA ($r(50) = 0.46$, $p = 0.0001$), attention and

motivation SA ($r(50) = 0.38$, $p = 0.003$), and overall SA ($r(50) = 0.43$, $p = 0.001$). In addition, greater volume in the globus pallidus was significantly related to greater social and self –regulation SA ($r(50) = 0.38$, $p = 0.003$), attention and motivation SA ($r(50) = 0.36$, $p = 0.004$), and overall SA ($r(50) = 0.34$, $p = 0.007$). Increased volume in the putamen related to greater social and self –regulation SA ($r(50) = 0.34$, $p = 0.007$). Volume in the temporal cortex significantly related to motivation and attention –related SA, $r(50) = 0.38$, $p = 0.003$. The temporal pole significantly correlated with all four indexes of the DEX-R (Fluency, Flexibility & Working memory, $r(50) = 0.49$, $p = 0.0001$; Social & Self –Regulation, $r(50) = 0.51$, $p = 0.0001$; Motivation & Attention, $r(50) = 0.53$, $p = 0.0001$; Overall SA, $r(50) = 0.55$, $p = 0.0001$). Insular volume was associated with SA skills relating to social and self –regulation ($r(50) = 0.38$, $p = 0.003$), motivation and attention ($r(50) = 0.41$, $p = 0.001$), and overall SA ($r(50) = 0.38$, $p = 0.003$). In addition, the caudate correlated with social and self –regulation SA ($r(50) = 0.41$, $p = 0.001$), motivation and attention SA ($r(50) = 0.37$, $p = 0.005$), and overall SA ($r(50) = 0.39$, $p = 0.002$). Finally, greater volume in the cingulate cortex was significantly correlated with greater social and self –regulation SA ($r(50) = 0.33$, $p = 0.008$), and greater overall SA ($r(50) = 0.33$, $p = 0.008$).

Further regression analyses revealed that the three indexes extracted from the DEX-R were predicted by the temporal pole. Greater volume in the temporal pole predicted more SA relating to the participants' abilities of social and self –regulation ($F(1, 53) = 11.10$, $p = 0.001$, Adjusted $R^2 = 0.27$, $B = 72.18$, $t(55) = 4.01$, $p = 0.0001$), motivation and attention ($F(1, 53) = 21.89$, $p = 0.0001$, Adjusted $R^2 = 0.28$, $B = 36.87$, $t(55) = 4.68$, $p = 0.0001$), and overall SA ($F(1, 53) = 13.64$, $p = 0.0001$, Adjusted $R^2 = 0.32$, $B = 152.166$, $t(55) = 4.49$, $p = 0.0001$).

Independent-Group

Similar findings were evident for the group with TBI with greater volume in the temporal pole associating with greater SA regarding executive cognition ($r(26) = 0.59$, $p = 0.0001$), social and self –regulation ($r(26) = 0.47$, $p = 0.006$), and motivation and attention abilities ($r(26) = 0.52$, $p = 0.002$), and overall SA ($r(26) = 0.55$, $p = 0.001$; Table 45, Appendix A).

However, as only one area correlated with each of the indexes, no regression analysis was further conducted. In addition, no significant correlations were revealed between the DEX-R discrepancy scores and the EF-related brain regions extracted, for the control group (Table 45, Appendix A).

SRSI

For the SRSI, using the participants with TBI, alone, both the putamen and the parahippocampal region were significantly correlated to both strategic awareness ($r(26) = -0.55$, $p = 0.001$, and $r(26) = -0.48$, $p = 0.005$, respectively) and emergent/online awareness ($r(26) = -0.51$, $p = 0.001$, and $r(26) = -0.46$, $p = 0.007$, respectively). In addition, decreased volume in Brodmann's 8 ($r(26) = -0.45$, $p = 0.008$), and the cingulate cortex ($r(26) = -0.44$, $p = 0.009$) was associated to lower strategic awareness. Finally, reduced volume within the temporal pole and the caudate was related to lower strategic ($r(26) = -0.49$, $p = 0.004$, and $r(26) = -0.46$, $p = 0.006$, respectively) and emergent/online awareness ($r(26) = -0.49$, $p = 0.004$, and $r(26) = -0.44$, $p = 0.009$, respectively; Table 46, Appendix A).

When stepwise regression analyses were conducted for each index, the putamen and Brodmann's area 8 appeared to hold predictive value for the strategy awareness index ($F(1, 30) = 10.62$, $p = 0.002$, Adjusted $R^2 = 0.39$), supporting that participants with TBI with less volume in the putamen ($B = -23.43$, $t(30) = -2.78$, $p = 0.009$) and Brodmann's 8 ($B = -16.16$, $t(30) = -2.66$, $p = 0.009$) were more likely to present with greater SA deficits in strategic

awareness. Furthermore, findings revealed that greater emergent/online SA impairment was predicted by reduced volume in the caudate (B = -20.97, t(30) = -2.64, p = 0.006) and the parahippocampal region (B = -14.41, t(30) = -2.13, p = 0.009), F(2, 30) = 8.70, p = 0.001, Adjusted R^2 = 0.34.

Summary

In summary, the regression analyses revealed that specific brain areas involved in executive functioning, i.e. the caudate, the insula, Brodmann's 8, the parahippocampal region, the temporal pole, and the putamen, may be predictive of performance in EF tasks, social and self –regulation, motivation and attention, strategic, emergent/online and overall awareness.

EF-related regions & QOL

WHOQOL-BREF

Whole-group & Independent-Group

No significant associations were found between any of the WHOQOL-BREF indexes and the EF–related brain regions, for neither the entire group (Table 47, Appendix A) nor the group with TBI or the non-injured controls (Table 48, Appendix A). Therefore, no regression analyses were further conducted for each group.

QOLIBRI

For the QOLIBRI, less volume in the left primary sensory cortex correlated with reporting of greater physical, r(26) = -0.56, p = 0.001, and overall HRQOL, r(26) = -0.55, p = 0.001. In addition, reduced insular volume significantly correlated with greater satisfaction with one's perception of self and overall HRQOL (r(26) = -0.48, p = 0.005, and r(26) = -0.44, p = 0.009, respectively; Table 49, Appendix A).

Regression analysis revealed that satisfaction with one's overall HRQOL was explained by the left primary sensory cortex, $F(1, 30) = 10.37$, $p = 0.000$, adjusted $R^2 = 0.34$. Individuals with reduced volume in the primary sensory cortex are more likely to report better subjective experience regarding their overall HRQOL, $B = -19.10$, $t(30) = -4.05$, $p = 0.0001$.

Summary

In conclusion, the primary sensory cortex, as implicated in EF-related brain regions, may hold predictive value on overall HRQOL.

Self-awareness

Given the associations yielded between EF and SA –related areas, and the relationships between the EF-related brain regions and the psychosocial measures, it was further investigated whether the SA-related brain areas, varying in volume between the two groups, could predict SA and QOL, as measured by DEX-R (self and informant -ratings), the SRSI, the WHOQOL-BREF, and the QOLIBRI.

Initially, one-tailed Pearson correlations were conducted to examine whether the SA-related regions were associated with any of these measures. For the areas and psychosocial indexes showing associations stepwise regression analyses was conducted to investigate the predictive value of the brain areas on the psychosocial indexes.

SA-related regions & SA

DEX-R

Whole-Group

For the DEX-R discrepancy scores, correlations revealed significant small to moderate associations between the Social and Self –Regulation index and the basal ganglia, $r(50) =$

0.39, p = 0.002, Brodmann's area 8, $r(50) = 0.40$, p = 0.002, the OFC, $r(50) = 0.44$, p = 0.001, the temporal cortex, $r(50) = 0.44$, p = 0.001, the temporal pole, $r(50) = 0.39$, p = 0.002, the temporal area, $r(50) = 0.36$, p = 0.004, the inferior frontal gyrus, $r(50) = 0.43$, p = 0.001, the putamen, $r(50) = 0.33$, p = 0.008, the hippocampus, $r(50) = 0.38$, p = 0.003, the corpus callosum, $r(50) = 0.40$, p = 0.002, and the MPFC, $r(50) = 0.40$, p = 0.002. Also, greater volume in the temporal cortex, $r(50) = 0.37$, p = 0.004, and the parahippocampal region, $r(50) = 0.35$, p = 0.006, related to greater SA cognition. In addition, greater awareness regarding motivational and attentional abilities was correlated with greater volume in the basal ganglia, $r(50) = 0.35$, p = 0.006, the OFC, $r(50) = 0.33$, p = 0.009, the insula, $r(50) = 0.36$, p = 0.005, the temporal pole, $r(50) = 0.37$, p = 0.004, the temporal cortex, $r(50) = 0.42$, p = 0.001, the inferior temporal gyrus, $r(50) = 0.36$, p = 0.004, the hippocampus, $r(50) = 0.39$, p = 0.002, and the pons, $r(50) = 0.34$, p = 0.008. For all correlations see table 50 in Appendix A.

Variables greater than r = 0.4 were entered into a regression model revealing that only the OFC appeared to hold predictive value for this index, $F(1, 54) = 8.89$, p = 0.0001, explaining just 22.6% of the variability. It appears that individuals with greater volume in the OFC were more likely to present with greater SA of social and self –regulation abilities, $B = 59.43$, $t(30) = 3.46$, p = 0.001.

Independent-Group

Independent one-tailed Pearson's correlation analyses were conducted for each group separately. For the group with TBI, greater volume in the temporal cortex related to better SA cognition, $r(26) = 0.47$, p = 0.006. However, due to the presence of one single correlation no regression was conducted. No significant associations were evident for the control group (Table 50, Appendix A). Thus, no regression analyses were performed.

SRSI

For the group with TBI, the scale measuring one's readiness to change showed a significant correlation with the right globus pallidus, $r(26) = -0.52$, $p = 0.002$, the hippocampus, $r(26) = -0.48$, $p = 0.005$, and the pons, $r(26) = -0.47$, $p = 0.006$. In addition, reduced strategic awareness was significantly correlated with less volume in the primary visual cortex, $r(26) = -0.53$, $p = 0.002$, the temporal cortex, $r(26) = -0.51$, $p = 0.003$, the cingulate cortex, $r(26) = -0.46$, $p = 0.006$, the temporal pole, $r(26) = -0.48$, $p = 0.009$, and the MPFC, $r(26) = -0.45$, $p = 0.009$. Finally, lower emergent/online awareness was associated with reduced volume in the primary visual cortex, $r(26) = -0.53$, $p = 0.002$, the temporal cortex, $r(26) = -0.48$, $p = 0.005$, the temporal pole, $r(26) = -0.46$, $p = 0.007$, and the temporal area, $r(26) = -0.50$, $p = 0.003$ (Table 51, Appendix A).

Stepwise regression revealed that individuals with TBI were most likely to be motivated to change given that they presented with less volume in the globus pallidus, $F(1, 30) = 9.53$, $p = 0.004$, adjusted $R^2 = 0.22$, $B = -109.70$, $t(30) = 3.09$, $p = 0.004$. Also, strategic awareness was predicted by the cingulate cortex, $F(1, 30) = 11.81$, $p = 0.002$, adjusted $R^2 = 0.27$, $B = -17.44$, $t(30) = -3.44$, $p = 0.002$, indicating that reduced volume in the cingulate cortex leads to greater strategy –related SA deficits. Finally, greater impairment regarding emergent/online SA was predicted by less volume in the temporal area, $B = -22.97$, $t(30) = -2.72$, $p = 0.004$, and the primary visual cortex, $B = -19.31$, $t(30) = -2.45$, $p = 0.005$, $F(2, 30) = 10.21$, $p = 0.0001$, adjusted $R^2 = 0.38$.

Summary

Summarizing these findings, it appears that in individuals with TBI readiness to change relates to the right globus pallidus, with greater volume in this area predicting less motivation to change. In addition, more deficits in emergent/online SA are predicted by

reduced volume in the temporal area and primary visual cortex. Finally, less volume in the cingulate cortex was predictive of greater strategy –related SA impairment. From these results it may be inferred that the greater the volume, the fewer deficits in SA, thus greater awareness of the effort required to manage possible difficulties, and thus one would be less motivated in engaging in further treatment.

SA-related regions & QOL

WHOQOL-BREF

Whole-Group

Significant correlations were found between the temporal area and the psychological, $r(50) = -0.36$, $p = 0.008$, and environmental scales, $r(50) = -0.41$, $p = 0.003$. Also, one significant association was found between the hippocampus and the environmental scale, $r(50) = -0.40$, $p = 0.004$ (Table 52, Appendix A).

Regression analysis revealed that all participants with increased volume in the hippocampus are more likely to report less satisfaction with the environmental aspects of their lives, $F(1, 30) = 7.45$, $p = 0.001$, adjusted $R^2 = 0.20$, $B = -29.47$, $t(30) = -2.73$, $p = 0.001$.

Independent-Group

One significant correlation was evident for the group with TBI, suggesting that individuals with less volume in the temporal area reported greater satisfaction with their psychological health, $r(26) = -0.53$, $p = 0.003$. No associations were found between the WHOQOL-BREF and the SA-related brain regions extracted for the control group (Table 53, Appendix A). Hence, no regression analyses were further conducted.

QOLIBRI

For the group with TBI, significant correlation was evident between the self-related scale and the temporal cortex, $r(26) = -0.46$, $p = 0.007$, the temporal area, $r(26) = -0.55$, $p = 0.001$, and the hippocampus, $r(26) = -0.45$, $p = 0.009$. Also, reduced volume in the temporal area was associated with greater satisfaction with one's cognitive abilities, $r(26) = -0.52$, $p = 0.003$, and overall HRQOL, $r(26) = -0.47$, $p = 0.006$ (Table 54, Appendix A).

The temporal area appeared to have predictive value for the self-related scales, $F(1, 30) = 15.66$, adjusted $R^2 = 0.33$, $p = 0.0001$, where reduced volume to the temporal area lead to greater satisfaction with one's perception of oneself, $B = -266.09$, $t(30) = -3.96$, $p = 0.0001$.

Summary

To summarize, individuals with greater volume in the hippocampus were more likely to report greater dissatisfaction with the environmental aspect of their lives. Additionally, participants with TBI presenting with less volume in the temporal area, were more likely to report greater satisfaction with their sense of self.

Discussion

Experiment 2 sought to investigate the chronic and persistent course of brain volume loss in areas relating to EF, and thus SA, and how these areas are implicated in executive functioning, self-awareness, and as an effect quality of life.

Group comparisons

As expected, participants with TBI displayed significant differences in overall GM, WM, and CSF volume, as compared to a matched group of neurologically healthy individuals. Specifically, the group with TBI displayed less GM and WM volume, than the control group. Volume loss in GM and WM was coupled with greater CSF volume, as compared to the

controls. These findings are consistent with past literature clearly depicting the persisting effect of brain volume loss, for several years post-injury (Bendlin et al., 2008; Faul et al., 2010; Konstantinou et al., 2016), thus further supporting the argument that TBI is a long-term condition with chronic and possibly progressive effects rather than a static condition following a short recovery phase (Bigler, 2013; Chiaravalloti, & Goverover, 2016; Green et al., 2014; Masel, & Dewitt, 2010).

It was further sought to identify whether specific neural systems implicated in the concepts of EF and SA differed in volume between the two groups. Significant volumetric differences were found for both EF and Self –related brain areas, with the group with TBI presenting with reduced volume, as compared to the control group. Findings support past evidence by indicating that brain atrophy following a TBI may be concentrated in a fronto-temporal network, the cerebellum, the hippocampus, and areas relating to the thalamic network, such as the insula, the caudate, the cingulate cortex, and the putamen.

A primary contribution of the present study was to investigate the interrelation between EF and SA brain related areas differing between the two groups. Results were supportive of an interrelational network between these areas, clearly indicating that brain regions implicated in the EF and SA share common neurophysiology. Therefore, these findings may enhance past theories arguing that there is a close relationship between the executive system and SA, highlighting the fact that for SA to be intact the EF should also be unimpaired (Caldwell et al., 2014).

Individual Differences Analyses

Given the findings in Experiment 1, i.e. differences in the psychosocial measures between the two groups, it was sought to examine whether these differences were reflected on

the neural substrates involving EF and Self, for the areas where two group showed volumetric differences.

Results investigating for individual differences revealed that EF-related cortical and subcortical regions presented with significant predictive value for executive functioning, SA, and QOL. Whole sample correlational analysis revealed that greater volume in the caudate and temporal cortex also performed better on EF tasks. However, stepwise regression showed that participants with greater volume in the caudate (bilateral), only, were more likely to perform better in EF tasks. This finding is consistent with past evidence supporting that greater brain volume in EF-related regions, including cortical and subcortical areas associated with better neuropsychological performance (Konstantinou et al., 2016), and specifically executive functioning (Constantinidou et al., 2012c).

This study also investigated the predictive value of both the EF and SA –related areas could have on measures of SA. From the whole-group analysis, significant associations were evident between a number of areas such as the bilateral OFC, the right temporal pole, the bilateral insula, and bilateral the caudate and the DEX-R, indicating that greater volume in these areas related to greater SA. However, from these areas only the predictive value of the temporal pole survived on for all indexes extracted from the DEX-R, indicating that greater volume in the temporal cortex was more likely to predict greater overall SA and SA of one's social and self-regulation, motivation and attention, and executive cognition –related abilities. These findings were replicated within the group with TBI showing that reduced volume in the right temporal pole predicts greater SA deficits in executive cognition, motivation and attention, and overall SA. These findings clearly highlight the implication of EF-related brain areas in SA, further enhancing the association between these two functions, and also indicate

how damage to the underlying neurocircuit of the EF system may be involved into anosoagnosia.

Greater SA in the social and self-regulation domains correlated with numerous areas implicated in SA, i.e. the MPFC, the DLPFC, the OFC, the temporal cortex, the inferior frontal gyrus, and the corpus callosum. Stepwise regression revealed that greater volume only in the OFC was predictive of better social and self –regulation awareness, within the whole group. Additionally, greater awareness of motivational and attentional abilities was predicted by greater volume in the temporal cortex. Findings strengthen past literature finings investigating the implication of a specific network involved in SA (see Fitzgerald et al., 2012).

Further supporting the aforementioned arguments was evidence showing that reduced volume in EF-related areas, such as the putamen may be predictive of lower SA abilities, i.e., strategy awareness, and emergent/anticipatory awareness. Also, greater volume in SA-related structures, such as the right globus pallidus was predictive of reduced motivation to change post-injury disability, in individuals with TBI. The latter finding may be informative of the concept of SA as a whole, as individuals with greater volume and thus fewer SA deficits, are more likely to be aware of the effort necessitated in order to engage in altering dysfunctional behaviors (Fleminger, Oliver, Williams, & Evans, 2003; Zimmermann et al., 2017). Finally, it appears that reduced volume in the cingulate cortex holds significant predictive value for strategy awareness, and the temporal area and the primary visual cortex emergent/online – related SA deficits.

These findings extend literature indicating chronic brain atrophy for EF and SA – related structures, and their lingering persisting effects in individuals with TBI for several years following the injury, as these areas hold predictive value for individuals with impaired EF and awareness, i.e. one's ability to be aware of oneself (Fitzgerald et al., 2012).

Furthermore, EF and SA –related brain structures were predictive of TBI-related HRQOL subjective experience. Regression analysis revealed that participants with increased hippocampal volume individuals were more likely to report less satisfaction with the environmental aspects of their live. The group with TBI presenting with reduced volume in the primary sensory cortex were more likely to report greater satisfaction with their overall HRQOL. Additionally, individuals with TBI with greater atrophy in the temporal area, as this has been implicated in SA-related brain regions, reflected on their HRQOL by reporting higher levels of satisfaction regarding their sense of self. Such findings are novel, as no findings investigating the implication of brain atrophy on QOL/ HRQOL, following chronic TBI have yet to be reported in the literature, and shed light to the perplexed relationship between EF, SA and QOL.

CHAPTER 6

Discussion

The current research project implemented extensive neuropsychological measures and brain volumetry to examine the neurophysiologic underpinnings of persistent neuropsychological impairment, including SA, in chronic moderate-severe TBI. A second aim of this study was to investigate whether greater impairment in SA related to, or could predict greater levels of QOL. A final aim was to explore the relationship between brain volume loss and QOL, thus examining whether greater degree of brain atrophy is related to or is predictive of greater satisfaction with QOL.

Group comparisons

As expected, individuals with TBI displayed significant executive dysfunction and neuropsychological impairment, as they underperformed on all tasks of EF, verbal memory, and attention, with the exception of two tasks of visual memory, as compared to the non-injured controls. Such effects highlight the chronic and possibly progressive neuropsychological impairment in moderate-severe TBI for a median time since injury of 3.00 years (Chiaravalloti, & Goverover, 2016; Konstantinou et al., 2016; Rabinowitz, & Levin, 2014).

As a result of executive dysfunction, self–awareness was also assessed through self–reports and informant ratings. The group with TBI showed significant SA deficits in social and self –regulation and motivation and attention, compared to the control group. Despite that, not all individuals with moderate-severe TBI will experience SA deficits (Prigatano, & Altman, 1990), the results of this study provide additional information to the argument that problematic

SA may persist for years following a brain injury (Chiaravalloti, & Goverover, 2016; Hoofien et al., 2004; May et al., 2017; O'Keeffe et al., 2007).

To further explore such differentiation between the two groups in EF and SA skills, the underlying effects were investigated, i.e. volumetric differences in EF and SA –related cortical and sub-cortical regions. Initially, overall GM, WM and CSF were investigated showing that participants with TBI displayed significantly less volume in overall GM and WM, than the control group. As a result, the group with TBI exhibited increased CSF volume, compared to the control group, a hallmark for brain atrophy (Konstantinou et al., 2016).

The group with chronic TBI presented with significantly less volume in EF–related cortical and subcortical regions, compared to the control group, including the Dorsolateral Prefrontal Cortex (DPFC) and its connection to areas such as the orbitofrontal cortex (OFC), the temporal cortex and related areas (right temporal pole, and the insula, the caudate), the thalamus, the cingulate cortex, and the medial frontal cortex (MPFC). These areas differentiating between the two groups have been shown to implicate in EF and TBI–related brain volume loss (Constantinidou et al., 2012c; Spitz et al., 2013; Taylor et al., 2007).

Similar and additional cortical and subcortical areas regarding SA have been detected to differentiate in volume between the two groups. Specifically, the group with TBI showed reduced volume in the MPFC and related frontal areas as the OFC, and the inferior frontal gyrus, but also temporal regions including the temporal cortex and pole, the hippocampuss, as well as the basal ganglia, and individual regions such as the insula, the putamen, the parahippocampus, and the globus pallidus. These regions have been reported to accompany TBI brain atrophy and SA (see Fitzgerald et al., 2012; Newcombe et al., 2011; O'Keeffe et al. 2007).

Such evidence is further enhanced by findings from this study supporting the effect of TSI on vocabulary loss, known as a temporal lobe function. Specifically, even though individuals with TBI were matched to the non-injured controls on education, they still exhibited greater vocabulary deficits. This was further explained when individuals with longer TSI were compared on hold intelligence tasks to individuals who had sustained the injury more recently (median TSI = 3 years), with the former exhibiting greater impairment on their vocabulary skills. Thus, it may be concluded that crystallized intelligence is negatively affected by TSI, and may be explained by greater atrophy within the temporal lobes.

Due to the fact that the group with TBI showed significantly reduced volume in similar areas, as these were extracted from two different brain maps, one specific to EF and the other to SA, the associations between these brain areas were investigated. Findings clearly show that EF–related brain regions and regions implicated in SA significantly relate, thus further informing literature and the associated biopsychosocial models on the interrelation between these two concepts (Caldwell et al., 2014; Fitzgerald et al., 2017).

To summarize, these volumetric differences indicate that brain atrophy following chronic TBI may be concentrated in a fronto–temporal network, the cerebellum, and the hippocampus, and areas relating to the thalamic network, such as the insula, the caudate, the cingulate cortex, and the putamen (Constantinidou et al., 2012c, Fitzgerald et al., 2012; Konstantinou et al., 2016). Furthermore, EF and SA seem to share common morphological network, with areas extracted from the EF brain maps, such as the DLPC significantly correlating with SA-related areas including the MPFC. Combining these findings, it may be argued that given the damage to areas relating to the EF system, such as the DLPFC, the cingulate, the OFC, the putamen, the temporal regions, including the thalamus, the insula, the caudate, the globus pallidus and the MPFC, and their implication in the SA system, allow for

strong inferences to be made regarding the existence of an interrelational network (Caldwell et al., 2014; Fitzgerald et al., 2017).

As an extent of these chronic and persistent deficits, it was further sought to investigate satisfaction with one's QOL. According to literature low SA may result into a similar response pattern between individuals with TBI and healthy controls regarding their QOL, aside from physical health, as the main outcome of physical trauma and related difficulties often accompanying TBI (Sasse et al., 2013; Sherer et al., 1998). This argument has been somewhat supported by this study, with individuals with TBI focusing on physical difficulties, thus stating greater dissatisfaction with their physical health, compared to the non–injured group. Also, no differences have been detected between the two groups on their levels of satisfaction regarding their social interactions, and psychological state, nor their overall QOL. The lack of differentiation on these dimensions may be assigned to the lower levels of SA detected in individuals with TBI, who are most likely to ignore cognitive and social challenges, and how these may hinder QOL (Chiaravalloti, & Goverover, 2016; Mathias, & Wheaton, 2007; Sasse et al., 2013). Furthermore the control group reported less satisfaction regarding the environment dimension of the WHOQOL–BREF centering on financial resources, physical safety, home environment, and related aspects. It would be expected that individuals with TBI would not focus on such aspects, as following the injury these aspects are attended to mainly by their significant others (Abrahamson, Jensen, Springett, & Sakel, 2017).

Coping mechanisms and mood disorders were also examined, as it has been argued that these factors appear to affect neuropsychological performance, SA, and QOL (Brands et al., 2014a; Brands et al., 2014b). The two groups did not differ on their coping mechanisms; however, the group with TBI expressed greater levels of melancholic diathesis. Despite the strong family support and social networks which are inherent in the tightly-knit Cypriot

society, patients with chronic moderate-severe TBI experience significant depression symptoms similar to those reported in larger, industrialized nations (Whelan-Goodinson et al., 2009).

Through the combination of extensive neuropsychological evaluation and MRI measures, current findings clearly depict the persistent and long–term effects of TBI on brain volume loss, neuropsychological impairment, including EF, and thus SA (Bendlin et al., 2008; Faul et al., 2010; Konstantinou et al., 2016), further reinforcing the notion that TBI is a chronic condition with long–term and possibly progressive effects rather than a static condition following a short recovery phase (Bigler, 2013; Chiaravalloti & Goverover, 2016; Green et al., 2014; Masel, & Dewitt, 2010), and may also pose as a precursor to pathological aging (Fegyveres et al., 2007; Fisoni, 2010; Lye, & Shores, 2000).

Individual Differences

Group differences were used as the basis of exploring the relationship between EF, SA, and QOL. However, due to the fact that literature proposes an interaction between coping mechanisms and diathesis (Anson & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011), the relationship between EF, SA, and QOL, to the former were initially examined. Findings show that, participants performing better on EF tasks and presenting with greater SA reported greater implementation of avoidant coping techniques. Furthermore, the healthy controls who employ more avoidant and problem-task -related techniques also reported greater dissatisfaction with psychological health and social relationships, respectively. On the other hand, it appears that the group with TBI engages in greater emotional coping which leads to greater satisfaction with their social relationships. Despite the fact that the two groups do not differentiate on the measure of coping, it appears that they present different correlational

patterns. The use of ineffective techniques reported by healthy controls and their subsequent associations to EF, SA, and QOL, may be attributed to gender, as it has been posed that males tend to use less emotion-focused; whereas individuals with TBI are more likely to seek the use of emotional support, mainly through their family members that is important in managing one's condition (Anson & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011). As an effect, these results do support and further complicate the involvement of coping techniques in the relationship of EF, SA, and QOL (Anson & Ponsford, 2006; Brands et al., 2014b; Wolters et al., 2011).

Diathesis was also investigated to detect any associations with the key concepts under investigation, i.e. EF, SA, and QOL (Chiaravalloti, & Goverover, 2016). For EF, non-injured individuals over performing in a cognitive flexibility task reported less asthenia and anxiety - related symptomatology. Despite that this association involves just one EF task, the association between cognitive and EF processes with psychiatric symptomatology has been well established for healthy populations, and is currently investigated in TBI the association (Chiaravalloti, & Goverover, 2016). In addition, greater SA regarding executive cognition and overall SA was associated with greater anxiety and asthenia –reported difficulties. Furthermore, greater dissatisfaction with one's QOL was associated with greater depressive and melancholic symptomatology; whereas more manic-like behavior related with greater life satisfaction. These findings forewarn about the moderating effects of diathesis on the associations between the variables of interest (Chiaravalloti, & Goverover, 2016).

As a result of the aforementioned findings, coping skills and mood and anxiety – related symptoms were partialled-out of the associations of interest. The associations detected between the EF and SA –related regions lead to the investigation of potential relationships amongst EF and SA measures and brain morphology. Findings clearly depict that greater

performance in EF relates to greater SA on fluency, flexibility and working memory, social and self –regulation, and motivation and attention, as well as overall SA. Further associations presented within the group with TBI, alone, enforce the argument that greater strategic and online/emergent SA impairment relates to greater executive dysfunction. Such evidence depicts a close relational framework between the two variables, and is also supportive of the argument that for SA to be intact, the EF system should be undamaged (Caldwell et al., 2014; Zimmermann et al., 2017).

Given the associations between EF and SA and the volumetric differences in the related cortical and subcortical regions, brain volume was examined as a predictor of these variables. Current findings provide isnsight on the relationship between EF and SA and related brain regions, which were extracted through meta-analytic maps pertaining to these concepts. Initially, whole sample stepwise regression showed that participants with greater volume in the caudate (bilateral) were more likely to perform better in EF tasks, and that those with greater volume in the right temporal pole presented with greater overall SA and SA in one's social and self-regulation, motivation and attention, and executive cognition –related abilities. When similar analyses were focused on the group with TBI, results showed that reduced volume in both the putamen and areas of the DLPFC may be predictive of lower strategy awareness. Additionally, greater deficits in emergent/online SA were predicted by reduced volume in the caudate and the parahippocampal region.

It is clear from these results that greater brain atrophy within areas implicated in EF that have also been described to comprise the neurocircuits underlying the EF system (Constantinidou et al., 2012c), hold significant predictive value of worst neuropsychological performance (Konstantinou et al., 2016), and specifically executive functioning (Spitz et al., 2013), and impaired SA (Fitzgerald, 2017).

To further examine this argument SA-related brain structures and their predictive value on SA was examined. Numerous areas were found correlating to SA, including the MPFC, and its connection to the DLPC, the OFC, the inferior frontal gyrus, and the temporal cortex. However, using stepwise regression findings were somewhat different with the individuals with greater volume in the OFC presenting with better social and self –regulation awareness. Results pertaining specifically to the group with TBI, further support previous findings from this study indicating that reduced volume in the cingulate cortex leads to greater strategy – related SA deficits. Also, less volume in the temporal area and the primary visual cortex appear predictive of greater impairment in emergent/online SA. Finally, greater volume in the right globus pallidus in participants with TBI was predictive of reduced motivation to change regarding an individual's post-injury disability. These findings may be informative of the concept of SA as a whole, with the latter finding further adding to the argument that when an individual presents with greater volume and thus fewer SA deficits, he/she is more likely to be aware of the effort necessitated in order to engage in altering dysfunctional behaviors (Chiaravalloti, & Goverover, 2016; Doig et al., 2001). Findings also strengthen past literature investigating the implication of a specific network involved in SA, between the MPFC and its connection to other structures, such as the fronto-temporo-thalamic regions (see Fitzgerald et al., 2012), and how this network may be part of the neuroanatomy of the EF system. Therefore, it is important for health care professional to perhaps focus on EF and SA during the acute phase, and simultaneously tackle potential lack of motivation that may impede with the therapeutic process.

The aforementioned findings extend the existing literature because they provide evidence on the neuro-anatomical underpinnings of EF and SA deficits several years post TBI and the predictive role of volume integrity in neuropsychological performance. Additionally,

findings demonstrate a direct association between impaired SA and executive dysfunction (Caldwell et al., 2014; Hart et al., 2005; Zimmermann et al., 2017), and that impaired SA may persist for years following an injury (Hoofien et al., 2004).

Novel and encouraging findings emerge from this study providing important information on the relationship between EF brain regions and QOL, which have yet to be explored in existing literature. The initial finding supported that, individuals who score higher on EF tasks report greater satisfaction with their physical health. As this was a single finding it was proposed that it be interpreted with caution. However, additional findings linking EF-related brain structures and HRQOL, specifically in TBI, allow for stronger inferences to be made. Specifically, reduced volume in the left primary sensory cortex correlated with reporting of greater physical and overall HRQOL. Additionally, reduced insular volume significantly correlated with greater satisfaction with one's perception of self and overall HRQOL. When stepwise regression was performed greater satisfaction with one's overall HRQOL was explained by reduced volume the left primary sensory cortex. The predictive value of the primary sensory cortex may be explained by the implication of brain regions relating to EF, such as the DLPFC and its connection to sensory and motor cortices, and how numerous areas and skills are required for one to present intact EF. These findings clearly support the hypothesis of a negative association between EF and QOL in individuals with TBI, which can be explained by the positive association between EF and SA, and the negative relationship between SA and QOL, detected in this study in individuals with TBI.

Overall, findings linking brain atrophy, EF, SA and QOL/ HRQOL, following chronic TBI have been reported in this study. Specifically, associations between EF and SA support a close interrelational framework with shared morphological sequelae in TBI. This network is further enhanced when associations extend between these two concepts and QOL. It appears

that individuals with TBI are more likely to present with anosoagnosia, i.e. be less aware of their deficits, and as a result they tend to perceive themselves as more capable and thus report greater subjective satisfaction with their QOL. Finally, it is noteworthy and firstly reported that areas implicated in EF and SA hold predictive value for HRQOL, in individuals with TBI. Though this is a small finding, these results do highlight the importance of cognitive rehabilitation following a TBI, and encourage replication analyses in larger samples to further detect such associations.

Implications

Findings of this study depict significant brain atrophy and its related effects lingering for several years following the injury. As a result of limited post-acute rehabilitation services offered in Cyprus this study allowed capturing the true post-injury effects, which highlight the significance for systematic post-acute comprehensive rehabilitation and community re-integration. Furthermore, the interrelation of EF and SA –related brain regions along with findings regarding executive functioning and metacognition with the subjective experience of QOL may lead to the development of related biopsychosocial models or enhancement of existing models discussing these links. Therefore, such evidence linking impaired SA to EF and to QOL in individuals with TBI not having received systematic post-acute rehabilitation may guide health-care professionals in designing more comprehensive acute and post-acute rehabilitation programs by focusing on the impact of impairments in EF and SA on daily participation and QOL. At the same time, it is important that counseling services are offered to counteract depressive symptoms and also teach effective coping techniques. Combined together, such methods will further improve patients' recovery. In addition, these results may inform on the significance of more appropriate and well-equipped rehabilitation centers specific to neurological conditions, including TBI.

Limitations & Future Research

The results yielded from this study clearly support the chronic and persistent effects of moderate-severe TBI, including brain atrophy, executive dysfunctioning, SAs deficits and their association to QOL. However, further investigation is required on how these constructs act as moderators to recovery using larger samples. Also, corrections for multiple comparisons were used by lowering the α level to 0.01. Even though such adjustments were conducted some of the significance within these findings may be questionable. Furthermore, this study attempted to preserve a homogeneous sample by recruiting male participants, alone. Hence, future studies should replicate these analyses using female participants with TBI in order to investigate potential variance in findings due to gender differences (e.g. hormones, see Berry et al., 2009). In addition, the effects reported within this study may not be replicable for individuals with less severe injuries such as mild TBI, or of greater chronic course, i.e. greater than 6 years, or during the acute phase. Therefore, further exploration of these effects should be investigated on a severity and TSI continuum. Also, it is important to highlight the need for longitudinal studies and repeated assessments in populations with chronic conditions such as TBI, in order to keep track of potential neuropsychological and psychosocial changes, as well as further brain atrophy. Finally, factors such as the cognitive reserve need to be explored as potential mediators to overall brain injury outcome, as well as protective factors of fewer neuropsychological deficits and thus better SA.

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APPENDIXES

APPENDIX A

Table 1. *Reliabilities, Means, Standard Deviations for the DEX-R*

	Participants' Ratings			Signficant Others' Ratings		
Factors	*a (90% CI)*	*M*	*SD*	*a (90% CI)*	*M*	*SD*
Social and Self-Regulation	0.94	22.02	14.51	0.93	28.97	14.27
Flexibility, Fluency and Working Memory	0.91	10.49	7.64	0.90	12.35	7.94
Motivation and Attention	0.92	6.90	6.78	0.91	8.90	6.69

Table 2. *Reliability, Means, Standard Deviations for the QOLIBRI*

	Group with TBI		
Factors	*a (90% CI)*	*M*	*SD*
Cognition	0.90	71.21	18.45
Self	0.86	73.70	16.09
Daily Life & Autonomy	0.83	68.40	20.17
Social Relationships	0.68	70.83	17.24
Emotional Difficulties	0.83	62.73	23.29
Physical Problems	0.77	57.42	25.68
Total	0.65	-10.02	2.12

Table 3. *Reliability, Means, Standard Deviations for the MPAI-4*

	Group with TBI		
Factors	*a (90% CI)*	*M*	*SD*
Ability	0.77	11.7	7.06
Adjustment	0.87	14.64	8.16
Participation	0.85	8.00	6.06
Total	0.71	31.61	15.85

Table 4. *Reliability, Means, Standard Deviations for the Brief Cope*

Factors	*a (90% CI)*	*M*	*SD*
Problem-Task Coping	0.72	11.70	7.06
Emotion Focused Coping	0.63	14.64	8.16
Avoidance Coping	0.71	8.00	6.06

Table 5. *Demographic Information of the group with TBI*

Participant No.	Age (Years)	Education (Years)	TSI (Years)	Cause of Injury	GOSe	LoHS (days)	PTA (days)
1	26	13	8	Motor Vehicle Injury	6	61	61
2	26	14	5	Motor Cycle Injury	6	10	181
3	45	15	17	Assault	5	61	45
4	40	14	14	Assault	7	30	61
5	32	18	18	Motor Cycle Injury	8	30	17
6	34	12	2	Motor Vehicle Injury	6	30	30
7	25	13	3	Motor Cycle Injury	6	60	25
8	20	12	1	Motor Vehicle Injury	6	56	7
9	23	12	2	Motor	7	30	30

				Vehicle Injury			
10	43	19	19	Pedestrian - Vehicle Collision	7	42	42
11	44	12	1	Fall or Work-related Injury	6	61	61
12	22	15	4	Pedestrian - Vehicle Collision	7	30	7
13	36	12	17	Motor Vehicle Injury	3	180	17
14	44	8	6	Fall or Work-related Injury	5	45	21
15	23	12	3	Motor Cycle Injury	3	60	20

16	30	9	4	Motor Cycle Injury	6	122	75
17	21	12	2	Motor Cycle Injury	6	30	27
18	18	11	4	Motor Cycle Injury	3	95	244
19	29	16	1	Motor Cycle Injury	6	61	60
20	29	11	4	Motor Cycle Injury	3	61	331
21	24	12	2	Motor Cycle Injury	4	88	61
22	38	12	1	Object Falling	4	53	25

23	37	12	2	Pedestrian - Vehicle Collision	6	18	2
24	20	13	2	Motor Vehicle Injury	4	49	45
25	31	8	1	Motor Vehicle Injury	3	122	61
26	34	12	1	Motor Vehicle Injury	5	61	61
27	29	8	1	Motor Cycle Injury	4	183	28
28	33	16	1	Motor Vehicle Injury	3	61	61

29	28	16	1	Motor Vehicle Injury	6	30	61
30	38	16	11	Motor Cycle Injury	7	30	30
31	31	8	4	Motor Cycle Injury	6	30	30
32	51	6	9	Pedestrian - Vehicle Collision	6	30	183
33	21	11	2	Motor Vehicle Injury	6	61	30

TSI, time since injury; GOSe, Galveston Outcome Scale extended; LoHS, Length of Hospitalization Stay; PTA, Posttraumatic Amnesia.

Table 6. *Means and Standard Deviations for the AVLT, Logical Memory, TMT & Rey Figure*

	M		SD	
	TBI	Control	TBI	Control
Learning Trials				
AVLT List 1	4.76	6.63	1.97	1.84
AVLT List 2	6.67	9.92	2.94	2.08
AVLT List 3	7.61	11.63	2.77	2.16
AVLT List 4	8.55	12.29	3.10	2.29
AVLT List 5	9.15	12.63	3.06	2.39
Recall Trials				
AVLT List 5	9.15	12.57	3.06	2.43
Short delay free recall	6.55	10.57	4.06	3.09
Long delay free recall	5.97	10.74	4.24	3.09
Logical Memory				
Immediate recall, story A	11.36	15.96	4.21	2.77
Immediate recall, story B	10.09	15.33	3.97	3.14
Delayed recall, story A	8.45	14.54	4.94	2.73
Delayed recall, story B	8.33	14.17	4.66	3.52
TMT				
TMT A (reversed)	-54.18	-29.29	37.23	10.87
TMT B (reversed)	-120.12	-72.75	62.77	27.13
Rey Figure Recall				
Immediate	14.76	19.79	6.23	5.02
Delayed	13.58	19.06	6.32	5.04

M, mean; SD, standard deviation

Table 7. *Statistics for all Other Neuropsychological Tests*

Measure	Group with TBI	Control Group	Statistics			
	M (SD)		*t*	df	*p*	Cohen's *d*
Rey Figure Copy	26.39 (6.28)	31.29 (1.65)	-4.28	37.91	.000	1.07
Digit span Forward	6.21 (2.26)	7.83 (1.88)	-2.86	55	.006	0.78
Digit span Backward	5.00 (2.24)	7.04 (2.07)	-3.51	55	.001	0.95
Spatial span Forward	7.88 (1.83)	8.74 (1.79)	-1.74	54	.087	0.47
Spatial span Backward	7.24 (1.64)	8.04 (2.18)	-1.57	54	.123	0.41
Symbol Digits Modalities	33.00 (13.36)	51.25 (7.25)	-6.62	51.45	.000	1.70
COWAT - Animals	13.58 (4.87)	17.50 (4.03)	-3.22	55	.002	0.88
COWAT – F Words	8.70 (3.27)	12.13 (4.25)	-3.44	55	.001	0.90
PPVT	20.94 (6.86)	27.13 (2.66)	-4.72	43.98	.000	1.19
Pseudo Correct in 45 secs	23.94 (13.35)	38.63 (11.30)	-4.37	55	.000	1.19

M, mean; SD, standard deviation; df, degrees of freedom; $p < 0.05$

Table 8. *Group comparisons of the DEX-R Discrepancy Indexes*

	Group with TBI	Control Group	Statistics		
Indexes	M (SD)		*F*	df	*p*
Fluency, Flexibility, Working Memory	-34.04 (9.94)	0.29 (2.24)	3.22	55	0.078
Social & Self -Regulation	-11.79 (14.63)	-0.29 (3.99)	13.99	55	0.0001
Motivation & Attention	-3.94 (6.46)	0.67 (1.43)	11.72	55	0.001

Table 9. *Group comparisons of the COPE Brief*

	Group with TBI	Control Group	Statistics		
Indexes	M (SD)		*F*	df	*p*
Avoidance	9.30 (3.04)	8.13 (2.44)	2.46	55	0.123
Problem-Task	29.06 (4.24)	30.12 (3.34)	1.04	55	0.312
Emotion- Focused	12.55 (2.59)	11.36 (2.04)	3.38	54.12	0.071

M, mean; SD, standard deviation; df, degrees of freedom; $p < 0.05$

Table 10. *Group comparisons of the SRSDA*

	Group with TBI	Control Group	Statistics		
Indexes	M (SD)		*F*	df	*p*
Long Depression	7.00 (7.52)	4.30 (5.11)	2.22	53	0.143
Short Depression	4.75 (5.36)	2.43 (3.13)	3.43	53	0.070
Melancholia	4.66 (4.85)	2.22 (3.30)	4.35	53	0.042
Asthenia	3.50 (4.57)	3.43 (5.12)	0.00	53	0.961
Anxiety	3.50 (4.83)	3.78 (5.08)	0.04	53	0.835
Mania	-0.31 (1.64)	-0.17 (1.49)	0.11	53	0.738

M, mean; SD, standard deviation; df, degrees of freedom; $p < 0.05$

Table 11. *Correlations between Executive Functions Tasks and the COPE-Brief*

Measure	1	2	3	4	5	6	7	8
1. Avoidance Coping	-							
2. Problem-Task Coping	-0.05	-						
3. Emotion-Focused Coping	0.38*	0.25	-					
4. Rey Figure Copy	-0.28	0.04	-0.24	-				
5. TMT A (Valenced)	-0.21	-0.04	-0.34*	0.69*	-			
6. TMT B (Valenced)	-0.20	0.01	-0.26	0.66*	0.68*	-		
7. SDMT	-0.27	0.02	-0.47*	0.66*	0.68*	0.65*	-	
8. COWAT Animal Naming	-0.11	0.00	-0.27	0.49*	0.38*	0.34*	0.54*	-
9. COWAT Letter F	-0.23	0.07	-0.28	0.37*	0.37*	0.36*	0.44*	0.33

p<0.01; Whole-group N = 57.

Table 12. *Correlations between Executive Functions Tasks and the COPE-Brief*

Measure	1	2	3	4	5	6	7	8
1. Avoidance Coping	-							
2. Problem-Task Coping	0.06	-						
3. Emotion-Focused Coping	0.48*	0.23	-					
4. Rey Figure Copy	-0.27	-0.06	-0.21	-				
5. TMT A (Valenced)	-0.17	-0.08	-0.28	0.66*	-			
6. TMT B (Valenced)	-0.20	-0.04	-0.15	0.63*	0.65*	-		
7. SDMT	-0.28	-0.15	-0.43*	0.59*	0.67*	0.57*	-	
8. COWAT Animal Naming	-0.27	-0.01	-0.25	0.48*	0.32	0.27	0.51*	-
9. COWAT Letter F	-0.16	-0.05	-0.16	0.38	0.38	0.30	0.40	.45*

p<0.01; Group with TBI, N = 33.

Table 13. *Correlations between Executive Functions Tasks and the COPE-Brief*

Measure	1	2	3	4	5	6	7	8
1. Avoidance Coping	-							
2. Problem-Task Coping	-0.20	-						
3. Emotion-Focused Coping	0.03	0.42	-					
4. Rey Figure Copy	-0.04	0.27	0.11	-				
5. TMT A (Valenced)	-0.05	-0.33	-0.30	-0.12	-			
6. TMT B (Valenced)	0.16	-0.08	-0.30	0.09	0.19	-		
7. SDMT	0.14	0.14	-0.42	0.27	0.13	0.40	-	
8. COWAT Animal Naming	0.45	-0.17	-0.08	-0.06	0.02	0.01	0.12	-
9. COWAT Letter F	-0.19	0.10	-0.28	-0.20	-0.02	0.09	0.00	-0.13

p<0.01; Control group N = 24.

Table 14. *Correlations between DEX-R and the COPE-Brief*

Measure	1	2	3	4	5	6
1. Avoidance Coping	-					
2. Problem-Task Coping	-0.05	-				
3. Emotion-Focused Coping	0.38*	0.25	-			
4. Fluency, Flexibility, Working Memory	-0.07	-0.03	-0.15	-		
5. Social & Self - Regulation	0.04	0.14	-0.15	0.79*	-	
6. Motivation & Attention	0.01	0.03	-0.15	0.74*	0.83*	-
7. Overall SA	0.00	0.07	-0.16	0.90*	0.97*	0.90*

p<0.01; Whole-group N = 57.

Table 15. *Correlations between DEX-R and the COPE-Brief*

Measure	1	2	3	4	5	6
1. Avoidance Coping	-					
2. Problem-Task Coping	0.06	-				
3. Emotion-Focused Coping	0.48*	0.23	-			
4. Fluency, Flexibility, Working Memory	-0.11	-0.02	-0.06	-		
5. Social & Self - Regulation	0.16	0.12	0.03	0.81*	-	
6. Motivation & Attention	0.07	0.05	0.01	0.72*	0.80*	-
7. Overall SA	0.06	0.07	0.00	0.92*	0.96*	0.88*

p<0.01; Group with TBI, N = 33.

Table 16. *Correlations between DEX-R and the COPE-Brief*

Measure	1	2	3	4	5	6
1. Avoidance Coping	-					
2. Problem-Task Coping	-0.20	-				
3. Emotion-Focused Coping	0.03	0.42	-			
4. Fluency, Flexibility, Working Memory	0.59*	-0.48*	-0.45*	-		
5. Social & Self - Regulation	0.22	-0.11	-0.56*	0.37*	-	
6. Motivation & Attention	0.47	-0.71*	-0.68*	0.72*	0.48*	-
7. Overall SA	0.46	-0.40	-0.67*	0.75*	0.87*	0.79*

p<0.01; Control group N = 24.

Table 17. *Correlations between SRSI and the COPE-Brief*

Measure	1	2	3	4	5
1. Avoidance Coping	-				
2. Problem-Task Coping	0.06	-			
3. Emotion-Focused Coping	0.48*	0.23	-		
4. Strategy Awareness	0.28	-0.23	0.08	-	
5. Emergent/ online Awareness	0.06	-0.18	0.00	0.93*	-
6. Readiness to Change	0.04	-0.03	0.03	-0.11	-0.10

p<0.01; Group with TBI, N = 33.

Table 18. *Correlations between WHOQOL-BREF and the COPE-Brief*

Measure	1	2	3	4	5	6	7
1. Avoidance Coping	-						
2. Problem-Task Coping	-0.05	-					
3. Emotion-Focused Coping	0.38*	0.25	-				
4. Physical Health	-0.10	0.30	0.16	-			
5. Psychological Health	-0.26	0.03	0.18	0.53*	-		
6. Social Relationships	0.03	-0.12	0.16	0.29	0.64*	-	
7. Environment	0.02	0.13	0.31	0.41*	0.66*	0.35*	-
8.Total	-0.10	0.14	0.27	0.75*	0.89*	0.64*	0.82*

p<0.01; Whole-group N = 57.

Table 19. *Correlations between WHOQOL-BREF and the COPE-Brief*

Measure	1	2	3	4	5	6	7
1. Avoidance Coping	-						
2. Problem-Task Coping	0.06	-					
3. Emotion-Focused Coping	0.48*	0.23	-				
4. Physical Health	0.00	0.24	0.20	-			
5. Psychological Health	-0.12	0.14	0.23	0.61*	-		
6. Social Relationships	0.04	0.07	0.40	0.31	0.70*	-	
7. Environment	0.10	0.32	0.27	0.55*	0.64*	0.46*	-
8.Total	0.00	0.25	0.32	0.82*	0.90*	0.70*	0.82*

p<0.01; Group with TBI, N = 33.

Table 20. *Correlations between WHOQOL-BREF and the COPE-Brief*

Measure	1	2	3	4	5	6	7
1. Avoidance Coping	-						
2. Problem-Task Coping	-0.20	-					
3. Emotion-Focused Coping	0.03	0.42	-				
4. Physical Health	-0.16	0.41	0.41	-			
5. Psychological Health	-0.54*	-0.19	0.10	0.45	-		
6. Social Relationships	0.14	-0.69*	-0.25	0.06	0.53*	-	
7. Environment	-0.24	0.00	0.28	0.66*	0.76*	0.40*	-
8.Total	-0.30	-0.08	0.22	0.71*	0.88*	0.55*	0.95*

p<0.01; Control group, N = 33.

Table 21. *Correlations between QOLIBRI and the COPE-Brief*

Measure	1	2	3	4	5	6	7	8	9
1. Avoidance Coping	-								
2. Problem-Task Coping	0.06	-							
3. Emotion-Focused Coping	0.48*	0.23	-						
4. Cognition Scale	-0.20	0.09	0.11	-					
5. Self Scale	-0.06	0.07	0.18	0.56*	-				
6. Daily Life & Autonomy Scale	-0.07	0.06	0.26	0.51*	0.60*	-			
7. Social Relationships Scale	0.21	0.06	0.46*	0.42	0.51*	0.42*	-		
8. Emotions Scale	-0.11	0.09	-0.19	0.14	0.17	0.19	-0.01	-	
9. Physical Problems Scale	-0.11	-0.16	-0.04	0.21	0.24	0.39	0.08	0.33	-
10. Total Scale	-0.10	0.04	0.16	0.69*	0.73*	0.78*	0.55*	0.52*	0.64*

p<0.01; Group with TBI, N = 33.

Table 22. *Correlations between Executive Functions and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Rey Figure Copy	-										
2. TMT A (Valenced)	.688**	-									
3. TMT B (Valenced)	.662**	.676**	-								
4. SDMT	.66*	.68*	.65*	-							
5. COWAT Animal Naming	.49*	.38*	0.34	.54*	-						
6. COWAT Letter F	.37*	.37*	.36**	.44*	0.33	-					
7. Long Depression	-0.02	0.14	0.03	0.03	0.06	-0.01	-				
8. Short Depression	-0.11	0.05	-0.07	-0.03	0.05	-0.09	.97*	-			
9. Melancholia	-0.05	0.1	-0.04	-0.05	0.02	-0.03	.96*	.92*	-		
10. Asthenia	0.06	0.19	0.03	0.07	0.05	0.22	.72*	.60*	.73*	-	
11. Anxiety	0.07	0.14	0.06	0.07	0.13	0.28	.74*	.62*	.72*	.94*	-
12. Mania	0.23	0.12	0.12	0.04	-0.02	0.17	-0.34	-.41*	-0.32	0.03	-0.02

p<0.01; Whole-group N = 57.

Table 23. *Correlations between Executive Functions and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9	10	11	
1. Rey Figure Copy	-											
2. TMT A (Valenced)	0.66*	-										
3. TMT B (Valenced)	0.63*	0.65*	-									
4. SDMT	0.57*	0.67*	0.57*	-								
5. COWAT Animal Naming	0.49*	0.32*	0.27	0.51*	-							
6. COWAT Letter F	0.38*	0.38	0.30*	0.40*	0.45*	-						
7. Long Depression	0.11	0.30	0.23	0.36	0.20	0.25	-					
8. Short Depression	0.01	0.20	0.10	0.28	0.19	0.18	0.97*	-				
9. Melancholia	0.11	0.30	0.20	0.35	0.16	0.24	0.97*	0.92*	-			
10. Asthenia	0.18	0.31	0.25	0.37	0.16	0.36	0.75*	0.66*	0.77*	-		
11. Anxiety	0.17	0.21	0.25	0.28	0.24	0.34	0.83*	0.75*	0.81*	0.93*	-	-0.12
12. Mania	0.30	0.10	0.18	0.04	0.07	0.15	-0.37	-0.44	-0.39	-0.09		

* * *

p<0.01; Group with TBI, N =33.

Table 24. *Correlations between Executive Functions and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Rey Figure Copy	-										
2. TMT A (Valenced)	-0.12	-									
3. TMT B (Valenced)	0.09	0.19	-								
4. SDMT	0.27	0.13	0.40*	-							
5. COWAT Animal Naming	-0.06	0.02	0.01	0.12	-						
6. COWAT Letter F	-0.20	-0.02	0.09	0.00	-0.13	-					
7. Long Depression	-0.10	-0.08	-0.37	-0.39	0.05	-0.18	-				
8. Short Depression	-0.06	-0.09	-0.27	-0.31	0.11	-0.28	0.96*	-			
9. Melancholia	-0.11	-0.09	-0.47	-0.49*	0.11	-0.13	0.94*	0.91*	-		
10. Asthenia	-0.32	-0.04	-0.56*	-0.49*	-0.08	0.14	0.77*	0.61*	0.80*	-	
11. Anxiety	-0.39	-0.04	-0.53	-0.44	-0.04	0.27	0.68*	0.49*	0.68*	0.95*	-

12. Mania	-0.08	0.20	-0.14	-0.09	-0.30	0.20	-0.25	-0.36*	-0.16	0.23	0.15

p<0.01; Control group N = 24.

Table 25. *Correlations between DEX-R and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9
1. Long Depression	-								
2. Short Depression	0.97*	-							
3. Melancholia	0.96*	0.92*	-						
4. Asthenia	0.72*	0.60*	0.73*	-					
5. Anxiety	0.74*	0.62*	0.72*	0.94*	-				
6. Mania	-0.34*	-0.41*	-0.32	0.03	-0.02	-			
7. Fluency, Flexibility, Working Memory	0.27	0.20	0.19	0.34*	0.42*	0.17	-		
8. Social & Self - Regulation	0.14	0.10	0.00	0.13	0.25	0.12	0.80*	-	
9. Motivation & Attention	0.20	0.15	0.11	0.20	0.28	0.09	0.74*	0.83*	-
10. Overall SA	0.20	0.15	0.09	0.22	0.33*	0.14	0.90*	0.97*	0.90*

p<0.01; Whole-group N = 57.

Table 26. *Correlations between DEX-R and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9
1. Long Depression	-								
2. Short Depression	0.97*	-							
3. Melancholia	0.97*	0.92*	-						
4. Asthenia	0.75*	0.66*	0.77*	-					
5. Anxiety	0.83*	0.75*	0.81*	0.93*	-				
6. Mania	-0.37	-0.44	-0.39	-0.09	-0.12	-			
7. Fluency, Flexibility, Working Memory	0.40	0.32	0.33	0.52*	0.59*	0.24	-		
8. Social & Self - Regulation	0.25	0.22	0.14	0.20	0.35	0.21	0.81*	-	
9. Motivation & Attention	0.39	0.32	0.31	0.36	0.44*	0.11	0.72*	0.80*	-
10. Overall SA	0.35	0.29	0.25	0.36	0.48*	0.22	0.92*	0.96*	0.88*

p<0.01; Group with TBI, N =33.

Table 27. *Correlations between DEX-R and the SRSDA*

Measure	1	2	3	4	5	6	7	8	9
1. Long Depression	-								
2. Short Depression	0.96*	-							
3. Melancholia	0.94*	0.91*	-						
4. Asthenia	0.77*	0.61*	0.80*	-					
5. Anxiety	0.68*	0.49	0.68*	0.95*	-				
6. Mania	-0.25	-0.36	-0.16	0.23	0.15	-			
7. Fluency, Flexibility, Working Memory	-0.13	-0.12	-0.06	-0.16	-0.01	-0.29	-		
8. Social & Self - Regulation	0.41	0.46	0.23	0.06	0.07	-0.49	0.37	-	
9. Motivation & Attention	-0.12	-0.06	-0.09	-0.22	-0.16	-0.13	0.72*	0.48	-
10. Overall SA	0.19	0.23	0.11	-0.07	0.00	-0.45	0.75*	0.87*	0.79*

p<0.01; Control group N = 24.

Table 28. *Correlations between SRSI and the SRSDA*

Measure	1	2	3	4	5	6	7	8
1. Long Depression	-							
2. Short Depression	0.97*	-						
3. Melancholia	0.97*	0.92*	-					
4. Asthenia	0.75*	0.66*	0.77*	-				
5. Anxiety	0.83*	0.75*	0.81*	0.93*	-			
6. Mania	-0.37	-0.44	-0.39	-0.09	-0.12	-		
7. Strategy Awareness	-0.20	-0.15	-0.22	-0.28	-0.37	-0.24	-	
8. Emergent/Online Awareness	-0.32	-0.28	-0.33	-0.26	-0.38	-0.16	0.93*	-
9. Readiness to Change	-0.04	-0.05	0.00	-0.03	0.02	0.06	-0.11	-0.10

p<0.01; Group with TBI, N =33.

Table 29. *Partial Correlations between EF and MPAI-4*

Measure	1	2	3	4	5	6	7	8	9
					Bivariate				
1. MPAI-4 Ability Index	-								
2. MPAI-4 Adjustment Index	0.50	-							
3. MPAI-4 Participation Index	0.54	0.62	-						
4. MPAI-4 Total Index	0.84*	0.85*	0.80*	-					
5. Rey Figure Copy score	-0.19	-0.08	-0.45	-0.24	-				
6. TMT A (Valenced)	-0.09	0.26	-0.16	0.06	0.66	-			
7. TMT B (Valenced)	-0.18	0.17	-0.3	-0.08	0.63	0.65	-		
8. SDMT	-0.26	0.23	-0.23	-0.06	0.6	0.69	0.59	-	
9. COWAT Animals	-0.33	-0.13	-0.32	-0.3	0.48	0.32	0.26	0.52	-

10. COWAT Letter F	-0.04	0.01	-0.39	-0.11	0.39	0.39	0.32	0.39	0.46
				Partial Controlling for Coping and Diathesis					
1. MPAI-4 Ability Index	-								
2. MPAI-4 Adjustment Index	0.49	-							
3. MPAI-4 Participation Index	0.51	0.59	-						
4. MPAI-4 Total Index	0.87*	0.83*	0.77*	-					
5. Rey Figure Copy score	-0.08	-0.17	-0.42	-0.21	-				
6. TMT A (Valenced)	0.01	0.14	-0.16	0.04	0.62*	-			
7. TMT B (Valenced)	-0.12	0.14	-0.23	-0.04	0.51	0.51	-		
8. SDMT	-0.29	0.00	-0.39	-0.25	0.53	0.46	0.46	-	
9. COWAT Animals	-0.39	-0.33	-0.52	-0.47	0.43	0.31	0.15	0.56*	-

10. COWAT Letter F	0.00	-0.11	-0.40	-0.13	0.27	0.26	0.15	0.25	0.43

p<0.01; Group with TBI, N=33.

Table 30. *Partial Correlations between EF and GOSe*

Measure	1	2	3	4	5	6
			Bivariate			
1. GOSe	-					
2. Rey Figure Copy score	0.37	-				
3. TMT A (Valenced)	0.18	0.66*	-			
4. TMT B (Valenced)	0.28	0.63*	0.65*	-		
5. SDMT	0.35	0.60*	0.69*	0.59*	-	
6. COWAT Animals	0.44*	0.48*	0.32	0.26	0.52*	-
7. COWAT Letter F	0.26	0.39	0.39	0.32	0.39	0.46*
			Partial Controlling for Coping and Diathesis			
1. GOSe	-					
2. Rey Figure Copy score	0.10	-				
3. TMT A (Valenced)	-0.04	0.61*	-			
4. TMT B (Valenced)	-0.06	0.51*	0.51*	-		

5. SDMT	0.40	0.53*	0.46	0.46	-	
6. COWAT Animals	0.51	0.43	0.31	0.15	0.56*	-
7. COWAT Letter F	0.16	0.27	0.26	0.15	0.25	0.43

p<0.01; Group with TBI, N=33.

Table 31. *Partial Correlations between WHOQOL-BREF and SRSI*

Measure	1	2	3	4	5	6	7
				Bivariate			
1. Strategy Awareness	-						
2. Emergent/Online Awareness	0.93*	-					
3. Readiness To Change	-0.14	-0.12	-				
4. Physical Health	-0.17	-0.10	0.09	-			
5. Psychological	0.08	0.24	0.33	0.61*	-		
6. Social Relationships	0.16	0.32	-0.05	0.32	0.70*	-	
7. Environment	0.12	0.19	0.21	0.55*	0.64*	0.46*	-
8. Total	0.03	0.16	0.19	0.82*	0.90*	0.70*	0.82*
				Partial Controlling for Coping and Diathesis			
1. Strategy Awareness	-						

2. Emergent/Online Awareness	0.94*	-					
3. Readiness To Change	-0.07	-0.04	-				
4. Physical Health	-0.18	-0.11	0.08	-			
5. Psychological	-0.03	0.08	0.45	0.58*	-		
6. Social Relationships	-0.04	0.12	0.00	0.38	0.61*	-	
7. Environment	0.10	0.15	0.31	0.39	0.57*	0.35	-
8. Total	-0.06	0.06	0.28	0.81*	0.87*	0.67*	0.75*

p<0.01; Group with TBI, N =33.

Table 32. *Partial Correlations between QOLIBRI and SRSI*

Measure	1	2	3	4	5	6	7	8	9
					Bivariate				
1. Strategy Awareness	-								
2. Emergent/ Online Awareness	0.93	-							
3. Readiness To Change	-0.14	-0.12	-						
4. Cognition	0.13	0.24	0.28	-					
5. Self	0.16	0.27	0.27	0.56*	-				
6. Daily Life & Autonomy	-0.09	0.01	0.24	0.52*	0.60*	-			
7. Social Relationships	0.02	0.11	-0.06	0.42	0.51	0.43	-		
8. Emotions	0.09	0.17	0.14	0.14	0.17	0.20	-0.02	-	
9. Physical Problems	0.04	0.13	-0.03	0.23	0.25	0.39	0.10	0.35	-
10. Total	0.08	0.23	0.20	0.69*	0.73*	0.78*	0.56*	0.52*	0.65*
					Partial Controlling for Coping and Diathesis				

1. Strategy Awareness	-								
2. Emergent/ Online Awareness	0.94*	-							
3. Readiness To Change	-0.07	-0.04	-						
4. Cognition	0.16	0.23	0.30	-					
5. Self	0.02	0.05	0.33	0.43	-				
6. Daily Life & Autonomy	-0.15	-0.06	0.30	0.38	0.57*	-			
7. Social Relationships	-0.23	-0.11	-0.08	0.38	0.30	0.22	-		
8. Emotions	0.09	0.11	0.15	0.05	-0.04	0.20	-0.06	-	
9. Physical Problems	0.02	0.14	-0.14	0.11	0.19	0.42	0.10	0.35	-
10. Total	-0.02	0.11	0.20	0.56*	0.58*	0.75*	0.45	0.53*	0.70*

p<0.01; Group with TBI, N =33.

Table 33. *Partial Correlations between SRSI and GOSe*

Measure	1	2	3
		Bivariate	
1. GOSe	-		
2. Strategy Awareness	-0.49*	-	
3. Emergent/ Online Awareness	-0.44*	0.93*	-
4. Readiness To Change	-0.21	-0.14	-0.12
		Partial Controlling for Coping and Diathesis	
1. GOSe	-		
2. Strategy Awareness	-0.40	-	
3. Emergent/ Online Awareness	-0.30	0.94*	-
4. Readiness To Change	-0.42	-0.07	-0.04

p<0.01; Group with TBI, N=33.

Table 34. *Partial Correlations between SRSI and MPAI-4*

Measure	1	2	3	4	5	6
			Bivariate			
1. MPAI Ability Index	-					
2. MPAI Adjustment Index	0.51*	-				
3. MPAI Participation Index	0.54*	0.62*	-			
4. MPAI Total Index	0.84*	0.85*	0.80*	-		
5. Strategy Awareness	0.41	0.03	0.41	0.33	-	
6. Emergent/ Online Awareness	0.35	-0.03	0.37	0.26	0.93*	-
7. Readiness To Change	0.11	-0.16	0.03	-0.02	-0.14	-0.12
			Partial Controlling for Coping and Diathesis			
1. MPAI Ability Index	-					

2. MPAI Adjustment Index	0.49*	-				
3. MPAI Participation Index	0.51*	0.59*	-			
4. MPAI Total Index	0.87*	0.83*	0.77*	-		
5. Strategy Awareness	0.55*	0.12	0.47	0.45	-	
6. Emergent/ Online Awareness	0.44	0.06	0.45	0.37	0.94*	-
7. Readiness To Change	0.12	-0.20	0.07	0.00	-0.07	-0.04

p<0.01; Group with TBI, N=33.

Table 35. *Partial Correlations between DEX-R and GOSe*

Measure	1	2	3	4
		Bivariate		
1. GOSe	-			
2. DEX-R Overall	0.34	-		
3. DEX-R Fluency, Flexibility, WM	0.39	0.92*	-	
4. DEX-R Social & Self -Regulation	0.29	0.96*	0.81*	-
5. DEX-R Motivation & Attention	0.29	0.88*	0.73*	0.80*
		Partial Controlling for Coping and Diathesis		
1. GOSe	-			
2. DEX-R Overall	0.26	-		
3. DEX-R Fluency, Flexibility, WM	0.30	0.90*	-	
4. DEX-R Social & Self -Regulation	0.21	0.97*	0.82*	-

5. DEX-R Motivation & Attention	0.19	0.83*	0.61*	0.75*

p<0.01; Group with TBI, N=33.

Table 36. *Partial Correlations between DEX-R and MPAI-4*

Measure	1	2	3	4	5	6	7
				Bivariate			
1. MPAI Ability Index	-						
2. MPAI Adjustment Index	0.50*	-					
3. MPAI Participation Index	0.54*	0.62*	-				
4. MPAI Total Index	0.84*	0.85*	0.80*	-			
5. DEX-R Overall	-0.23	-0.31	-0.40*	-0.35	-		
6. DEX-R Fluency, Flexibility, WM	-0.20	-0.24	-0.45*	-0.32	0.92*	-	
7. DEX-R Social & Self - Regulation	-0.18	-0.37	-0.31	-0.33	0.96*	0.81*	-
8. DEX-R Motivation & Attention	-0.29	-0.18	-0.40	-0.31	0.88*	0.73*	0.80*
				Partial Controlling for Coping and Diathesis			

1. MPAI Ability Index	-						
2. MPAI Adjustment Index	0.49*	-					
3. MPAI Participation Index	0.51*	0.59*	-				
4. MPAI Total Index	0.87*	0.83*	0.77*	-			
5. DEX-R Overall	-0.22	-0.57*	-0.47	-0.47	-		
6. DEX-R Fluency, Flexibility, WM	-0.23	-0.55*	-0.52	-0.49	0.90*	-	
7. DEX-R Social & Self - Regulation	-0.12	-0.58*	-0.34	-0.39	0.97*	0.82*	-
8. DEX-R Motivation & Attention	-0.32	-0.37	-0.48	-0.44	0.83*	0.61*	0.75*

p<0.01; Group with TBI, N=33.

Table 37. *Partial Correlations between WHOQOL-BREF and MPAI-4*

Measure	1	2	3	4	5	6	7	8
				Bivariate				
1. MPAI Ability Index	-							
2. MPAI Adjustment Index	0.50*	-						
3. MPAI Participation Index	0.54*	0.62*	-					
4. MPAI Total Index	0.84*	0.85*	0.80*	-				
5. Physical Health	-0.58*	-0.49*	-0.60*	-0.68*	-			
6. Psychological	-0.21	-0.36	-0.30	-0.36	0.61*	-		
7. Social Relationships	-0.14	-0.21	-0.21	-0.22	0.32	0.70*	-	
8. Environment	-0.13	-0.41	-0.19	-0.32	0.55*	0.64*	0.46*	-
9. Total	-0.36	-0.48*	-0.43	-0.52*	0.82*	0.90*	0.70*	0.82*
				Partial Controlling for Coping and Diathesis				
1. MPAI Ability Index	-							

2. MPAI Adjustment Index	0.49	-						
3. MPAI Participation Index	0.51	0.59*	-					
4. MPAI Total Index	0.87*	0.83*	0.77*	-				
5. Physical Health	-0.55*	-0.32	-0.56*	-0.58*	-			
6. Psychological	-0.22	-0.15	-0.20	-0.23	0.58*	-		
7. Social Relationships	-0.29	-0.02	-0.12	-0.19	0.38	0.61*	-	
8. Environment	-0.08	-0.28	-0.13	-0.19	0.39	0.57*	0.35	-
9. Total	-0.39	-0.28	-0.36	-0.42	0.81*	0.87*	0.67*	0.75*

p<0.01; Group with TBI, N=33.

Table 38. *Partial Correlations between WHOQOL-BREF and GOSe*

Measure	1	2	3	4	5
			Bivariate		
1. GOSe	-				
2. Physical Health	0.48*	-			
3. Psychological	0.10	0.61*	-		
4. Social Relationships	0.09	0.32	0.70*	-	
5. Environment	0.12	0.55*	0.64*	0.46*	-
6. Total	0.27	0.82*	0.90*	0.70*	0.82*
			Partial Controlling for Coping and Diathesis		
1. GOSe	-				
2. Physical Health	0.40	-			
3. Psychological	0.18	0.58*	-		
4. Social Relationships	0.45	0.38	0.61*	-	
5. Environment	0.18	0.39	0.57*	0.35	-
6. Total	0.38	0.81*	0.87*	0.67*	0.75*

p<0.01; Group with TBI, N=33.

Table 39. *Partial Correlations between QOLIBRI and GOSe*

Measure	1	2	3	4	5	6	7
				Bivariate			
1. GOSe	-						
2. Cognition	0.13	-					
3. Self	-0.01	0.57*	-				
4. Daily Life & Autonomy	0.34	0.52*	0.60*	-			
5. Social Relationships	0.20	0.42	0.51*	0.43	-		
6. Emotions	-0.04	0.14	0.17	0.20	-0.02	-	
7. Physical Problems	0.43	0.23	0.25	0.39	0.10	0.35	-
8. Total	0.28	0.69*	0.73*	0.78*	0.56*	0.52*	0.65*
				Partial Controlling for Coping and Diathesis			
1. GOSe	-						
2. Cognition	0.12	-					
3. Self	0.17	0.43	-				
4. Daily Life & Autonomy	0.19	0.38	0.57*	-			
5. Social Relationships	0.35	0.38	0.30	0.22	-		
6. Emotions	0.02	0.05	-0.04	0.20	-0.058	-	

7. Physical Problems	0.57*	0.11	0.19	0.42	0.10	0.35	-
8. Total	0.42	0.56*	0.58*	0.75*	0.45	0.53*	0.70*

p<0.01; Group with TBI, N=33.

Table 40. *Partial Correlations between QOLIBRI and MPAI-4*

Measure	1	2	3	4	5	6	7	8	9	10	11
					Bivariate						
1. MPAI Ability Index	-										
2. MPAI Adjustment Index	0.50*	-									
3. MPAI Participation Index	0.54*	0.62*	-								
4. MPAI Total Index	0.84*	0.85*	0.80*	-							
5. Cognition	-0.18	-0.27	-0.15	-0.25	-						
6. Self	-0.05	-0.41	-0.32	-0.30	0.56*	-					
7. Daily Life & Autonomy	-0.43	-0.46*	-0.63*	-0.58*	0.52*	0.60*	-				
8. Social Relationships	-0.11	-0.17	-0.21	-0.19	0.42	0.51*	0.43	-			
9. Emotions	-0.19	-0.12	0.19	-0.10	0.14	0.17	0.20	-0.02	-		
10. Physical Problems	-0.32	-0.27	-0.21	-0.35	0.23	0.25	0.39	0.10	0.35	-	

11. Total	-0.35	-0.42	-0.32	-0.45*	0.69*	0.73*	0.78*	0.56*	0.52*	0.65*
				Partial Controlling for Coping and Diathesis						
1. MPAI Ability Index	-									
2. MPAI Adjustment Index	0.49	-								
3. MPAI Participation Index	0.51	0.59*	-							
4. MPAI Total Index	0.87*	0.83*	0.77*	-						
5. Cognition	-0.20	-0.21	-0.03	-0.20	-					
6. Self	-0.02	-0.24	-0.36	-0.19	0.43	-				
7. Daily Life & Autonomy	-0.39	-0.31	-0.51	-0.46	0.38	0.57*	-			
8. Social Relationships	-0.07	0.19	0.04	0.06	0.38	0.30	0.22	-		
9. Emotions	-0.17	-0.06	0.25	-0.05	0.05	-0.04	0.20	-0.06	-	
10. Physical Problems	-0.36	-0.22	-0.28	-0.36	0.11	0.19	0.42	0.10	0.35	-

11. Total	-0.37	-0.24	-0.23	-0.35	0.56*	0.58*	0.75*	0.45	0.53*	0.70*

p<0.01; Group with TBI, N=33.

Table 41. *Differences between EF-related regions*

	Executive Functions								
		Group with TBI		Control Group		Statistics			
Areas	Side	M	SD	M	SD	*F*	P	η^2	Observed Power
Angular Area	L & R	0.42	0.07	0.42	0.07	0.26	0.61	0.01	0.08
Brodmann's 7	L & R	0.29	0.04	0.28	0.05	0.10	0.75	0.00	0.06
Brodmann's 8	L & R	0.33	0.04	0.36	0.05	11.63	0.00	0.19	0.92
Caudate	L & R	0.20	0.04	0.24	0.04	21.36	0.00	0.30	0.99
Cerebellum	L & R	0.29	0.04	0.31	0.04	4.45	0.04	0.08	0.54
Cingulate Cortex	R	0.44	0.07	0.51	0.09	13.39	0.00	0.21	0.95
Fusiform Gyrus	L & R	0.53	0.07	0.56	0.08	4.11	0.05	0.08	0.51
Globus Pallidus	L	0.08	0.01	0.09	0.01	29.48	0.00	0.37	1.00
Insula	L & R	0.45	0.06	0.54	0.07	32.15	0.00	0.39	1.00
Medulla		0.08	0.03	0.09	0.02	0.01	0.94	0.00	0.05
MPFC	L & R	0.33	0.04	0.36	0.05	11.56	0.00	0.19	0.92
OFC	L & R	0.35	0.05	0.43	0.06	33.31	0.00	0.40	1.00
Parahippocampus	R	0.37	0.05	0.37	0.04	0.39	0.53	0.01	0.09
Primary Sensory Cortex	L	0.46	0.07	0.53	0.11	10.35	0.00	0.17	0.88
Putamen	L & R	0.27	0.03	0.31	0.05	17.02	0.00	0.25	0.98
Supramarginal Gyrus	L & R	0.40	0.07	0.40	0.07	0.02	0.89	0.00	0.05
Temporal Cortex	L & R	0.45	0.06	0.51	0.06	24.68	0.00	0.33	1.00
Temporal Pole	R	0.40	0.08	0.47	0.06	17.32	0.00	0.26	0.98
Thalamus	L	0.40	0.08	0.49	0.10	15.34	0.00	0.23	0.97

Visual Association Cortex	L	0.28	0.04	0.28	0.04	0.07	0.80	0.00	0.06

p < 0.05; Group with TBI, N = 33; Control group, N=24.

Table 42. *Differences between SA-related regions*

	Self-awareness								
		Group with TBI		Control Group		Statistics			
Areas	Side	M	SD	M	SD	*F*	P	η^2	Observed Power
Angular Area	L & R	.45	.06	.48	.07	5.30	.025	.10	.62
Basal Ganglia	L & R	.23	.03	.27	.04	30.61	.000	.38	1.00
Brodmann's 7	L & R	.47	.07	.52	.09	7.37	.009	.13	.76
Brodmann's 8	L & R	.35	.04	.43	.07	31.74	.000	.39	1.00
Cingulate Cortex	L & R	.44	.05	.53	.08	29.62	.000	.37	1.00
Corpus Callosum	L & R	.27	.04	.33	.05	34.00	.000	.40	1.00
Globus Pallidus	R	.10	.01	.11	.01	15.65	.000	.24	.97
Hippocampus	L & R	.51	.06	.58	.05	30.27	.000	.38	1.00
Inferior Frontal Gyrus	L & R	.48	.06	.53	.06	10.77	.002	.18	.90
Insula	L	.50	.05	.57	.08	22.35	.000	.31	1.00
MPFC	L & R	.35	.04	.41	.06	28.71	.000	.36	1.00
OFC	L & R	.41	.06	.52	.09	35.87	.000	.42	1.00
Parahippocampus	R	.47	.06	.51	.05	8.23	.006	.14	.80
Pons	L & R	.11	.02	.13	.02	9.76	.003	.16	.86
Premotor Cortex	R	.26	.05	.26	.05	.12	.728	.00	.06
Primary Visual Cortex	L & R	.33	.04	.34	.04	3.57	.065	.07	.46

Putamen	L	.39	.04	.45	.05	31.86	.000	.39	1.00
Supramarginal Gyrus	L & R	.30	.05	.32	.07	2.06	.158	.04	.29
Temporal Area	L & R	.19	.04	.23	.06	15.22	.000	.23	.97
Temporal Cortex	L & R	.42	.06	.49	.06	28.15	.000	.36	1.00
Temporal Pole	L & R	.41	.07	.47	.06	18.06	.000	.27	.99
Visual Association Area	L	.38	.06	.41	.07	4.43	.048	.08	.54

$p < 0.05$; Group with TBI, N = 33; Control group, N=24.

Table 43. *Correlations between EF-related regions & SA-related regions*

Executive Functions	Basal Ganglia	Premotor Cortex	Insula	Parahippocampus	Supramarginal Gyrus	Temporal Area	Inferior Frontal Gyrus	Hippocampus	Corpus Callosum
Brodmann's 7	0.42*	0.60*	0.34	0.48*	0.68*	0.46*	0.36	0.42*	0.46*
Brodmann's 8	0.72*	0.61*	0.61*	0.81*	0.75*	0.55*	0.57*	0.63*	0.63*
Globus Pallidus	0.70*	0.22	0.72*	0.68*	0.43*	0.35	0.69*	0.59*	0.76*
Medulla	0.21	0.18	0.10	0.23	0.17	0.05	0.32	0.38	0.29
OFC	0.90*	0.33	0.79*	0.84*	0.56*	0.37*	0.64*	0.55*	0.80*
Primary Sensory Cortex	0.67*	0.50*	0.65*	0.79*	0.62*	0.47*	0.77*	0.50*	0.62*
Putamen	0.71*	0.28	0.68*	0.68*	0.53*	0.42*	0.72*	0.70*	0.73*
Parahipoccamp	0.43*	0.53*	0.36	0.52*	0.53*	0.37	0.27	0.51*	0.49*

us									
Temporal Cortex	0.74*	0.52*	0.65*	0.72*	0.63*	0.41	0.52*	0.62*	0.73*
Temporal Pole	0.72*	0.36	0.60*	0.59*	0.69*	0.39	0.48*	0.66*	0.67*
Thalamus	0.65*	0.36	0.60*	0.68*	0.51*	0.46*	0.49*	0.59*	0.62
Visual Association Cortex	0.35	0.59*	0.39	0.36	0.56*	0.34	0.10	0.33	0.35
Fusiform Gyrus	0.67*	0.39	0.64*	0.64*	0.77*	0.36	0.35	0.44*	0.66*
Insula	0.88*	0.33	0.91*	0.83*	0.55*	0.40	0.69*	0.62*	0.85*
Angula Area	0.52*	0.57*	0.48*	0.55*	0.87*	0.61*	0.31	0.42*	0.48*
Supramarginal Gyrus	0.39	0.53*	0.32	0.41	0.75*	0.55*	0.21	0.44*	0.37
Caudate	0.60*	0.24	0.54*	0.67*	0.38	0.37	0.60*	0.66*	0.68*
Cerebellum	0.67*	0.58*	0.60*	0.58*	0.61*	0.38	0.56*	0.58*	0.69*

Cingulate Cortex	0.80*	0.56*	0.65*	0.87*	0.64*	0.48*	0.75*	0.61*	0.76*
MPFC	0.74*	0.64*	0.62*	0.79*	0.72*	0.51*	0.58*	0.61*	0.71*

p<0.001, N=57.

Table 44. *Correlations between EF-related regions & EF construct*

	Executive Functions Construct		
		Group with	
	Whole Group	TBI	Control Group
1. Brodmann's 7	0.02	0.19	-0.39
2. Brodmann's 8	0.33*	0.13	-0.18
3. Globus Pallidus	0.35*	0.21	-0.40
4. Medulla	-0.04	-0.01	-0.03
5. OFC	0.33*	-0.02	-0.22
6. Primary Sensory	0.30	0.22	-0.27
7. Putamen	0.25	0.28	-0.58*
8. Parahippocampus	0.15	0.20	-0.13
9. Temporal Cortex	0.40*	0.16	-0.17
10. Temporal Pole	0.32	0.04	-0.14
11. Thalamus	0.33*	0.24	-0.31
12. Visual Association Cortex	0.09	0.10	0.03
13. Fusiform Gyrus	0.18	0.01	-0.04
14. Insula	0.38*	0.16	-0.29
15. Angular Area	0.13	0.15	-0.14
16, Supramarginal Gyrus	0.08	0.07	-0.13
17. Caudate	0.46*	0.36	-0.26
18. Cerebellum	0.11	-0.10	-0.34
19. Cingulate Cortex	0.37*	0.31	-0.27
20. MPFC	0.29	0.03	-0.17

p<0.01; Whole-group, N = 54; Group with TBI, N = 33; Control group, N =24.

Table 45. *Correlation between EF-related regions and DEX-R*

	Social & Self - Regulation	Flexibility, Fluency & WM	Motivation & Attention	Overall SA
1. Brodmann's 7	0.01	-0.09	0.01	-0.04
2. Brodmann's 8	0.27	0.30	0.24	0.30
3. Globus Pallidus	0.19	0.38*	0.36*	0.34*
4. Medulla	0.14	0.07	0.03	0.09
5. OFC	0.34*	0.46*	0.38*	0.43*
6. Primary Sensory	0.24	0.32	0.31	0.32
7. Putamen	0.24	0.34*	0.28	0.32
8. Parahippocampus	0.32	0.10	0.12	0.18
9. Temporal Cortex	0.24	0.29	0.38*	0.31
10. Temporal Pole	0.49*	0.51*	0.53*	0.55*
11. Thalamus	0.20	0.21	0.23	0.23
12. Visual Association	-0.04	-0.13	-0.09	-0.10
13. Fusiform Gyrus	0.06	0.02	0.03	0.03
14. Insula	0.26	0.38*	0.41*	0.38*
15. Angular Area	-0.02	-0.09	0.00	-0.05
16, Supramarginal Gyrus	0.00	-0.13	-0.04	-0.08
17. Caudate	0.31	0.41*	0.36*	0.39*
18. Cerebellum	0.30	0.25	0.29	0.29
19. Cingulate Cortex	0.27	0.33*	0.31	0.33*
20. MPFC	0.19	0.24	0.25	0.25

p<0.01; Whole-group, N = 54.

Table 45. *Correlation between EF-related regions and DEX-R*

	DEX-R Discrepancy Scores							
	Social & Self - Regulation		Flexibility, Fluency & WM		Motivation & Attention		Overall SA	
	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group
1. Brodmann's 7	-0.07	0.09	-0.19	-0.10	0.00	-0.14	-0.12	-0.07
2. Brodmann's 8	0.23	0.00	0.17	-0.18	0.10	-0.18	0.19	-0.16
3. Globus Pallidus	0.14	0.02	0.17	0.04	0.24	-0.08	0.19	0.01
4. Medulla	0.16	0.24	0.05	0.42	0.05	0.08	0.09	0.39
5. OFC	0.42	-0.11	0.37	-0.23	0.28	-0.17	0.39	-0.23
6. Primary Sensory	0.27	-0.03	0.23	0.09	0.36	-0.20	0.29	0.00
7. Putamen	0.30	-0.09	0.20	0.02	0.21	-0.15	0.25	-0.06
8. Parahippocampus	0.36	-0.13	0.06	-0.18	0.12	-0.15	0.18	-0.20
9. Temporal Cortex	0.20	-0.32	0.01	-0.14	0.27	-0.30	0.14	-0.28
10. Temporal Pole	0.59*	-0.25	0.47*	-0.06	0.52*	-0.26	0.55*	-0.19
11. Thalamus	0.15	-0.12	-0.02	-0.22	0.13	-0.31	0.07	-0.27
12. Visual Association Cortex	-0.09	0.08	-0.28	0.28	-0.18	0.06	-0.21	0.23
13. Fusiform Gyrus	-0.02	0.04	-0.15	-0.20	-0.14	-0.05	-0.11	-0.13
14. Insula	0.30	-0.13	0.24	-0.19	0.34	-0.15	0.30	-0.20
15. Angular Area	-0.13	0.05	-0.21	-0.17	-0.08	-0.09	-0.17	-0.11
16, Supramarginal Gyrus	-0.12	0.25	-0.28	-0.10	-0.18	0.11	-0.22	0.05
17. Caudate	0.27	0.06	0.21	0.06	0.22	-0.07	0.25	0.04
18. Cerebellum	0.28	0.25	0.18	-0.12	0.24	-0.07	0.24	0.00
19. Cingulate Cortex	0.27	-0.05	0.15	0.16	0.22	-0.09	0.22	0.06
20. MPFC	0.12	0.02	0.03	0.03	0.11	-0.01	0.08	0.03

p<0.01; Group with TBI, N = 33; Control group, N =24.

Table 46. *Correlation between EF-related regions and SRSI*

	SRSI		
	Strategy Awareness	Emergent/ Online Awareness	Readiness To Change
1. Brodmann's 7	-0.13	-0.08	-0.25
2. Brodmann's 8	-0.45*	-0.31	-0.14
3. Globus Pallidus	-0.32	-0.38	-0.37
4. Medulla	-0.45*	-0.41	-0.22
5. OFC	-0.36	-0.36	-0.11
6. Primary Sensory	-0.27	-0.28	-0.15
7. Putamen	-0.55*	-0.51*	-0.23
8. Parahippocampus	0-.48*	-0.46*	-0.25
9. Temporal Cortex	-0.41	-0.26	-0.17
10. Temporal Pole	-0.49*	-0.45*	-0.30
11. Thalamus	-0.30	-0.26	-0.30
12. Visual Association Cortex	-0.01	0.13	-0.19
13. Fusiform Gyrus	-0.35	-0.22	0.16
14. Insula	-0.38	-0.37	-0.39
15. Angular Area	-0.08	-0.04	-0.15
16, Supramarginal Gyrus	-0.01	0.01	-0.19
17. Caudate	-0.46*	-0.44*	-0.39
18. Cerebellum	-0.22	-0.17	-0.15
19. Cingulate Cortex	-0.44*	-0.35	-0.26
20. MPFC	-0.29	-0.19	-0.23

p<0.01; Group with TBI, N = 31.

Table 47. *Correlation between EF-related regions and WHOQOL-BREF*

	WHOQOL-BREF				
	Physical Health	Psychological	Social Relationships	Environment	Total
1. Brodmann's 7	0.14	0.05	-0.01	-0.05	0.04
2. Brodmann's 8	0.17	-0.06	0.00	-0.16	-0.02
3. Globus Pallidus	0.09	-0.23	0.04	-0.32	-0.16
4. Medulla	-0.16	-0.22	-0.13	-0.17	-0.23
5. OFC	0.08	-0.17	-0.07	-0.26	-0.14
6. Primary Sensory	0.03	-0.30	-0.20	-0.33	-0.26
7. Putamen	0.03	-0.19	0.04	-0.28	-0.15
8. Parahippocampus	0.00	-0.13	-0.13	-0.13	-0.12
9. Temporal Cortex	0.34	0.08	0.12	-0.17	0.11
10. Temporal Pole	0.12	-0.17	0.00	-0.25	-0.11
11. Thalamus	0.14	-0.02	0.11	-0.16	0.00
12. Visual Association Cortex	0.03	-0.02	-0.04	-0.07	-0.03
13. Fusiform Gyrus	0.12	0.07	-0.09	0.01	0.05
14. Insula	0.08	-0.19	-0.03	-0.28	-0.15
15. Angular Area	0.17	0.10	0.06	-0.04	0.09
16, Supramarginal Gyrus	0.06	0.09	0.18	-0.05	0.07
17. Caudate	0.21	-0.24	-0.04	-0.33	-0.14
18. Cerebellum	0.01	-0.19	-0.06	-0.16	-0.13
19. Cingulate Cortex	0.08	-0.22	-0.06	-0.34	-0.19
20. MPFC	0.07	-0.14	0.00	-0.29	-0.13

p<0.01; Whole-group, N = 55.

Table 48. *Correlation between EF-related regions and WHOQOL-BREF*

	WHOQOL-BREF									
	Physical Health		Psychological		Social Relationships		Environment		Total	
	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group
1. Brodmann's 7	0.22	-0.05	0.12	0.01	0.02	-0.01	-0.09	0.00	0.10	-0.01
2. Brodmann's 8	0.05	0.06	-0.03	0.08	-0.02	0.00	-0.17	0.13	-0.05	0.10
3. Globus Pallidus	-0.07	-0.11	-0.29	-0.12	-0.10	-0.02	-0.09	-0.20	-0.17	-0.16
4. Medulla	-0.08	-0.44	-0.18	-0.29	-0.17	-0.10	-0.20	-0.22	-0.18	-0.32
5. OFC	-0.17	0.00	-0.35	0.20	-0.31	-0.05	-0.28	0.14	-0.33	0.11
6. Primary Sensory	-0.18	-0.08	-0.37	-0.22	-0.25	-0.31	-0.27	-0.21	-0.32	-0.24
7. Putamen	-0.13	-0.12	-0.23	-0.10	-0.08	-0.04	-0.26	-0.05	-0.22	-0.09
8. Parahippocampus	-0.12	0.13	-0.31	0.32	-0.28	0.16	-0.45	0.28	-0.34	0.29
9. Temporal Cortex	0.35	0.00	0.20	0.21	0.07	0.10	0.06	0.04	0.23	0.10
10. Temporal Pole	0.02	-0.02	-0.28	0.11	-0.10	0.04	-0.27	0.17	-0.18	0.11
11. Thalamus	-0.03	0.06	-0.04	0.23	0.23	-0.14	-0.18	0.14	-0.03	0.13
12. Visual Association Cortex	0.05	-0.04	0.12	-0.22	-0.03	-0.05	-0.19	0.05	-0.01	-0.06
13. Fusiform Gyrus	0.02	0.11	0.04	0.20	-0.22	0.04	-0.02	0.24	-0.03	0.21
14. Insula	-0.15	0.02	-0.42	0.14	-0.23	-0.04	-0.35	0.13	-0.35	0.10
15. Angular Area	0.15	0.18	0.12	0.14	0.14	0.03	-0.21	0.15	0.06	0.16
16, Supramarginal Gyrus	0.02	0.11	0.06	0.18	0.16	0.34	-0.32	0.22	-0.03	0.25
17. Caudate	0.15	-0.16	-0.26	-0.10	-0.18	-0.02	-0.24	-0.14	-0.13	-0.14
18. Cerebellum	-0.09	-0.04	-0.28	0.01	-0.12	-0.01	-0.27	0.16	-0.23	0.06
19. Cingulate Cortex	0.01	-0.23	-0.26	-0.08	-0.07	-0.15	-0.43	-0.07	-0.22	-0.14
20. MPFC	-0.04	-0.12	-0.14	-0.02	-0.06	0.05	-0.25	-0.11	-0.15	-0.08

p<0.01; Group with TBI, N = 31; Control group, N = 24.

Table 49. *Correlation between EF-related regions and QOLIBRI*

	QOLIBRI						
	Cognition Scale	Self Scale	Daily Life & Autonomy Scale	Social Relationships Scale	Emotions Scale	Physical Problems Scale	Total Scale
1. Brodmann's 7	0.16	0.09	0.07	-0.01	-0.10	0.09	0.07
2. Brodmann's 8	-0.06	-0.12	-0.16	-0.13	-0.28	-0.13	-0.22
3. Globus Pallidus	-0.42	-0.27	-0.20	0.17	0.00	-0.01	-0.17
4. Medulla	-0.30	-0.31	-0.20	-0.10	-0.20	-0.14	-0.31
5. OFC	-0.28	-0.29	-0.29	-0.37	-0.39	-0.05	-0.40
6. Primary Sensory	-0.31	-0.37	-0.27	-0.09	-0.42	-0.56*	-0.55*
7. Putamen	-0.27	-0.16	-0.23	0.20	-0.03	-0.09	-0.15
8. Parahippocampus	-0.09	-0.35	-0.06	-0.16	-0.19	-0.10	-0.23
9. Temporal Cortex	0.22	-0.01	0.35	0.03	0.00	0.26	0.24
10. Temporal Pole	-0.22	-0.25	-0.12	0.06	-0.28	-0.01	-0.20
11. Thalamus	0.02	-0.01	0.07	0.36	-0.39	-0.23	-0.10
12. Visual Association Cortex	0.24	0.15	0.10	0.00	-0.26	-0.03	0.03
13. Fusiform Gyrus	0.28	-0.04	0.02	-0.25	0.09	0.06	0.06
14. Insula	-0.44	-0.48*	-0.33	-0.21	-0.40	-0.03	-0.44*
15. Angular Area	0.16	0.15	0.06	0.06	-0.15	0.11	0.09
16, Supramarginal Gyrus	0.13	0.13	-0.01	0.18	-0.19	0.01	0.04
17. Caudate	-0.36	-0.28	0.01	-0.15	-0.08	0.29	-0.08
18. Cerebellum	-0.30	-0.27	-0.18	-0.06	-0.23	-0.28	-0.35
19. Cingulate Cortex	-0.13	-0.25	-0.19	-0.13	-0.24	0.07	-0.20
20. MPFC	0.03	-0.27	-0.31	0.01	-0.17	0.15	-0.12

p<0.01; Group with TBI, N = 31.

Table 50. *Correlation between SA-related regions and DEX-R*

	DEX-R Discrepancy Scores								
	Flexibility, Fluency & WM			Social & Self - Regulation			Motivation & Attention		
	Whole Group	Group with TBI	Control Group	Whole Group	Group with TBI	Control Group	Whole Group	Group with TBI	Control Group
1. Basal Ganglia	0.30	0.40	-0.07	0.39*	0.29	-0.17	0.35*	0.29	-0.21
2. Premotor Cortex	0.25	0.27	0.07	0.12	0.15	-0.12	0.24	0.37	-0.13
3. Brodmann's 7	0.10	-0.05	0.04	0.10	-0.21	0.02	0.10	-0.10	-0.16
4. Brodmann's 8	0.26	0.26	0.01	0.40*	0.24	-0.01	0.31	0.16	-0.11
5. OFC	0.28	0.31	-0.08	0.44*	0.32	-0.08	0.33*	0.20	-0.22
6. Insula	0.17	0.23	-0.12	0.27	0.14	-0.19	0.36*	0.35	-0.11
7. Primary Visual Cortex	0.19	0.21	-0.24	0.12	0.00	-0.11	0.19	0.15	-0.32
8. Visual Association Area	-0.01	-0.11	-0.06	0.07	-0.15	-0.02	0.15	0.09	0.01
9. Temporal Cortex	0.37*	0.47*	-0.15	0.44*	0.35	-0.15	0.42*	0.38	-0.22
10. Cingulate Cortex	0.20	0.16	-0.08	0.31	0.02	0.01	0.28	0.10	-0.17
11. Parahippocampal	0.35*	0.39	-0.22	0.26	0.09	-0.11	0.29	0.21	-0.27
12. Temporal Pole	0.29	0.33	-0.28	.39*	0.28	-0.05	.37*	0.30	-0.31
13. Angular Area	0.11	0.00	0.17	0.09	-0.09	-0.12	0.17	0.05	0.07
14. Supramarginal Gyrus	0.14	0.11	0.02	0.27	0.31	-0.13	0.32	0.38	-0.01
15. Temporal Area	0.21	0.17	-0.12	0.36*	0.19	0.13	0.27	0.18	-0.23
16. Inferior Frontal Gyrus	0.27	0.28	-0.03	0.42*	0.42	0.00	0.36*	0.32	-0.17
17. Putamen	0.20	0.18	-0.06	0.33	0.12	-0.19	0.32	0.14	-0.15
18. Globus Pallidus	0.30	0.28	0.20	0.31	0.12	-0.04	0.30	0.15	-0.05
19. Hippocampus	0.32	0.32	-0.14	0.38*	0.12	0.00	0.39*	0.24	-0.22
20. Corpus Callosum	0.26	0.27	0.01	0.40*	0.23	-0.07	0.33*	0.19	-0.14

21. MPFC	0.27	0.27	0.03	0.40*	0.25	0.00	0.31	0.18	-0.10
22. Pons	0.29	0.31	-0.07	0.24	0.15	-0.27	0.34*	0.30	-0.05

p<0.01; Whole-group, N = 55; Group with TBI, N = 31; Control group, N = 24.

Table 51. *Correlation between SA-related regions and SRSI*

	SRSI		
	Strategy Awareness	Emergent/ Online Awareness	Readiness To Change
1. Basal Ganglia	-0.34	-0.31	-0.24
2. Premotor Cortex	-0.21	-0.16	-0.30
3. Brodmann's 7	-0.34	-0.29	-0.27
4. Brodmann's 8	-0.43	-0.31	-0.15
5. OFC	-0.29	-0.24	-0.09
6. Insula	-0.34	-0.34	-0.25
7. Primary Visual Cortex	-0.53*	-0.53*	-0.12
8. Visual Association Area	0.00	0.05	-0.19
9. Temporal_Cortex	-0.51*	-0.48*	-0.10
10. Cingulate Cortex	-0.46*	-0.37	-0.29
11. Parahippocampal	-0.43	-0.33	-0.33
12. Temporal Pole	-0.45*	-0.46*	-0.13
13. Angular Area	-0.29	-0.18	-0.20
14. Supramarginal Gyrus	-0.17	-0.19	-0.30
15. Temporal Area	-0.37	-0.50*	-0.13
16. Inferior Frontal Gyrus	-0.40	-0.35	-0.20
17. Putamen	-0.19	-0.15	-0.10
18. Globus Pallidus	-0.37	-0.36	-0.52*
19. Hippocampus	-0.30	-0.24	-0.48*
20. Corpus Callosum	-0.37	-0.31	-0.20
21. MPFC	-0.44*	-0.32	-0.17
22. Pons	-0.26	-0.20	-0.47*

p<0.01; Group with TBI, N = 31;

Table 52. *Correlation between SA-related regions and WHOQOL-BREF*

	WHOQOL-BREF				
	Physical Health	Psychological	Social Relationships	Environment	Total
1. Basal Ganglia	0.12	-0.12	-0.02	-0.22	-0.09
2. Premotor Cortex	0.10	-0.07	-0.01	-0.07	-0.01
3. Brodmann's 7	0.01	-0.18	-0.13	-0.28	-0.19
4. Brodmann's 8	0.08	-0.18	-0.02	-0.28	-0.14
5. OFC	0.11	-0.20	-0.07	-0.24	-0.13
6. Insula	0.16	-0.08	0.00	-0.19	-0.04
7. Primary Visual Cortex	0.16	-0.08	-0.09	-0.12	-0.03
8. Visual Association Area	0.17	-0.02	0.10	-0.11	0.03
9. Temporal_Cortex	0.23	-0.16	-0.11	-0.28	-0.10
10. Cingulate Cortex	0.10	-0.18	-0.04	-0.30	-0.15
11. Parahippocampal	0.09	-0.16	-0.08	-0.23	-0.13
12. Temporal Pole	0.14	-0.19	-0.08	-0.25	-0.12
13. Angular Area	0.23	0.03	0.04	-0.09	0.06
14. Supramarginal Gyrus	0.32	0.06	0.10	-0.04	0.14
15. Temporal Area	-0.01	-0.36*	-0.18	-0.41*	-0.32
16. Inferior Frontal Gyrus	0.09	-0.13	-0.10	-0.12	-0.08
17. Putamen	0.16	-0.07	0.10	-0.19	-0.02
18. Globus Pallidus	0.09	-0.16	0.01	-0.26	-0.12
19. Hippocampus	0.13	-0.24	-0.09	-0.40*	-0.21
20. Corpus Callosum	0.10	-0.18	0.01	-0.26	-0.12
21. MPFC	0.09	-0.17	-0.01	-0.28	-0.14
22. Pons	0.15	0.08	0.14	-0.09	0.07

p<0.01; Whole-group, N = 55.

Table 53. *Correlation between SA-related regions and WHOQOL-BREF*

	WHOQOL-BREF									
	Physical Health		Psychological		Social Relationships		Environment		Total	
	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group	Group with TBI	Control Group
1. Basal Ganglia	-0.10	0.02	-0.29	0.16	-0.20	-0.08	-0.27	0.16	-0.25	0.11
2. Premotor Cortex	0.02	0.18	-0.14	0.05	0.02	0.73	-0.37	0.50	-0.14	0.20
3. Brodmann's 7	-0.17	0.04	-0.26	0.05	-0.27	0.74	-0.34	0.41	-0.31	-0.03
4. Brodmann's 8	-0.16	-0.11	-0.18	-0.09	-0.10	-0.04	-0.11	-0.04	-0.17	-0.12
5. OFC	-0.18	0.12	-0.30	0.17	-0.22	0.14	-0.12	0.09	-0.25	-0.01
6. Insula	0.03	0.06	-0.33	0.07	-0.20	0.06	-0.29	0.21	-0.22	0.16
7. Primary Visual Cortex	0.10	-0.09	-0.12	-0.06	-0.09	0.10	-0.30	0.13	-0.11	0.14
8. Visual Association Area	0.20	0.12	0.05	-0.22	0.20	0.15	-0.03	0.23	0.13	-0.27
9. Temporal Cortex	0.08	0.12	-0.25	0.06	-0.27	0.06	-0.27	0.12	-0.18	0.05
10. Cingulate Cortex	-0.15	0.09	-0.33	0.05	-0.19	-0.14	-0.29	-0.01	-0.29	-0.03
11. Parahippocampal	-0.11	0.10	-0.32	0.02	-0.18	0.07	-0.42	0.01	-0.31	0.09
12. Temporal Pole	0.00	0.04	-0.27	0.08	-0.14	0.10	-0.17	0.12	-0.16	0.24
13. Angular Area	0.20	0.11	0.08	0.13	0.00	0.06	-0.11	0.07	0.07	0.14
14. Supramarginal Gyrus	0.46	0.05	0.08	0.17	0.10	0.01	0.08	0.16	0.25	0.27
15. Temporal Area	-0.32	0.08	-0.53*	0.06	-0.22	0.15	-0.43	0.12	-0.47	0.23
16. Inferior Frontal Gyrus	0.01	0.13	-0.20	0.04	-0.17	0.08	0.03	0.22	-0.08	0.26

17. Putamen	-0.01	0.08	-0.16	0.10	-0.03	0.06	-0.11	0.18	-0.10	0.10
18. Globus Pallidus	-0.06	0.76	-0.24	0.47	-0.18	0.73	-0.35	0.47	-0.24	0.49
19. Hippocampus	-0.04	0.95	-0.34	0.90	-0.22	0.58	-0.41	0.52	-0.29	0.65
20. Corpus Callosum	-0.14	0.95	-0.31	0.86	-0.15	0.44	-0.20	0.80	-0.24	0.74
21. MPFC	-0.13	1.00	-0.16	0.91	-0.09	0.75	-0.10	0.86	-0.15	0.95
22. Pons	0.06	0.62	0.01	0.68	0.07	0.60	-0.22	0.64	-0.02	0.57

p<0.01; Group with TBI, N = 31; Control group, N = 24.

Table 54. *Correlation between SA-related regions and QOLIBRI*

	QOLIBRI						
	Cognition	Self	Daily Life & Autonomy	Social Relationships	Emotions	Physical Problems	Total
1. Basal Ganglia	-0.16	-0.29	-0.26	-0.11	-0.24	0.04	-0.24
2. Premotor Cortex	-0.15	-0.11	0.03	0.11	-0.29	-0.17	-0.17
3. Brodmann's 7	0.03	-0.26	-0.32	-0.21	-0.29	-0.14	-0.30
4. Brodmann's 8	-0.08	-0.34	-0.37	-0.08	-0.18	-0.03	-0.26
5. OFC	-0.13	-0.38	-0.37	-0.34	-0.22	-0.02	-0.34
6. Insula	-0.31	-0.37	-0.13	0.00	-0.29	0.03	-0.25
7. Primary Visual Cortex	-0.03	-0.31	0.11	-0.18	-0.14	-0.17	-0.18
8. Visual Association Area	-0.04	0.02	0.13	0.04	0.12	0.09	0.11
9. Temporal_Cortex	-0.31	-0.46*	-0.01	-0.18	-0.11	0.00	-0.23
10. Cingulate Cortex	-0.10	-0.41	-0.27	-0.33	-0.24	-0.01	-0.31
11. Parahippocampal	-0.13	-0.34	-0.14	-0.10	-0.19	0.00	-0.21
12. Temporal Pole	-0.35	-0.43	-0.13	-0.14	0.06	-0.02	-0.22
13. Angular Area	0.14	0.07	0.06	-0.10	-0.18	0.05	0.01
14. Supramarginal Gyrus	-0.10	0.18	0.06	0.24	-0.12	0.08	0.07
15. Temporal Area	-0.52*	-0.55*	-0.34	-0.03	-0.17	-0.29	-0.47*
16. Inferior Frontal Gyrus	-0.31	-0.31	-0.20	-0.31	0.21	0.15	-0.14
17. Putamen	-0.14	-0.18	-0.17	0.01	-0.13	0.04	-0.13
18. Globus Pallidus	-0.20	-0.29	-0.28	-0.04	-0.40	0.00	-0.29
19. Hippocampus	-0.23	-0.45*	-0.20	-0.16	-0.31	0.14	-0.26
20. Corpus Callosum	-0.08	-0.36	-0.30	-0.19	-0.15	0.10	-0.22
21. MPFC	-0.08	-0.32	-0.35	-0.06	-0.16	-0.03	-0.24
22. Pons	-0.15	-0.09	0.00	0.21	-0.25	-0.08	-0.11

p<0.01; Group with TBI, N = 31.