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**THE IMPACT OF POSTTRAUMATIC STRESS DISORDER ON  
EXECUTIVE FUNCTIONING**

**by**

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## DEDICATION

Dedicated in gratitude for the loving support from both my parents, Charl and Sophia de Kock, whose love, continuous support, and encouragement made this work possible.

*Never stop just because you feel defeated. The journey to the other side is attainable only after great suffering. – Santosh Kalwar*

## DECLARATION

I, Cornelius Johannes de Kock, declare that this dissertation hereby submitted by me as part of the Magister Artium degree in Counselling Psychology in the Faculty of Humanities in the Department of Psychology at the University of Pretoria is my own independent work and has not previously been submitted for any other degree, part of degree, or examination at this or any other faculty or university. According to my knowledge, this manuscript does not represent any conflict of interest and did not receive any research funding. Furthermore, I cede copyright of this dissertation in favour of the University of Pretoria.

\_\_\_\_\_ day of \_\_\_\_\_ 2019  
Cornelius Johannes de Kock

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## ABSTRACT

**Background:** Most of the neurocognitive research in Posttraumatic Stress Disorder (PTSD) thus far focused on impairment in learning and memory, neglecting the impact of PTSD on executive functioning processes. Therefore, this study specifically aims to investigate the impact of PTSD on frontal lobe executive functioning. Given the high prevalence rate for traumatic event exposure in South Africa, this study provides important findings on the role intact executive functioning plays in all areas of daily functioning, including the maintenance of good mental and physical health.

**Methods:** Executive functions were assessed using an Executive Functioning Battery consisting of the three subtests of the Delis Kaplan Executive Functioning System (e.g., Trail Making Test, Colour-Word Interference Test, and Tower Test), as well as the Executive Functioning Index. The study sample consisted of 88 adult South African citizens who were divided into two groups (PTSD+;  $n = 44$ ; PTSD-;  $n = 44$ ) with different levels of trauma exposure.

**Results:** PTSD was linked with impairment in executive functioning domains such as attention, cognitive flexibility, inhibition, working memory, and planning. Important gender differences were also reported in terms of empathy and organisation. In addition, education also appeared to affect frontal lobe executive functioning differently.

**Conclusions:** The data suggest that overall, PTSD impaired executive functioning processes. It is therefore critical that assessment of executive functioning form part of a comprehensive treatment plan for individuals diagnosed with PTSD.

**Key Words:** Stress; Trauma; Posttraumatic Stress Disorder (PTSD); Cognition; Executive Functions; Prefrontal Lobes, Neuropsychological Impairments; Working Memory; Inhibitory Control; Cognitive Switching; Planning; Attention; Gender Differences; Education; Delis Kaplan Executive Function System (D-KEFS); D-KEFS Trail Making Test; D-KEFS Colour-Word Interference Test; D-KEFS Tower Test; Executive Functioning Index (EFI).



## INTRODUCTION

In the field of health psychology, the concept of health is described as a biopsychosocial process (Taylor, Peplau, & Sears, 2006). A person's state of health is a complex interaction of biological factors (e.g., a genetic predisposition to a particular disease or exposure to a flu virus); psychological factors (e.g., the experience of stress); and social factors (e.g., the amount of social support a person receives from friends and family) (Taylor et al., 2006).

In health psychology it is further argued that psychological trauma or repeated traumata can play an essential role in the development of several mental illnesses. According to both the international and American diagnostic systems, the fundamental characteristic clinical picture resulting from psychological trauma, is Posttraumatic Stress Disorder (PTSD). Epidemiological studies furthermore reveal that PTSD poses genuine medical and social dilemmas (Hašto, Vojtová, Hrubý, & Tavel, 2013). Although the current investigation follows a biopsychosocial model of mental illness, the focus lies specifically on the neurocognitive aspects associated with PTSD.

Serious concerns arise regarding the impact of the high levels of exposure to trauma and violence of various kinds on the South African society at large. According to Kaminer and Eagle (2010), South African citizens are not only personally affected by traumatic events, but are also frequently and extensively confronted with anecdotal accounts of trauma experienced by friends and family, as well as by traumatic events reported in the media. These accounts are often articulated in the discourse of living in a dangerous and traumatized society. We live in a world torn and scarred by violence and numerous socio-environmental stressors that are experienced as trauma. All these affect the neurology of the brain (Kirmayer, Lemelson, & Barad, 2007) and in turn is mediated by social input (Christopher, 2004). Kirmayer et al. (2007)

further express that globalization has increased the tempo, as well as the magnitude of conflicts and catastrophes. According to Kirmayer et al. (2007), violence has been integral to the human condition from our earliest origins. It is therefore not surprising to stumble upon its traces in the architecture of our brains and bodies. Violence is part of the fabric of society. We are more aware of global tragedies through our technologies, which carries the raw reality of violence and individual's genuine sense of insecurity into our living rooms. Violence has become the defining organisational principle for society. It mediates all social relations. It does not matter whether we are actual victims of violence. Just the possibility that we could encounter some form of violence is enough to direct our lives. New media technologies have placed a traumatised world in our hands and redefined the relationship between producer and audience.

In an interview between Natasha Lennard, reporter for The New York Times and Brad Evans, senior lecturer from the University of Bristol in England (Lennard & Evans, 2015), Evans states that violence remains a complex problem that defies description. According to Evans, violence is too often considered in an objective and neutral way, forgetting that it is humans being violated and that such experiences are horrific and devastating. Evans continues by emphasising that violence will remain poorly understood if we only focus on mere bodily attacks. Psychological abuse is also a form of violence, just as social neglect, unnecessary suffering caused by preventable diseases, and environmental depravity are, given their effects on human life. Thinking holistically against violence is therefore not only timely, but also crucial in the contemporary moment.

Evans draws our attention to Auguste Rodin's sculpture "The Thinker", which according to him is still one of the most famous human embodiments of philosophical and critical enquiry. The form given to Rodin's isolated and contemplative sculpture struck a chord with this researcher. Embroidering on Evans' perspective, this researcher is wondering what the thinker is in fact contemplating. Sitting alone on his plinth, he is hopefully thinking of

something serious. Rodin however did not intend any ambiguity in his original 1880 sculpture. The thinker appears kneeling before the Gates of Hell. The context of Rodin's thinker is thus a scene of violence. Thinking begins for the thinker in the presence of the reality of violence and suffering, forcing him to suffer into truth. Evans continues to recognise the tension in the thinker's relationship to violence. Being placed before the Gates of Hell, the thinker appears to be turning away from the unbearable scene behind him. This could be our tendency also when thinking about violence today. Too often we turn away toward abstraction and/or a scientific neutral position—opting for objectivity. Evans believes however that Rodin's thinker is actually Dante, who is considering the nine circles of hell as told by Dante Alighieri in his 14<sup>th</sup> century epic poem, the Divine Comedy. The thinker is therefore not looking away, but actually staring directly into the abyss below. This viewpoint raises within Evans the fundamental ethical question of what it means to be a forced witness to violence? For this researcher, it begs the question, what is the effect of trauma on the human brain and especially, how does it affect the executive functioning abilities of individuals suffering from PTSD? “The Thinker” was originally seen by Rodin as a tortured being, yet as a freethinking being, he is determined to surpass his suffering through poetry. This researcher will endeavour to re-think the impact of PTSD on frontal lobe executive functions, imagining a better understanding of this relationship and thereby enabling researchers to create a future worth living for those affected by trauma.

This journey will begin by reviewing a wide collection of literature on executive functioning as the most important frontal lobe function. This will include an exploration of the pivotal relationship between the frontal lobes and executive functions. Chapter 2 will be devoted to the understanding of stress and its relationship to PTSD. This relationship will be placed within the South African context. In the third chapter the main constructs will be brought together by connecting prefrontal executive functions, stress, and PTSD. In Chapter 4 the methodology employed in this study will be presented. In this chapter the rationale of the study

will also be explained. The findings will be presented in the fifth chapter and finally this study will conclude in Chapter 6 with a discussion of the results, where after conclusions will be formulated and recommendations for further investigation into the relationship between PTSD and frontal lobe executive functioning in the South African context will be made.

## CHAPTER 1

### FRONTAL LOBE EXECUTIVE FUNCTIONS

*“Dubito ergo cogito, cogito ergo sum.”*

*(I doubt therefore I think; I think therefore I am.) – René Descartes (1596–1650)*

According to Lezak, Howieson, Bigler, and Tranel (2012), it is possible to envisage human behaviour through three functional systems: (1) Cognition, which serves as the information-handling aspect of behaviour; (2) emotionality, which includes feelings and motivation; and (3) executive functions, which has to do with how behaviour is expressed.

We can consider each of these three sets of functions fundamental in every aspect of behaviour. Each can be conceptualised and dealt with separately even though they are intimately interconnected in complex behaviour. Lezak et al. (2012) consider executive functions the most complex of behaviours. Executive functions are intrinsic to the ability to respond in an adaptive manner to novel situations and furthermore form the basis of many cognitive, emotional, and social skills (Lezak et al., 2012).

This chapter specifically sets out to investigate executive functioning as the most important frontal lobe function. This investigation is twofold and will begin with an attempt to define and discuss frontal lobe functioning, in particular executive functions. Once the theoretical constructs associated with frontal lobe executive functioning have been addressed the second part of this chapter will endeavour to dissect the neuro-anatomical correlates of frontal lobe executive functioning in order to provide a comprehensive understanding of what constitutes frontal lobe executive functioning.

In the first section of this chapter, the theoretical underpinnings of frontal lobe functioning will be addressed by focusing on five specific theories that attempt to explain

frontal lobe functioning. Thereafter, frontal lobe executive functioning will be defined and discussed by distinguishing executive functions from cognitive functions and by addressing the hot and cold components of executive functioning, which represents the metacognitive and emotional/motivational activities of the frontal lobes. Thereafter a discussion will follow attempting to establish if frontal lobe executive functions can be divided into recognisable sub-processes or constitute a single mental process. In other words, an attempt will be made to answer the question whether Norman and Shallice's supervisory attentional system can be fractionated into unitary or non-unitary aspects by reviewing current research opinions and existing tripartite models of executive functioning. Given the valuable contribution that the unitary versus non-unitary debate holds for future research regarding executive functioning components, the researcher will align himself with a particular definition that supports his stance towards the debate concerning whether executive functioning processes constitutes a unitary or non-unitary construct. This section will be concluded by a discussion regarding the similarities and disparities of executive functions and their related terms followed by the differentiation between executive and non-executive functions, after which the value of executive functions will be addressed.

In the second section, after having defined executive functioning, attention will shift towards the neuroanatomical underpinnings of frontal lobe executive functions, with specific interest towards the sub-divisions of the frontal lobes and their general sub-cortical circuitry. An overview of three major frontal-subcortical circuits will be provided with particular interest in the dorsolateral prefrontal cortex, because of its role in mediating executive functions. A schematic illustration of the fractionation of frontal lobe executive functioning is also provided to assist the reader with a concise summary of the anatomical and functional connectivity of the general sub-divisions of the frontal lobes. This chapter will conclude with a brief discussion

about the complexity of localising executive control processes to a single brain region such as the prefrontal cortex.

## **1.1 Theoretical Underpinnings of Frontal Lobe Functioning**

There are numerous existing theories that attempt to explain frontal lobe functioning. Amongst these theories there appear to be similarities and differences regarding the functioning of the frontal lobes. In the subsequent section, Luria's classical view, Norman and Shallice's supervisory attentional system, Stuss and Benson's tripartite model, Rolls' theory of orbitofrontal functioning, and Damasio's somatic marker hypothesis, will be briefly discussed. Thereafter, the researcher will attend to align himself with Luria as well as Norman and Shallice's theory of frontal lobe functioning as his guiding theoretical framework in this research.

### **1.1.1 Luria's theory: The classical view.**

Luria (1973) believes that the human brain is made up of three basic functional units that are interactively connected. Located in the brain stem, the first unit is responsible for regulating and maintaining arousal of the cortex. The second unit is responsible for the encoding, processing, and storage of information and include the temporal, parietal, and occipital lobes. Luria (1973) views the frontal lobes as the third functional brain unit that is responsible for the programming, regulation, and verification of human behaviour. In addition, Luria views the prefrontal cortex as the area that is responsible for the control of the general state of the cortex and basic human mental activity (Chan, Shum, Touloupoulou, & Chen, 2008).

According to Luria (1973), one of the main functions of the frontal lobes is the ability to compare the outcome of an action with the original intent of that specific action. The impediment of this function is commonly regarded as the clearest sign that damage to the frontal lobes has occurred following head trauma. Luria further emphasises that the prefrontal

areas are essential for creating meaningful plans as well as for acting upon the consequences of those plans. Damage to the frontal cortex results in an inability to follow complex plans for behaviour and leads to more basic forms of behaviour or stereotypical behaviour that is irrelevant to the situation or illogical, thus making it difficult to disregard or inhibit irrelevant information.

Luria (1973) also noted that damage to the lateral regions of the frontal lobes results in profound disturbance in motoric behaviour, because the frontal lobes are closely connected to motor brain structures. Damage to areas that are connected to the limbic system and the reticular formation results in disinhibition and changes in affect.

Patients with damage to their prefrontal regions struggle to ignore or disinhibit irrelevant information. The reason being that damage occurred to the filtrating mechanisms of the frontal lobes (Beer, Shimamura, & Knight, 2004). The role of the filtrating mechanism is to inhibit irrelevant information. The reason why diverse symptoms follow damage to different areas of the prefrontal cortex is not that each part is responsible for the disturbing symptoms, but that different connections in the prefrontal cortex are responsible for the filtration of different aspects of cognition. Consequently, difficulties in attention can be seen as a failure to inhibit irrelevant stimuli or information. Memory problems can be perceived as an inability to inhibit previous memories. Difficulty with problem-solving can be attributed to the failure to inhibit irrelevant or incorrect search strategies (Gazzaniga, Ivry, Mangun, & Steven, 2009).

### **1.1.2 Norman and Shallice's supervisory attentional system (SAS).**

Norman and Shallice's (1986) theory of supervisory attentional system (SAS) and dysfunction is based on a similar idea like those ascribed by Luria in his classical view of frontal lobe functioning. That is, the frontal lobes are responsible for the programming,



regulation, and verification of human activity. Therefore, supporting the notion that the prefrontal cortex is the seat of high-level processes that modulate lower-level ones.

Norman and Shallice's (1986) theory proposes that there are two frontal lobe systems involved in the selection and control of thoughts and action, namely contention scheduling and frontal lobe supervision of attention (or executive attention). According to Norman and Shallice (1986) the environment in which a man finds himself often requires a specific action. The contention scheduling system of the frontal lobes serves as a mediator between the environmental demands and the selection of automatic or routine action. Activation of the contention scheduling system therefore inhibits inappropriate forms of automatic action within a specific environment, so that the most appropriate action can be selected based on the environmental demands. The frontal supervisory attentional system comes into effect during non-routine situations when action needs to be changed or inhibited for the sake of a new environmental demand or decision-making process. The system of frontal supervision is responsible for the selection of appropriate action with the help of contention scheduling. In addition, the frontal supervisory system is also required in situations that involve (1) planning or decision-making, (2) correction of errors or troubleshooting, (3) behaviour that is ill-learned or contain novel sequences of actions, (4) are judged to be dangerous or technically difficult, and (5) that requires of an individual to ignore a strong habitual response or the resisting of temptation (Chan et al., 2008; Norman & Shallice, 1986; Posner & DiGirolamo, 1998). Frontal supervision thus requires the formulation of potential action that is based on environmental demands, selection of a possible action, and the evaluation of whether the selected action brought about the desired outcome. After frontal lobe damage, it is possible that one or more of these processes may be impaired which may result in the frontal supervisory system being fragmented instead of acting as a coherent unit (Shallice, Burgess, & Robertson, 1996).

Burgess and colleagues (Burgess, 2000; Burgess, Veitch, de Lacy Costello, & Shallice, 2000), extended Norman and Shallice's (1986) SAS concept to include multitasking performance in everyday life. They identified the following seven features of multitasking behaviour:

1. Numerous tasks: The individual must complete multiple discreet tasks.
2. One task at a time: The individual must be able to perform one task at a time.
3. Interleaving required, also referred to by Lezak et al. (2012) as cognitive set-shifting: Finding the most effective course of action by dovetailing tasks.
4. Delayed intentions: The time for an individual to return to a task which is already running and is not directly coupled to the situation.
5. Interruptions: Sporadic interruptions and unexpected outcomes.
6. Differing task characteristics: Tasks differing in priority, difficulty, and amount of time they will take to complete.
7. No feedback: The individual continues with a task without receiving feedback about his/her progression.

Burgess et al. (2000) is aware that not all laboratory-based tasks include all of the abovementioned features in assessing the components of multitasking performance in clinical settings.

### **1.1.3 Stuss and Benson's tripartite model.**

Stuss and Benson (1986) believe that there are three interactive systems located inside the human brain that collectively work together to monitor an individual's attention and executive functions. These systems include (1) the anterior reticular activating system (ARAS), (2) the diffuse thalamic projection system, and (3) the fronto-thalamic gating system.

According to Stuss and Benson (1986), the ARAS and the diffuse thalamic projection system are responsible for maintaining an individual's alertness, while the fronto-thalamic gating system is engaged during executive attentional control. More specifically, the ARAS is responsible for maintaining the general arousal level of an individual and damage sustained to this system will result in loss of consciousness. The diffuse thalamic projection system enables an individual to maintain his/her alertness towards external stimuli over a short period of time, and a breakdown occurring in this system will result in an individual being distracted by external stimuli. The fronto-thalamic gating system is in charge of higher-order cortical functioning, which includes planning, stimuli and response selection, as well as monitoring of daily performance. If a disruption occurs in this system, it will cause symptoms such as inattention, inability to view aspects in a logical manner, and goal-neglect behaviour that corresponds with similar dysfunctions associated with a breakdown in the SAS. The role of the frontal lobes in attention was expanded when Stuss, Shallice, Alexander, and Picton (1995) examined the role of the frontal lobes in the control of attention from two fundamental perspectives. According to Stuss et al. (1995), the first perspective is concerned with a set of supervisory component processes (e.g., monitoring, energizing, inhibiting, adjusting) that are active during different attentional tasks (e.g., sustained attention, concentrating, sharing, suppressing, switching, preparing, and goal setting). The second perspective is concerned with cerebral mechanisms (neuro-anatomical structures) that are critical for the execution of these different attentional tasks. The strength of this theory is embedded in the way these two fundamental perspectives led Stuss et al. (1995) to identify the neural basis of different executive attentional components in the frontal lobes during the execution of various attentional tasks (Chan et al., 2008).

By broadening their understanding of the relationship between the schema and the SAS, Stuss et al. (1995) were able to link sustained attentional tasks to the right frontal brain

region, concentrating to the anterior cingulate area, sharing of attention to the anterior cingulate and orbitofrontal areas, suppressing to dorsolateral prefrontal cortex, switching to both the dorsolateral prefrontal and medial frontal areas, preparing to the dorsolateral prefrontal cortex, and goal setting to the left dorsolateral prefrontal cortex (Chan et al., 2008; Stuss, 2011; Stuss et al., 1995). Stuss et al. (1995) defines a schema as a “network of connected neurons that can be activated by sensory input, by other schemata, or by the Supervisory System” (p. 194). Drawing on the theoretical framework of the Supervisory Attentional System proposed by Norman and Shallice (1986), Stuss et al.’s (1995) expanded theory reflects the basic principle of other theories of executive abilities by distinguishing between routine and non-routine activities.

#### **1.1.4 Rolls’s theory of orbitofrontal functioning: Stimulus reward.**

Rolls (1990) theory of frontal lobe functioning is directed more at explaining the neural basis of emotion rather than cognition. Therefore, the emphasis is on the orbitofrontal cortex. According to Rolls (1990), emotions can be described as states that are produced by instrumental reinforcing stimuli. Some stimuli, such as pain and the taste of food are considered unlearned/primary reinforcers, while other stimuli become reinforced by learning due to their association with the primary reinforcers. Such stimuli are referred to as secondary reinforcers. Rolls (1990) termed this type of learning as stimulus-reinforcement association and it is thought to occur via the process of classical conditioning. A person learns from a young age to associate certain consequences (punishment or reward) with certain stimuli because of this stimulus-reinforcement association.

Rolls’ theory of orbitofrontal functioning argues that frontal lobe damage leads to a failure to react normally to non-reward in different contexts. Consequently, a person with frontal lobe damage will react inappropriately to stimuli (that was previously rewarded) if they are not rewarded.

Rolls, Everitt, and Roberts (1996) state that the orbitofrontal cortex of normal human beings is responsible for learning when a response to stimuli that was previously rewarded are no longer rewarded. This information will enable the individual to change his/her behaviour to adapt to the new situation (absence of reward) in his/her environment. A healthy orbitofrontal cortex therefore evaluates when a reward can be expected and enables an individual to alter his/her behaviour in a short time period in situations when previously rewarded behaviour is no longer rewarded. An individual with orbitofrontal damage will not be able to make such quick adjustments to their behaviour in this regard.

#### **1.1.5 Damasio's somatic marker hypothesis.**

Damasio's (1994) model of frontal lobe functioning attempts to explain the role of the frontal cortex within emotion and social behaviour. The case of Phineas P. Gage indicated that emotion and the neurology that is responsible for emotion is implicated within social behaviour. Because the damage sustained to Phineas Gage's brain was in the ventromedial prefrontal area, it appears that emotions and the accompanying neurological processes that leads to decisions on how to react and behave within social contexts depends on a healthy ventromedial system (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). Gage's cortices of the lateral aspects of the frontal lobes appeared to be intact. Damage to these areas are known for disrupting a person's ability to control attention, perform calculations, and shift appropriately from stimulus to stimulus (Damasio, 1994).

According to Damasio's theory, a person learns from a young age what is socially acceptable with the help of reward or punishment, that give rise to some somatic changes. In other words, social learning occurs due to associations between specific behaviour and punishment or reward. These associations are connected to subsequent somatic conditions within the brain and sensory and limbic association cortices. The somatic changes enable a person to finally experience the consequences of the rewards and punishment as emotions.

Inappropriate behaviour following frontal brain damage is a result of somatic conditions that were not activated or provoked through the learning process.

Damasio, Everitt, and Bishop (1996) proposes a somatic marker hypothesis to explain the emotional changes that occur following damage to the ventromedial cortices. The hypothesis proposes that a patient with ventromedial damage can comprehend the consequences and implications of inappropriate behaviour, but they are unable to identify these implications using a signal or sign that helps them to automatically distinguish between appropriate and inappropriate behaviour. Thus, patients' shortcomings who suffer from frontal lobe damage can be attributed to their inability to choose or implement behaviour that is deemed appropriate. Damasio et al. (1996) however caution against viewing the somatic marker hypothesis as a general theory for frontal lobe functioning, because of the frontal lobes' involvement in several separate although cooperative functions.

#### **1.1.6 Towards a decision on a theoretical framework of frontal lobe functioning.**

Luria's theory corresponds with Norman and Shallice's theory of supervisory attentional system because both theories view the frontal lobes as being responsible for the programming, regulation, and verification of human behaviour and activity. Furthermore, both theories emphasise the frontal lobes' inability to maintain adequate cognitive control when damage has occurred.

The theories mentioned above discussed the overall functioning of the intact frontal lobes. This researcher aligns himself with Chan et al. (2008) by opting for Luria (1973) understanding of the functioning of the frontal lobes, as well as Norman and Shallice's (1986) theory, which is an extension of Luria's theory. Norman and Shallice's supervisory attentional system also possess a monitoring function that includes planning ability, decision-making skills, and the ability to suppress a dominant response (Besnard et al., 2010), cognitive abilities

that are commonly collectively referred to as executive functions (Miyake et al., 2000; Burgess, 1997). These abilities serve as an important construct under investigation in the current study. The functioning of the frontal lobes is thus seen to programme, regulate, and verify human behaviour. This researcher's view is thus further validated by Stuss and Benson's (1986) and Donald Stuss's (2011) understanding of the functioning of the frontal lobes, which confirms and extends Norman and Shallice's understanding of the functioning of the frontal lobes. Stuss (2011) believes there is no central executive; instead, numerous domain general processes are discretely distributed across several frontal regions acting in concert to accomplish control. Stuss's (2011) research conducted over a period of 10 years further found that beyond these functions, there are two additional frontal anatomical or functional relationships called the ventromedial or orbitofrontal for emotional and behavioural regulation and the fronto-polar regions for integrative functions, or even metacognitive functions.

This researcher is therefore satisfied with the view that the frontal lobes functions in its broader sense is responsible for the programming, regulation, and verification of human behaviour. The idea that the prefrontal lobes serve as the seat of higher-level processes which are responsible for modulating lower-level ones is also supported by the researcher. Given that the environment in which a person finds him/herself generally requires a specific action, the researcher also agrees that the frontal lobes serve as integral structures in the formulation of potential action that is based on environmental demands. There are further numerous domain general processes discretely distributed across the frontal regions that act together in the control of selecting the appropriate action and the evaluation of that action in reaching a desired outcome.

## **1.2 Defining and Discussing Frontal Lobe Executive Functions**

The discussion will commence with a few initial remarks on the concept: Executive functions. In the field of neuroscience, the term “Executive Functions” is regarded as a relatively new term (Ardila, 2008). The direct ancestor of the concept of executive functions, Luria (1973), differentiated between three functional units in the human brain: (1) Arousal-motivation (limbic and reticular systems); (2) receiving, processing, and storing information (post-rolandic cortical regions); and (3) programming, controlling, and verifying activity, depending on the activity of the prefrontal cortex. This third unit according to Luria has an executive role (Ardila, 2008).

Drawing from Luria’s concept, McCullagh and Feinstein (2011) are of the impression that executive functioning is a concept that refers to a collection of higher-order abilities attributed to the domain of the frontal lobes and their projections. In neuropsychological literature, the terms executive function and frontal lobe function have developed side-by-side. These terms are used interchangeably, because of evidence of executive dysfunction in patients with frontal lobe damage (Benton, 1968; Luria (1973); Walsh, 1987; Welsh & Pennington, 1988). According to McCullagh and Feinstein (2011), the executive functions are responsible for engaging and directing various mental activities such as memory, attention, and motor behaviour during the engagement with daily challenges. From the beginning, these processes were associated with the frontal lobes (Benton, 1991) and damage incurred to the frontal lobes resulted in a range of symptoms previously referred to as frontal lobe syndrome (Benton, 1968).



The concept of executive function is generally defined as an umbrella term comprising a wide range of cognitive processes and behavioural competencies necessary for purposeful, independent, goal-directed, and self-serving behaviour (Anderson, 2001; Elliot, 2003; Funahashi, 2001; Gilbert & Burgess, 2008; Lezak et al., 2012; Miyake et al., 2000; Rabbitt, 1997; Stuss, 2011).

This definition is mostly derived from work done by scholars such as Lezak and colleagues, Elliot, Funahashi, and Stuss. Lezak et al. (2012) contributed to the definition by stressing that executive functions are capacities that enable a person to engage successfully in independent, purposeful, and self-serving behaviours. They propose that these behaviours may be conceptualized as having five components: (1) Volition, (2) planning and decision-making, (3) purposive action, (4) self-regulation, and (5) effective performance. Other authors concur with the aforementioned definition but also add several additional components of executive functioning. These include initiation, self-monitoring, attention as well as working memory (Anderson, 2001; Aupperle, Melrose, Stein, & Paulus, 2012; Delis, Kaplan, & Kramer, 2001). Stuss (2011) adds that inhibition and cognitive flexibility (or shifting) are terms that are also commonly linked to executive functioning. On her part, Rebecca Elliot (2003) contributed to the definition of executive functioning by describing executive functions as those functions that are involved in complex cognitions, such as solving novel problems, altering behaviour with regards to new information, and the creation of strategies or the sequencing of complex actions. Funahashi's (2001) contribution to the definition is the consideration of executive function as "a product of a coordinated operation of various processes to accomplish a particular goal in a flexible manner" (p. 147). Ardila (2008) and Mesulam (2002) define the meaning of the term executive function, by embedding in the term the concepts of mental flexibility, the ability to filter interferences, engagement in goal-directed behaviour, and the anticipation of behavioural consequences. According to the above-mentioned scholars, the

responsibility of executive control systems is therefore the flexible coordination of sub-processes to achieve a specific goal. If these systems break down, it will lead to behaviour becoming poorly controlled, disjointed, and disinhibited (Elliot, 2003). Thus, at the heart of the concept of executive functioning lie control, coordination, and goal orientation (Stuss, 2011).

Lezak et al. (2012) explain the difference between executive functions and cognitive functions in the following way: In executive functioning, the how or whether of human behaviour is contrasted to cognitive functioning's what or how much of human behaviour. They argue that as long as a person's executive functions remain intact, a person would be able to continue to be independent, constructively self-serving, and productive, despite sustaining considerable cognitive loss. It is however when the executive functions of a person are impaired, even if only to a small extent, that an individual may no longer be able to engage in satisfactory self-care, execute remunerative or valuable work independently, or be capable to uphold normal social relationships despite how well preserved the cognitive abilities are. Thus, to prevent any misunderstanding between executive functions and cognitive abilities (and disabilities) Sivan and Benton (1992) define cognitive abilities as:

“...the functional properties of the individual that are not directly observed but instead are inferred from his or her behaviour. Moreover, the specification of these abilities and the nature of their interrelationships are still unresolved questions. In any case, all behaviour (including neuropsychological test performances) is multiply determined. Thus, a patient's failure on a test of abstract reasoning may not be due to a specific impairment in conceptual thinking but to attention disorder, verbal ability, or inability to discriminate the stimuli of the test instead” (p. 39).

Impairment in cognitive abilities generally affect specific functions or functional areas, while executive functioning deficits tend to appear globally, affecting all aspects associated with human behaviour (Lezak et al., 2012). Baddeley decided in 1986 to group these behaviours into cognitive domains that included problems in planning, organisation of behaviours, disinhibition, perseveration, reduced fluency, and initiation. McCullagh and Feinstein (2011) identified the following aspects of frontal lobe executive functions that can potentially be impaired following brain damage (Table 0.1).

*Table 0.1. Aspects of frontal lobe executive functions that potentially can be impaired following brain damage*

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**Various executive functioning aspects affected by brain damage.**

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- Goal establishment, planning, and anticipation of consequences.
- Initiation and inhibition of responses; temporal sequencing of behaviour.
- Generation of novel response alternatives (vs. perseverative or stereotyped responses).
- Mental flexibility/ease of mental and behavioural switching.
- Transcending of the immediately salient aspects of a situation (vs. stimulus-bound behaviour or environmental dependency).
- Conceptual/inferential reasoning; problem-solving in novel or complex circumstances.
- Decision-making.
- Working memory.
- Executive attentional and memory processes.
- Self-monitoring and self-regulation, including emotional responses.
- Social adaptive functioning: perspective-taking, use of social feedback, adherence to social norms.

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*Note.* Reprinted from “Cognitive changes” by S. McCullagh and A. Feinstein, 2011, In J.M. Silver, T.W. McAllister, & S.C. Yudofsky, (Eds.), *Textbook of traumatic brain injury*, p. 282. Washington, DC, United States of America: American Psychiatric Publishers. Copyright 2011 by the American Psychiatric Association.

Fuster (2001; 2002a) and Happaney, Zelazo, and Stuss (2004) estimate that there are two different, but closely related types of prefrontal lobe activities. They identified these as metacognitive executive functions and emotional/motivational executive functions. The first includes verbal reasoning, problem-solving, planning, sequencing, the ability to sustain

attention, resistance to interference, utilization of feedback, multitasking, cognitive flexibility, and the ability to deal with novelty (Burgess et al., 2000; Damasio, 1995; Grafman & Litvan, 1999; Stuss et al., 2005). These functions have also been referred to as the “cold” components of executive functioning, because they do not involve emotional arousal and are somewhat mechanistic or logically based (Grafman & Litvan, 1999).

The second type of frontal lobe activity, the emotional/motivational executive functions, is responsible for coordinating cognition and emotion. This entails the ability to fulfil basic social impulses following socially adequate strategies, making use of the limbic system. This involves the inhibition of selfish or unsociable basic impulses, known as the inhibitory control of behaviour (Miller & Wang, 2006), although not necessarily arriving at the best conceptual solution. Because these functions involve more emotional, belief, or desire aspects such as the experience of reward and punishment, regulation of one’s own social behaviour, and decision-making involving emotional and personal interpretation, they are referred to as the “hot” components of executive functioning (Brock, Rimm-Kaufman, Nathanson, & Grimm, 2009; Chan et al., 2008; Simon, Stenstrom, & Read, 2015).

### **1.2.1 Hot and cold components of executive functions.**

It was during the 1960’s that Walter Mischel discovered the two systems, known as the hot and cold components of executive functioning. He made this discovery while conducting experiments on delayed gratification amongst children. During an experiment, known as The Marshmallow Test, Mischel presented children with a dilemma. Eat the marshmallow immediately, or wait for up to 20 minutes while sitting and waiting alone in a room with the reward of receiving two marshmallows. If the child manages to wait the full period, he or she would be rewarded with not only an extra marshmallow, but also an additional one. The mental struggles the children endured in their attempts to resist that what was in front of them, was both fascinating and comic to observe. Mischel continued to follow smaller

cohorts into their twenties, thirties, and forties. He found interesting correlations between the child's ability to delay gratification and various indicators of success and well-being. He found that the ability of a child to delay gratification reflected in the child achieving higher scores on intelligence test batteries. He further found that these children could exhibit better attention span, as well as having a higher sense of self-worth. Moreover, they were able to portray an improved ability to pursue goals effectively and cope better with stress. They also displayed better trust in their own judgement and even seemed to possess the ability to establish healthier relationships (Buttler-Bowdon, 2017; Mischel, Shoda, & Rodriguez, 1989). According to Buttler-Bowdon (2017), the children in Mischel's experiments successfully managed to control themselves by displaying the following three important elements of executive functioning:

1. *Recalling* the chosen goal and reminding themselves of the contingency ("If I eat one now, I won't get the two later).
2. *Monitoring* their progress toward the goal, and self-correcting by refocusing on it or using temptation-reducing techniques; and
3. *Inhibiting* impulsive responses that would stop them from achieving their goal.

Walter Mischel's research demonstrated that individuals who exhibit the above-mentioned elements could keep instructions in mind, control their impulses, and focus their attention on selected goals. In the absence of such executive functions, they would struggle to follow instructions, and will most probably end up in potentially difficult situations because they will be unable to foresee the consequences of their actions.

Thus, Walter Mischel identified an important aspect of two systems at work in the frontal lobes: The hot emotional, reflexive, and unconscious system, and the cold thinking, reflective, and rational system (Fonseca et al., 2012). As mentioned above, the limbic system drives hot thinking. This system is responsible for generating fear responses, sexual urges, and

desire for food. The amygdala, which forms part of the limbic system, does not hesitate to react and consider long-term consequences of its actions. The limbic system is more powerful than the deliberate, rational part of the brain—the prefrontal cortex (PFC). The hot system engages most easily when an individual is experiencing stress, because of the role the amygdala plays in ensuring survival. During this process the cold system, which is associated with rational and strategic thinking, sinks to the background. These two systems continuously and seamlessly engage in a reciprocal relationship. As the one becomes aroused, the other quiets down with the result that during occasions when we need creative problem-solving most, we tend to lose the ability to do so. The result thereof is thus, that stress and anxiety over a long period leads to poor decisions, brought about by irrational thinking, strong emotions, and the inability to remember what is important (Mischel, 2014). Research done by Goel, Grafman, Tajik, Gana, and Danto (1997), as well as Grafman et al. (1996) and Green, Kern, Braff, and Mintz (2000) confirms that impairments in either the cold or the hot components of executive functions have adverse effects on people's daily lives. Activities that are affected includes common abilities such as the ability to work and attend school, function independently at home, and even the development and maintenance of healthy social relations.

The term 'executive function' is thus employed as an umbrella for numerous complex cognitive processes and sub-processes. A point of vigorous discussion amongst researcher is whether executive functions, which is responsible for controlling and organising other mental processes, can be divided into recognisable sub-processes. In other words, can the so-called supervisory attentional system (SAS) be fractionated (Gilbert & Burgess, 2008)? The above attempt to define executive functions turned to a list of examples of hot and cold thinking processes, which reflects the fact that executive functioning does not constitute a unitary concept. However, some disagreement does exist among researchers about the questions of unity or diversity (non-unitary) of executive functions (Baddeley, 1998; De Frias, Dixon, &

Strauss, 2006; Duncan, Emslie, Williams, Johnson, & Freer, 1996; Kimberg, D'Esposito, & Farah, 1997; Parkin & Java, 1999). For some researchers who propagate the unitary aspect, behaviour inhibition is a possible element or single factor accountable for successful performance in various executive tests, or in combination with working memory (Pennington & Ozonoff, 1996). For Salthouse (1996; 2005) reasoning ability and perceptual speed represents the underlying factors related to all executive functions. On the other hand, authors such as Godefroy, Cabaret, Petit-Chenal, Pruvo, and Rousseaux (1999) challenge the existence of a unitary factor. They argue that some frontal lobe patients perform well on some tests intending to assess executive abilities, but not on others. Correlations among different executive tests are frequently moderate or low, and many times lacking statistical significance (Fisk & Sharp, 2004; Friedman et al., 2006; Lehto, 1996; Salthouse, Atkinson, & Berish, 2003). Gilbert and Burgess (2008) supports this argument because according to them, it seems unlikely that executive function refers to a single, undifferentiated cognitive process. They came to this conclusion through behavioural studies that confirmed that a positive correlation exists between participants' scores on different executive functioning tests tend to be lower (generally  $r < 0.4$ ) than correlations with non-executive tests. Gilbert and Burgess (2008) reports other sources of supporting their stance. These include evidence that damage to different regions of the frontal lobes exerts differential effects on various executive functioning tests. Furthermore, neuroimaging studies showed that the engagement of different aspects of executive function are accompanied by the changes in blood flow within the different parts of the frontal lobes (Robbins, 2007).

A third group of researchers chose an intermediate position. Miyake et al. (2000) studied the three affirmed aspects of executive functions, namely: (1) Shifting: An individual's ability to shift attention between different sub-tasks or different elements of the same task; (2) Updating: The ability to evaluate incoming information and revising the existing contents of

working memory by erasing irrelevant information and integrating more recent relevant information; and (3) Inhibition: The individual's ability to withhold responses which are inappropriate to the situation. According to Collins and Koechlin (2012), as well as Lunt et al. (2012), higher-order executive functions such as planning, reasoning, and problem-solving are built from these three affirmed aspects of executive functions. Miyake et al. (2000) found that although these three aspects are clearly distinguishable, they do share some underlying commonality. Their studies led them to believe that executive functions are separable but moderately correlated constructs, therefore suggesting both unitary and non-unitary components of the executive system. A schematic representation of this pattern of unity and diversity can be viewed in Miyake et al. (2000).

In 2012, Miyake and Friedman published an improved unitary and non-unitary framework schema (Figure 0.1) in which each individual executive functioning ability (like updating for example) can be broken down into what is typical across all three affirmed aspects of executive functions, or unity (termed common executive functions), and that what is unique to that specific ability, or diversity (e.g., updating-specific ability). The focus of the unity/diversity framework proposed by Miyake and Friedman (2012) is largely directed towards the elements found on the right side of the equation. Thus, instead of focusing on the elements that demonstrate mixed influences from both unity and diversity (updating, shifting, and inhibition) on the left side of the equation, Miyake and Friedman (2012) shifts the attention towards those specific elements that are more clearly associated with underlying cognitive processes (common executive functions, updating specific, and shifting-specific abilities) and attempt to determine their underpinnings.



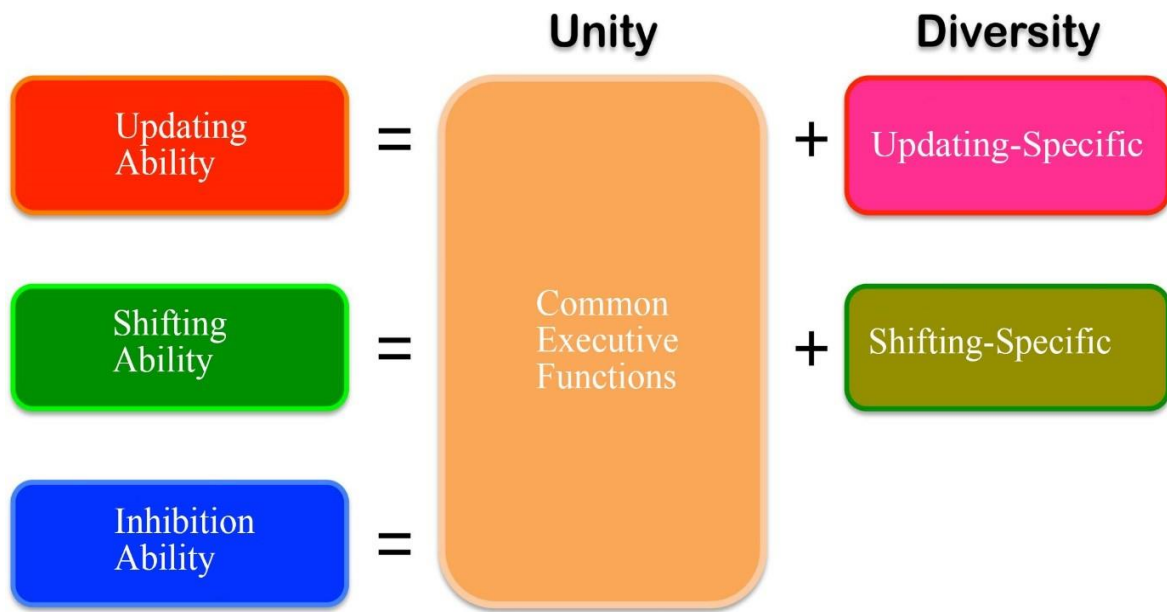


Figure 0.1. A schematic representation of the unity and diversity of the three executive functions.

Each of the three affirmed aspects of executive functions (e.g., inhibition, shifting, updating) is indeed a combination of what is common to all three executive functions (common executive function) and what is specific to that executive function (e.g., updating-specific). The inhibition-specific component is absent in this figure, because Miyake and Friedman (2012) repeatedly found that, once the unity (common executive function) is accounted for, there is no unique variance left for the inhibition-specific factor. Reprinted from “The nature and organization of individual differences in executive functions: Four general conclusions,” by A. Miyake and N.P. Friedman, 2012, *Current Directions in Psychological Sciences*, 21(1), p. 11. Copyright 2012 by The Authors.

According to Miyake and Friedman’s (2012) current perspective, common executive function is about an individual’s ability to actively maintain task goals and goal-related information and to use this information to effectively bias lower-level processing. They argue that this basic skill is essential for all three affirmed aspects of executive functions and has been proposed as a crucial requirement for response inhibition (Munakata et al., 2011). On the other hand, Miyake and Friedman (2012) propose that the shifting-specific component primarily reflects flexibility. That is, the ease in which an individual transition to a new task-set representation. They are however still less certain about what exactly the updating

component taps. Nonetheless, effective gating of information and controlled retrieval from long-term memory have been identified as two possible mechanisms.

Friedman et al. (2008) argue that the discovery of executive functions as consisting of both unitary and diverse processes holds essential methodological and theoretical implications for the various domains of psychological research. The acknowledgement of executive control as a multicomponent construct has contributed to the increased specificity regarding the nature of executive involvement in numerous cognitive, neuropsychological, and clinical constructs. For example, within the cognitive domain, three complex neuropsychological and cognitive measures that are generally used to examine general executive control were in fact found to be differentially related to updating, shifting, and inhibition among young adults. These measures include the Wisconsin Card Sorting Test (most closely related to shifting), random number generation (related to inhibiting and updating), and the Tower of Hanoi (related to inhibiting).

In their multifaceted twin study of the three types of executive functions (inhibiting dominant responses, updating working memory representations, and shifting between task sets), Friedman et al. (2008) assessed why individuals differ in these executive control abilities and why these abilities are correlated but separable from a behavioural genetic perspective. By measuring these three specific types of executive functions as latent variables, Friedman et al. (2008) found that in normal young adults, the individual differences regarding inhibiting, shifting, and updating abilities appear to be almost entirely genetic in origin. According to the results obtained, executive functions seem to be correlated, because they are influenced by a highly heritable common factor that stretches beyond general intelligence and perceptual speed. Moreover, they also appear separable because of additional genetic influences unique to executive functions. Therefore, Friedman et al. (2008) suggest that executive functions' unity and diversity are due primarily to genetic influences that operate at both the general (on all three executive functions) and specific (updating and shifting) levels. This combination of

general and specific genetic influences places executive functions among the most heritable psychological traits (Friedman et al., 2008). However, the argument of genetic versus cultural and/or environmental explanations for differences in intellectual abilities is the basis of much controversy and debate and this issue falls however outside the scope of this study.

Even though the executive system was initially regarded as a unitary system, it is now recognized that the executive system is derived from a variety of skills that collectively make up executive functions (Kroesbergen, Van Luit, Van Lieshout, Van Loosbroek, & Van de Rijt, 2009). Still under debate is the relationship between executive functions and intelligence. Studies exist that reject such a relationship due to findings that indicate that patients with frontal lobe damage have often been reported to have dysexecutive symptoms, but intact intelligence (Crinella & Yu, 1999). In contrast, other studies reported such a relationship between executive functioning and intelligence (Brydges, Reid, Fox, & Anderson, 2012; Duncan et al., 1996; Friedman et al., 2006). Ferrer, Shaywitz, Holahan, Marchione, and Shaywitz (2010), refers to fluid intelligence as the ability to reason, solve problems, and identify patterns or relationships between items. It comprises of both inductive and deductive logical reasoning, and includes the ability to identify abstract relations underlying analogies. Fluid intelligence is also synonymous with sub-processes of higher-order executive functions such as reasoning and problem-solving (Figure 0.2). It is therefore without surprise that measuring instruments aimed at assessing fluid intelligence (for example, the Raven's Progressive Matrices (Raven, 2000)) have a strong correlation with independent measures of executive functions (Conway, Kane, & Engle, 2003; Duncan et al., 2008; Kane & Engle, 2002; Roca et al., 2010). In addition, Friedman et al. (2006) also found that both fluid and crystalized intelligence are especially related to updating, but not inhibiting or shifting.

It seems to this researcher that the research determining whether executive functions can be divided into recognisable sub-processes is still ongoing. Also encapsulated in the definition of executive function is the concept of morality, ethical behaviours, self-awareness, and the notion that the frontal lobes serves as the manager and programmer of the human psyche (Anderson et al., 1999; Damasio, 1994; Luria (1973); Moll, Zahn, de Oliveira-Souza, Krueger, & Grafman, 2005). Thus, in a broad sense, executive functions enable us to exercise intentional, conscious control over our actions, impulses, thoughts, and emotions. Executive functions allow us to cool down our urges while simultaneously providing conscious attention to our goals assisting us to achieve them.

As stated above, executive functioning can be defined in several ways. The exact nature of the processes that fall under the umbrella of executive functioning is however still not fully understood. Scholars are still debating an acceptable definition of executive functioning. However, there is evidence at this moment pointing in the direction that frontal lobe executive functioning is unlikely to be a single, undifferentiated cognitive process. This researcher chooses to follow this direction indicated by Anderson (2001), Delis et al. (2001), Elliot (2003), Gilbert and Burgess (2008), Godefroy et al. (1999), Kroesbergen et al. (2009), Lezak et al. (2012), Miyake et al. (2000), Stuss (2011), and Suchy (2009). These researchers' understanding of executive functioning, which I am comfortable to align myself with, is incorporated in the following definition proposed by Suchy (2009):

Executive functioning (EF) is a multifaceted neuropsychological construct that can be defined as (1) forming, (2) maintaining, and (3) shifting mental sets, corresponding to the abilities to (1) reason and generate goals and plans, (2) maintain focus and motivation to follow through with goals and plans, and (3) flexibly alter goals and plans in response to changing contingencies” (p. 106).

This chosen definition of executive functioning falls well within the ambit of Luria’s classical view of frontal lobe functioning, as well as Norman and Shallice’s supervisory attentional system (SAS). Despite the abundance of cognitive abilities and processes integrated in the definitions of executive functioning, it seems that there is some agreement between authors regarding the key attributes of executive functions (Anderson, 2001; Aupperle et al., 2012; Barkley, 2001; Blakemore & Choudhury, 2006; Diamond, 2013; Fuster, 2008; Garner, 2009; Happé, Booth, Charlton, & Hughes, 2006; Lezak et al., 2012; Meltzer, 2007; Rabinovici, Stephens, & Possin, 2015; Spinella, 2005; Stuss, 2011). Most of the authors agree that the key attributes consist of the following: Volition, planning and decision-making, inhibitory control, working memory, cognitive flexibility, purposive action, self-regulation, effective performance, and attention. A synopsis of the nine key attributes and their respective authors are also provided in Table 0.2 below.

*Table 0.2. Synopsis of the key attributes of executive functions*

<b>Key attributes of executive functioning</b>	<b>Respective Authors</b>
<i>Volition</i> : The ability to engage in intentional behaviour.	<ul style="list-style-type: none"> <li>– Baddeley (1986)</li> <li>– Garner (2009)</li> <li>– Lezak et al. (2012)</li> <li>– Stuss (2011)</li> </ul>
<i>Planning and decision-making</i> : The ability to set and maintain goals.	<ul style="list-style-type: none"> <li>– Anderson (2001)</li> <li>– Aupperle, Melrose, et al. (2012)</li> <li>– Baddeley (1986)</li> <li>– Diamond (2013)</li> <li>– Fuster (2008)</li> <li>– Garner (2009)</li> <li>– Lezak et al. (2012)</li> <li>– Stuss (2011)</li> </ul>
<i>Inhibitory control</i> : The ability to inhibit a response.	<ul style="list-style-type: none"> <li>– Aupperle, Melrose, et al. (2012)</li> <li>– Baddeley (1986)</li> <li>– Diamond (2013)</li> <li>– Fuster (2008)</li> <li>– Garner (2009)</li> <li>– Rabinovici et al. (2015)</li> </ul>
<i>Working memory</i> : The ability to temporarily store information used	<ul style="list-style-type: none"> <li>– Aupperle, Melrose, et al. (2012)</li> <li>– Diamond (2013)</li> </ul>

to perform a wide variety of cognitive tasks.	<ul style="list-style-type: none"> <li>– Fuster (2008)</li> <li>– Rabinovici et al. (2015)</li> <li>– Stuss and Alexander (2000)</li> </ul>
<i>Cognitive flexibility</i> : The ability to alter one’s behaviour in response to a changing environment.	<ul style="list-style-type: none"> <li>– Anderson (2001)</li> <li>– Aupperle, Melrose, et al. (2012)</li> <li>– Diamond (2013)</li> <li>– Rabinovici et al. (2015)</li> </ul>
<i>Purposive action</i> : The ability to change an intention or plan into a productive, self-serving activity.	<ul style="list-style-type: none"> <li>– Baddeley (1986)</li> <li>– Fuster (2008)</li> <li>– Lezak et al. (2012)</li> <li>– Stuss (2011)</li> </ul>
<i>Self-regulation</i> : The ability to exert control over thoughts, feelings, and actions.	<ul style="list-style-type: none"> <li>– Lezak et al. (2012)</li> <li>– Stuss (2011)</li> </ul>
<i>Effective performance</i> : The ability to self-regulate, -monitor, and -correct.	<ul style="list-style-type: none"> <li>– Anderson (2001)</li> <li>– Fuster (2008)</li> <li>– Lezak et al. (2012)</li> <li>– Stuss (2011)</li> <li>– Stuss and Alexander (2000)</li> </ul>
<i>Attention</i> : The ability to notice rare or unexpected stimuli over an extended period.	<ul style="list-style-type: none"> <li>– Aupperle, Melrose, et al. (2012)</li> <li>– Diamond (2013)</li> <li>– Fuster (2008)</li> <li>– Stuss and Alexander (2000)</li> </ul>

Although agreement exist among scholars regarding the key attributes of frontal lobe executive functioning, there do appear to be a debate about the similarities and differences amongst the respective executive functions and their related terminology. The discussion will now focus on the terms, self-regulation, effortful control, and executive attention to address the similarities and disparities.

### **1.2.2 Similarities and disparities among executive functions and their related terms.**

Eisenberg, Hofer, and Vaughan (2007) as well as Liew (2012) view self-regulation as those processes that allow individuals to maintain the highest levels of emotional, motivational, and cognitive arousal. Self-regulation primarily is the control and regulation that an individual has over his/her emotions (Eisenberg, Spinrad, & Eggum, 2010; Mischel & Ayduk, 2002), and overlaps considerably with inhibitory control (Figure 0.2). In the past, researchers who set out

to investigate executive functions predominantly concentrated on thoughts, attention, and actions, therefore primarily focussing on the lateral prefrontal cortex, in particular the dorso- and ventrolateral prefrontal regions.

On the other hand, those researchers who attempted to investigate self-regulation predominantly focussed their attention towards studying emotions and therefore were more interested towards the medial prefrontal cortex, specifically the orbitofrontal regions and the characteristics of the parasympathetic nervous system (Diamond, 2013). Researchers who studied executive functions viewed emotions as problems that necessitates inhibition, while researchers of self-regulation emphasised the relevance of motivation and interest as essential emotional responses for goal attainment (Blair & Diamond, 2008).

According to Rothbart (2007), effortful control constitutes as an aspect of temperament. It refers to an individual's ability to select a course of action under conflicting conditions as well as the ability to plan and detect errors. Effortful control is also associated with essential developmental outcomes, which include the development of behavioural problems and conscience. In addition, imaging studies also demonstrated that effortful control is linked to the executive attention network (Rothbart, 2007). Effortful control is an inherent predisposition to exert self-regulation effortlessly, such as easily slowing down or lowering one's voice, or maybe even being overregulated, such as lacking spontaneity, versus experiencing self-regulation as difficult or unnatural (Diamond, 2013).

Executive attention is the top-down regulation of attention (Posner & DiGirolamo, 1998). The attempt to define the exact functions or mechanisms of executive attention (or executive control) has not gone without difficulty. A great deal of confusion is caused by the excessive use of the term executive attention to apply to such abilities as working memory (Engle, 2002) and response inhibition or the solving of response conflicts (Jones, Rothbart, &

Posner, 2003). According to Norman and Shallice (1986) as well as Posner and DiGirolamo (1998), executive attention is essential during tasks that require planning, detection of errors, novelty, complicated processing, or conflict resolution. Executive attention is generally assessed using measures of selective attention such as the Flanker Task (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Rueda, Posner, & Rothbart, 2005).

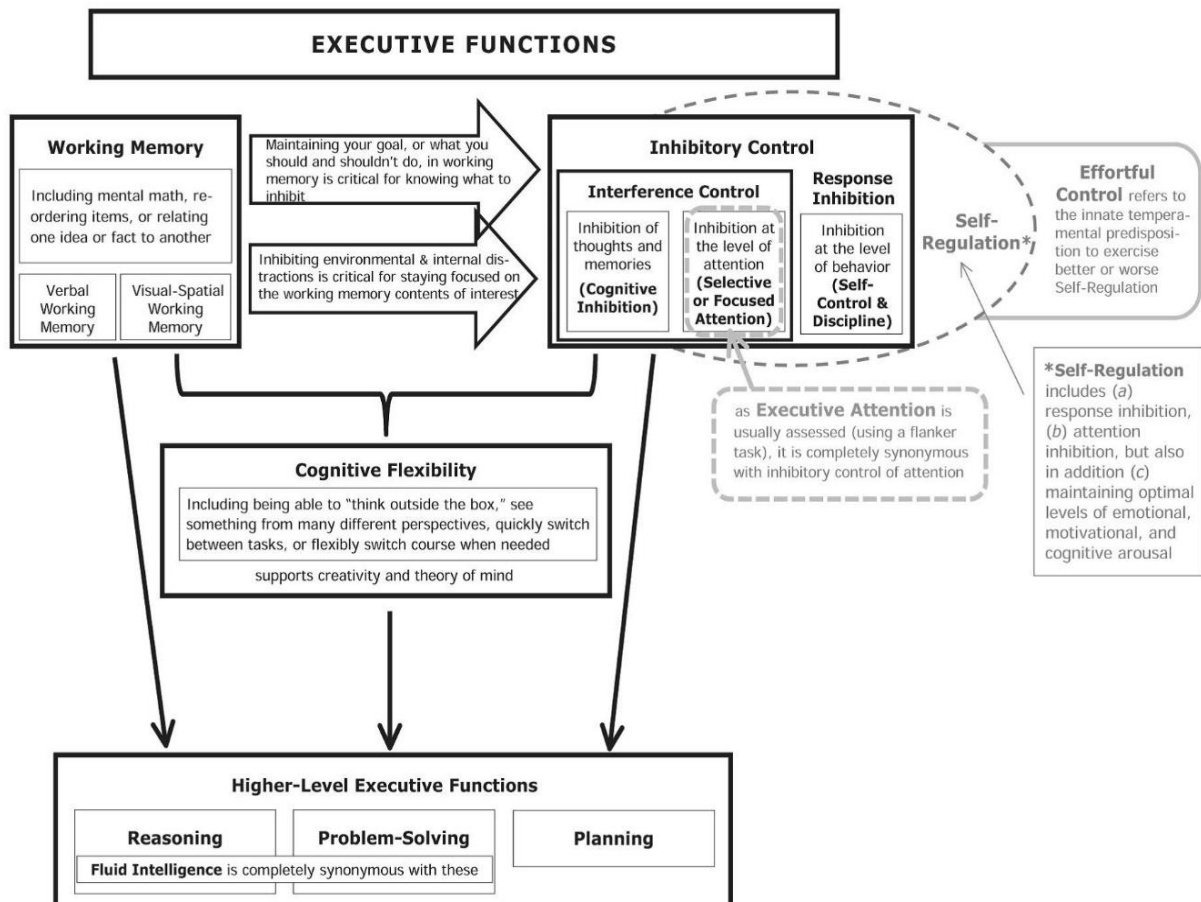


Figure 0.2. Executive functions and their related terms

Adapted from "Executive functions," by A. Diamond, 2013, *Annual Review of Psychology*, 64(1), p. 41. Copyright 2013 by Annual Reviews.

In the following paragraphs, the researcher will attempt, with the aid of Rabbitt (1997), to differentiate between executive and non-executive functions. This distinction will clarify performances, skills, and behaviours that are characteristic of executive function from those that are not.



### **1.2.3 Differentiating between executive and non-executive functions.**

Rabbitt (1997) generated an exhaustive list of distinctions. It is however sufficient to say that Rabbitt distinguishes between executive control and non-executive control by referring to the former as necessary to deal with new tasks that require the formulation of goals, planning, and the choice between alternative series of behaviour needed to reach this goal. Following this process, the individual needs to compare these plans in respect of their relative likelihood of success and efficiency in reaching the chosen goal. Thereafter, the selected plan must be initiated and implemented, while continuously amending the plan where necessary until it is successful or eminent failure is acknowledged.

It is important to keep in mind that executively controlled behaviour is more complex than non-executive behaviour. Rabbitt (1997) recognizes that even choices between very complex behaviour sequences as mentioned above, (e.g., the formulation and implementation of plans), can be executed “automatically” under non-executive control. An example he provides is that of a skilled driver negotiating complex traffic while engaging in non-trivial discussions with a passenger.

Rabbitt (1997) is of the opinion that the key distinction between executive and non-executive control of behaviour becomes visible during situations in which a person must for the first time, recognise, evaluate, and choose between various alternative options (executive control of behaviour) and situations in which a single effective behaviour sequence, which has been previously identified, and initiated by practice, is executed without the need to propose and evaluate alternatives. In contrast to executive behaviour, non-executive behaviour tends to be initiated by, and appear to carry on automatically in response to changes in the environment. Even very complex non-executive behaviour is externally driven and controlled by series of environmental events or by already learned plans preserved in long-term memory. In contrast, executive behaviours can be brought about and regulated separately from environmental input

and can maintain the adaptive flexibility to adjust plans as and when environmental demands adjust.

The primary purpose of the executive system as a process or set of processes is thus to facilitate adaption to novel situations (Karnath, Wallesch, & Zimmermann, 1991; Sirigu et al., 1995). It is therefore clear that the executive system functions through the modulation and control of the more fundamental or routine cognitive abilities of individuals (Duncan, 1986; Fuster, 2008; Luria (1973); Shallice, 1982). These routine cognitive abilities are commonly known as those abilities which have been overlearned by becoming habits, and therefore can contain for example motor, reading, or language skills and even semantic memory (Burgess & Shallice, 1996). However, real-life necessitates adaptation of these skills because no two situations are similar.

Broadening Rabbitt's (1997) perspective, Norman and Shallice (1986) present five types of situations where routine, automatic activation of behaviour would not be sufficient for optimal performance, hence requiring the activation and implementation of a healthy supervisory attentional system:

1. Situations that involve planning or decision-making.
2. Situations that involve error correction or troubleshooting.
3. Those in which responses are not well-learned or contain novel sequences of actions.
4. Situations that are potentially dangerous or technically difficult.
5. Situations that require overcoming engrained habits or the resistance to temptation.

#### **1.2.4 The value of executive functioning.**

Executive functioning is required during situations when changes in rules suddenly occur such as simulated in the case of the Wisconsin Card Sorting Task or its variants. Executive functioning allows a person to realise, from feedback that the card sorting rules have changed, necessitating different formulations and the testing of new plans until success is achieved. Lezak et al. (2012) and Martin (2006) emphasises that the inhibition or suppression of previously used rules is part of executive functioning enabling an individual to project, select, and utilize alternative outcomes.

Burgess and Shallice (1996) explain that deliberate retrieval of structured information from memory, to answer a question or to elaborate a partial description (executive functioning), may involve the conscious formation of well-articulated memory search-strategies. Executive functions are essential to launch new series of behaviour and to disturb other ongoing series of responses to do so. Consequently, executive functions enable a person to suppress, inhibit, or replace a habitual response with task-appropriate responses. In the same way, executive functions check involuntary perseveration by at the proper time switching attention to new sources of information. In addition, executive functions play a significant role in preventing behaviour that is improper in specific contexts (Rabbitt, 1997).

Within intact executive functioning, lies the ability to execute two-tasks simultaneously. This ability is a key indicator of an individual's planning and control capacity. When attending to both tasks, the strategic allocation of attention and the controlled synchronisation of responses are required to complete both (Rabbitt, 1997).

Executive functioning is required when carrying out plans to deal with unique demands. They are needed when monitoring performance to detect and correct errors or to alter plans once it becomes clear that they are going to fail. They help to recognise novel

opportunities and obtain goals that are more desirable. When new and more desirable goals are identified, executive functions again assist with the formulation and selection of the most appropriate goal. Thereafter initiating the execution of new plans to reach the changed goal. Monitoring of complex series of non-executive behaviours do not seem to take place at this level. Executive monitoring is seen only as a higher-level activity that, not only detects actions not being completed as planned, but also predicts when failure is imminent, or when a more attractive goal has gone unnoticed, unless a plan, which is being carried out precisely as intended, can be rapidly changed.

Robertson, Manly, Andrade, Baddeley, and Yiend (1997) assert that executive functions are also responsible for allowing attention to be continuously sustained for over long periods. This enables a person to predict the outcomes of long, complex sequences of events. It also seems that behaviour that is controlled by executive functioning is accessible to consciousness in contrast to behaviours, which is controlled by non-executive functioning.

Executive functions are therefore important top-down mental processes that are essential during instances when individuals need to concentrate and pay attention, especially during times when relying on instinct, automatic behaviour, or intuition are not recommended (Espy, 2004; Miller & Cohen, 2001). Engaging in executive functioning processes takes effort. Humans are more prone to succumb to temptation than to resist it. It takes less effort to continue with what one is doing, than to alter one's actions even if the change in behaviour promises better rewards. We feel more comfortable doing the "same old" than discovering something new. Executive functions are important skills for maintaining good mental and physical health, obtaining success in school, and throughout life in general, and is essential for psychological, cognitive, and social development (Table 0.3).

Table 0.3. Aspects in life in which executive functions are important

Aspects of life	Executive functions are impaired in various mental disorders, including:	References
Mental Health	Addictions	– Baler and Volkow (2006)
	Attention Deficit Hyperactivity Disorder (ADHD)	– Diamond (2005) – Lui and Tannock (2007)
	Conduct Disorder	– Fairchild et al. (2009)
	Depression	– Taylor Tavares et al. (2007)
	Obsessive-Compulsive Disorder (OCD)	– Penadés et al. (2007)
	Post-traumatic Stress Disorder (PTSD)	– Olf, Polak, Witteveen, and Denys (2014) – Schoeman, Carey, and Seedat (2009)
	Schizophrenia	– Barch (2005) – Crescioni et al. (2011)
Physical health	Poor executive functioning is linked to obesity, overeating, substance abuse, and poor treatment adherence.	– Miller, Barnes, and Beaver (2011) – Riggs, Spruijt-Metz, Sakuma, Chou, and Pentz (2010)
	Individuals with enhanced executive functioning enjoy a better quality of life.	– Brown and Landgraf (2010) – Davis, Marra, Najafzadeh, and Liu-Ambrose (2010)
School readiness	Executive functions are more important for school readiness than are IQ or entry-level reading or mathematical ability.	– Blair and Razza (2007) – Morrison, Ponitz, and McClelland (2010)
School success	Executive functions predict both mathematical and reading competence throughout the school years.	– Borella, Carretti, and Pelegrina (2010) – Duncan et al. (2007) – Gathercole, Pickering, Knight, and Stegmann (2004)
Career success	Poor executive functioning influence job productivity and lead to difficulty in finding and keeping a job.	– Bailey (2007)
Marital harmony	A partner with impaired executive functioning can be more difficult to get along with, less dependable,	– Eakin et al. (2004)

	and/or more inclined to act on impulse.	
General public safety	Poor executive functioning may lead to social problems such as crime, reckless behaviour, violence, and emotional outbursts.	<ul style="list-style-type: none"> <li>– Broidy et al. (2003)</li> <li>– Denson, Pedersen, Friese, Hahm, and Roberts (2011)</li> </ul>

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*Note.* Adapted from “Executive functions” by A. Diamond, 2013, *Annual Review of Psychology*, 64(1), p. 136. Copyright 2013 by Annual Reviews.

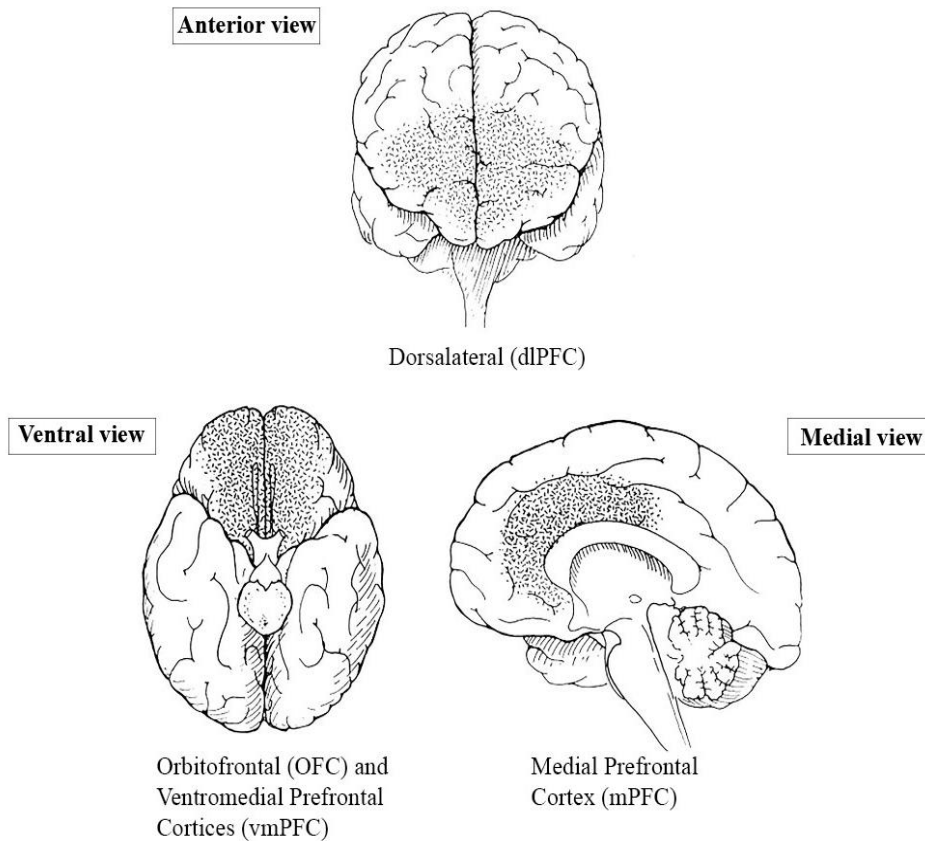
An overview of the neuro-anatomy of executive functions will follow next, with specific reference to the general organisation of the neuro-circuitry of the frontal lobe subdivisions.

### **1.3 Neuro-anatomy of Executive Functions**

A common belief concerning the neuroanatomic organisation of the executive functions is that they are seated solely in the prefrontal region of the brain (Gioia, Isquith, Guy, & Kenworthy, 2000; Suchy, 2009). This is however an oversimplification of the complex organisation of the brain. Ardila (2008), in line with Luria (1973), argues that the prefrontal cortex plays a primary role in the controlling and monitoring of the activity of the frontal lobes. Elliot (2003) believes although the prefrontal cortex plays an integral monitoring role in executive functions, various other brain regions are also involved. Damage to the frontal lobes may result in significant dysfunction of various executive sub-domains, which do not simply reside in the frontal lobes (Asarnow, Satz, Light, Lewis, & Neumann, 1991; Eslinger & Grattan, 1991; Fletcher, Ewing-Cobbs, Miner, Levin, & Eisenberg, 1990). Ardila (2008) further states that intact frontal processes, although not deemed synonymous with executive functioning, do form an integral part of executive functions. Filley (2000) mentions that research conducted amongst individuals with focal brain lesions provided most of the information about the localisation of executive function. However, efforts to localize executive functioning to distinct frontal regions have been inconclusive over the years (Ardila, 2008).

The emerging view supported by Ardila (2008) is that dynamic and flexible networks are responsible for mediating executive functions. Neuroimaging results further implicate posterior, cortical, and subcortical regions in executive functioning (Bonelli & Cummings, 2007; Seger, 2006). Various neuropsychological studies have further indicated that the frontal lobes, specifically the prefrontal areas are implicated in executive functions (Aupperle, Allard, et al., 2012; Elliot, 2003; Filley, 2000; Lezak et al., 2012; Fuster, 2002b; Stuss & Levine, 2002). Even though executive function is generally mediated by prefrontal cortical functioning, these complex behaviours are however modulated by dopaminergic, noradrenergic, serotonergic, and cholinergic inputs. The capacity of these neurotransmitter systems to modulate impulse control, response inhibition, attention, working memory, cognitive flexibility, planning, judgment, and decision-making make it possible to alter cognitive behaviour in response to changes in the environment. Given their critical role in regulating executive function, it is not surprising to find that disturbance in these systems may have a significant effect on executive functioning (Logue & Gould, 2014).

An important theoretical framework that illustrates the localisation of executive functions are the identification of three neuro-anatomical sub-divisions within the prefrontal lobes. These sub-divisions include: (1) The ventral prefrontal cortex (VPFC), which includes the ventral medial prefrontal cortex (vmPFC) and the orbital frontal cortex (OFC); (2) the dorsolateral prefrontal cortex (dlPFC); and (3) the superior medial prefrontal cortex (smPFC) Figure 0.3. The sub-divisions of the frontal lobes (Figure 0.3). In the following paragraphs, the focus will be on the neuro-anatomical topography of the three sub-divisions of the prefrontal cortices responsible for mediating executive functions and their respective neuro-circuitry.



*Figure 0.3. The sub-divisions of the frontal lobes*

Adapted from “Clinical neurology and executive dysfunction,” by C.M. Filley, 2000, *Seminars in Speech and Language*, 21(2), p. 96. Copyright 2000 by Thieme Medical Publishers Inc.

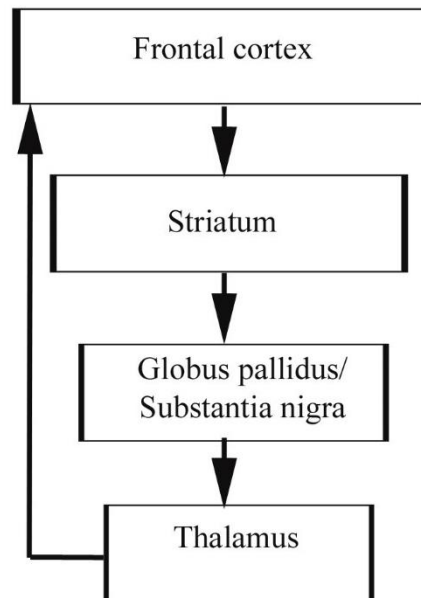
The frontal lobes play an important role in directing human behaviour as is evident in certain neuro-behavioural syndromes associated with frontal lobe dysfunction (Cummings, 1985). Dysfunction brought about by damage to the prefrontal areas, including the areas associated with the dorsolateral, orbitofrontal, and medial prefrontal cortices areas, may bring about distinctive syndromes. Cummings (1985) identifies three principal frontal lobe syndromes: (1) The orbitofrontal syndrome dominated by disinhibited and impulsive behaviour, (2) the frontal lateral convexity syndrome with apathy as its principle behavioural correlate, and (3) the medial frontal syndrome with different corresponding degrees of akinesia. These syndromes have unique behavioural modifications and can be coupled with different



anatomic systems within both frontal lobes. Traumatic, neoplastic, and other central nervous system (CNS) disorders are according to Cummings (1985), not solely confined to a contained area within a frontal lobe, nor to a single lobe either. The chief characteristics of each syndrome presents itself separately. The possibility of encountering mixed syndromes can however not be excluded. However, similar behavioural changes were identified in patients who had lesions in other areas of the brain (Mendez, Adams, & Lewandowski, 1989; Sandson, Daffner, Mesulam, & Carvalho, 1991). These findings challenge the view supporting the anatomic location of frontal lobe specific syndromes. Devinsky and D'Esposito (2004) further argues that frontal lobe syndromes appear as a result of an injury to a specific region of the brain involved, even though the influence of the functional localisation of the damage within the frontal cortex is still widely acknowledged.

Over the past three decades, neuroanatomical, neuropsychological, and functional imaging studies identified five parallel frontal-subcortical circuits that connect regions of the frontal lobes with subcortical structures (Alexander & Crutcher, 1990; Alexander, Crutcher, & DeLong, 1990; Alexander, DeLong, & Strick, 1986). Two of these circuits derive from outside of the prefrontal cortex, predominantly in the motor areas. The first of these circuits, known as the motor circuit, originates in the supplementary motor area, while the second circuit, the oculomotor circuit, originates in the frontal eye fields. The three remaining major frontal-subcortical circuits are the dorsolateral prefrontal circuit, the orbitofrontal circuit, and the anterior cingulate (ACC), or medial frontal circuit. Common to all three of the frontal-subcortical circuits are that they originate in the prefrontal cortex with projections to the striatal structures, which also project to the globus pallidus and substantia nigra, which links to the thalamus, with a final loop back to the prefrontal cortex (Figure 0.4) (Bonelli & Cummings, 2007). Despite remaining anatomically segregated, all five circuits share common structures

and are parallel and adjoining, with succeeding projections focused progressively onto smaller numbers of neurons.



*Figure 0.4. General organisation of the frontal-subcortical circuit*

Reprinted from *Neurology of cognitive and behavioural disorders* (p. 315), by O. Devinsky and M. D’Esposito, 2004, New York, United States of America: Oxford University Press. Copyright 2004 by Oxford University Press, Inc.

Cummings (1993) and various other researchers, such as Ardila (2008), Arnsten, (2009a), Bonelli and Cummings (2007), Fuster (2008), Gilbert and Burgess (2008), Lezak et al. (2012), Stuss (2011), and Zillmer et al. (2008) to name only a few, identified the dorsolateral prefrontal circuit as the most critical to executive functioning. This is because the dorsolateral prefrontal circuit is generally implicated in the execution of higher-order cognitive functions. This contributed to researchers’ tendency to refer to the dorsolateral prefrontal circuit as the “executive circuit” (Zillmer et al., 2008). Fuster (2002a) argues that the most fundamental executive function of the dlPFC is the temporal organisation of goal-directed actions in the domains of behaviour, cognition, and language. In a broader sense, the cognitive abilities that are governed by the dlPFC range on a continuum and generally reflect the ability to shift

between cognitive sets, employ already existing strategies, and the ability to organise information to ensure that demands in an ever-changing environment are met (Ardila, 2008). The most noteworthy deficit of this circuit is the inability to temporally organise a behavioural response to novel or complex stimuli (Ardila, 2008; Fuster, 2008).

Located within the ventral prefrontal cortex (VPFC), the orbitofrontal cortex (OFC) occupies the ventral floor of the prefrontal cortex (Nelson & Guyer, 2011). This distinct area of the prefrontal cortex is essentially linked to the limbic and basal forebrain regions (Ardila, 2008; Nelson & Guyer, 2011). The orbitofrontal cortex is particularly involved in bringing emotional and cognitive information together, accurately monitoring one's own behaviour, and making inferences about the mental states of other individuals (Beer et al., 2004). Therefore, it is not surprising to find that damage sustained to the orbitofrontal circuit may be associated with alterations in personality, irritability, mood liability, and an individuals' disregard for significant events (Saint-Cyr, Bronstein, & Cummings, 2002). The orbitofrontal area is commonly associated with appropriate social behaviour through the maintenance of personality and mannerisms. This area is responsible for the association of a stimulus with a reward, self-regulation of behaviour, reasoning, and complex decision-making (Malloy et al., 1999; Martin, 2006).

The medial frontal circuit is associated with arousal and motivation. According to Fuster (2002a), the ventromedial areas of the prefrontal cortex are involved in the expression and control of emotional and instinctual behaviour. McCullagh and Feinstein (2011) provide an overview of the categories of frontal executive functions and their location within the prefrontal cortex (Table 0.4).

Table 0.4. Categories of frontal executive functions

Functions	Region
<i>Executive cognitive functions:</i>	
<ul style="list-style-type: none"> <li>• Spatial, temporal, and conceptual reasoning</li> </ul>	Dorsolateral prefrontal cortex
<i>Activation-regulation functions:</i>	
<ul style="list-style-type: none"> <li>• Initiating and maintain mental processes</li> <li>• Monitoring response conflict</li> </ul>	Superior medial prefrontal cortex
<i>Behavioural self-regulation functions:</i>	
<ul style="list-style-type: none"> <li>• Emotional processing</li> <li>• Mediating response conflict</li> </ul>	Ventromedial prefrontal cortex
<i>Metacognitive processes:</i>	
<ul style="list-style-type: none"> <li>• Higher order integrative aspects of personality, self-awareness, and social cognition</li> <li>• Prospective memory, complex multitasking</li> </ul>	Frontal polar regions / Rostral prefrontal cortex

*Note.* Adapted from “Cognitive changes” by S. McCullagh and A. Feinstein, 2011, in J.M. Silver, T.W. McAllister, & S.C. Yudofsky, (Eds.), *Textbook of traumatic brain injury*, p. 283. Washington, DC, United States of America: American Psychiatric Publishers. Copyright 2011 by the American Psychiatric Association.

Another important aspect regarding the localisation of executive functions is the important role of white matter in the organisation of higher cognitive functioning (Filley, 2000). White matter tracts connect the frontal lobes with subcortical and posterior cortical structures. Given this involvement of white matter in subcortical circuits, it is not unexpected that damage to, or interference with, the integrity of white matter tracts may also contribute to executive dysfunction. White matter tracts are most likely implicated in all neurobehavioural domains, and are over the years more progressively recognised as mediated by widely distributed neural networks comprised of cortical, subcortical, and white matter components (Filley, 2000).

In addition, the functions associated with the prefrontal cortex can also further be subdivided by the left and right cerebral hemispheres. The left prefrontal cortex is linked with the initiation of responses including the processing of verbal, concrete, and detail-oriented

information. The right prefrontal cortex is linked with the inhibition of responses including the processing of information that is visual-spatial, abstract, or connotative, and gestalt-oriented (Lezak, Howieson, & Loring, 2004).

A final take on dorsolateral, orbitofrontal, and medial frontal mediated executive functions is the realisation that executive functioning is implicated in the mediation of emotional, motivational, and social behaviour (Zillmer et al., 2008). Impairment in executive functions appear when damage to the dorsolateral prefrontal circuit occur, resulting in problems relating to planning, sustained attention, the coordination of adaptive goal-directed behaviour, and inflexibility of thought in generating solutions to novel problems. Damage to the orbitofrontal circuit may bring about significant affective and social changes, including hyperactivity (inability to inhibit motor activity), the inability to appreciate the consequences of one's actions, emotional lability with euphoria or dysphoria, and increased aggressiveness. Damage to the medial prefrontal circuit (ACC) may lead to apathy (Figure 0.5) (Fuster, 2008).

These circuits provide a better understanding of how diverse anatomical lesions contribute to similar behavioural changes. Impairment in executive functions can be linked to frontal-subcortical dysfunction, because of their involvement in the regulation of higher cerebral processes that control cognition, the planning of complicated behavioural strategies, and decision-making (Bonelli & Cummings, 2007). Disorders, which involves the deep components of the frontal-subcortical circuits, may also affect executive function, based on their anatomical features (Filley, 2000). Hallmark consequences following damage to the three frontal-subcortical circuits include executive dysfunction, apathy, and disinhibition (Bonelli & Cummings, 2007). Filley (2000) provides a summary of the three distinctive prefrontal subdivisions and their neurobehavioural correlates in (Table 0.5).

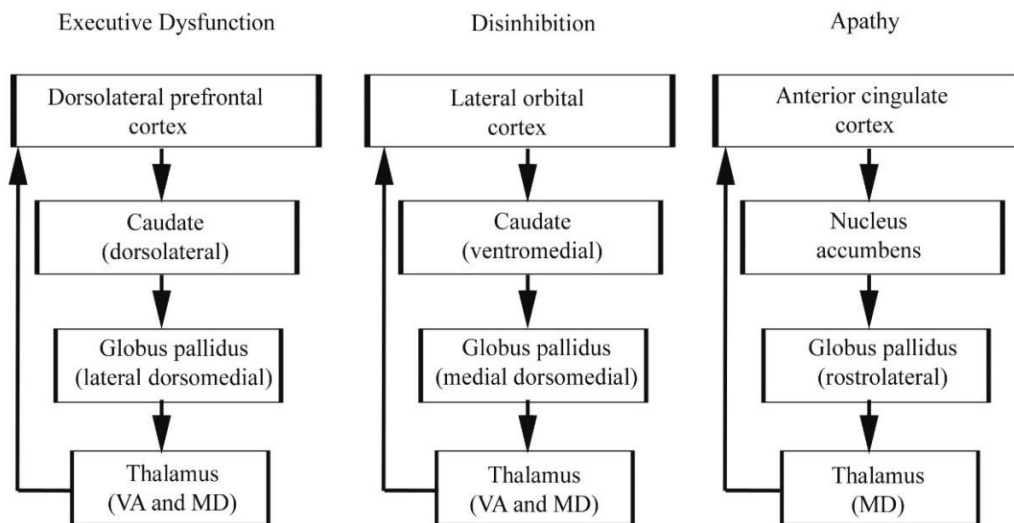


Figure 0.5. Organisation of the three frontal-subcortical circuits

Lesions in these circuits produce alterations of cognition and emotion (e.g., executive dysfunction, disinhibition, apathy). VA = Ventral anterior; MD = Medial dorsal. Reprinted from *Neurology of cognitive and behavioural disorders* (p. 314), by O. Devinsky and M. D’Esposito, 2004, New York, United States of America: Oxford University Press. Copyright 2004 by Oxford University Press, Inc.

Table 0.5. Prefrontal sub-divisions and their neurobehavioural correlates

Region	Dorsolateral	Orbitofrontal	Medial Frontal
<i>Functions</i>	Executive Function Working Memory	Personality Comportment	Arousal Motivation
<i>Clinical Frontal Lobe Syndromes</i>	Executive Dysfunction	Disinhibition	Apathy

Note. Adapted from “Clinical neurology and executive dysfunction” by C.M. Filley, 2000, *Seminars in Speech and Language*, 21(2), p. 92. Copyright 2000 by Thieme Medical Publishers, Inc.

To conclude the neuroanatomy of executive functions it is important to consider executive functions along with the other categories of frontal lobe functioning (energisation, emotional or behavioural regulation, and metacognition), each of which is associated with a different region within the frontal lobes. A schematic illustration (Figure 0.6) is provided below to aid in the conceptualisation of the discrete functional categories within the frontal lobes. Anatomical studies and the mapping of human brain development over the years managed to

highlight two main frontal lobe systems. These consisted of a lateral system with primarily bidirectional connections to and from the posterior cortices (referred to as the executive system) and a second inferior/medial system with prominent limbic connections (commonly referred to as the emotional system). Both these systems are energised by the superior medial region, while the fronto-polar region (Rostral Prefrontal Cortex) is responsible for integrating the executive and emotional processes. Given the five identified parallel frontal-subcortical circuits, three aligns with the functional categories of energisation, executive, and emotion. The fronto-polar region however does not have major frontal-subcortical connections because of its integrative role within the frontal lobes and other brain regions. Understanding the role of specific brain regions within the frontal lobes provides the basis for investigating the role of separate circuits as well as the integration between and among circuits. The frontal lobes do not equal a central executive. Only a single functional category within the frontal lobes are represented by executive functions. These frontal lobe functions are domain general. This is due to the extensive reciprocal connections with various other brain regions through the integration of information from these regions. It is this additional integration with emotional and motivational processes that allows for the most intricate human behaviours (Grafman, 2002; 2006).

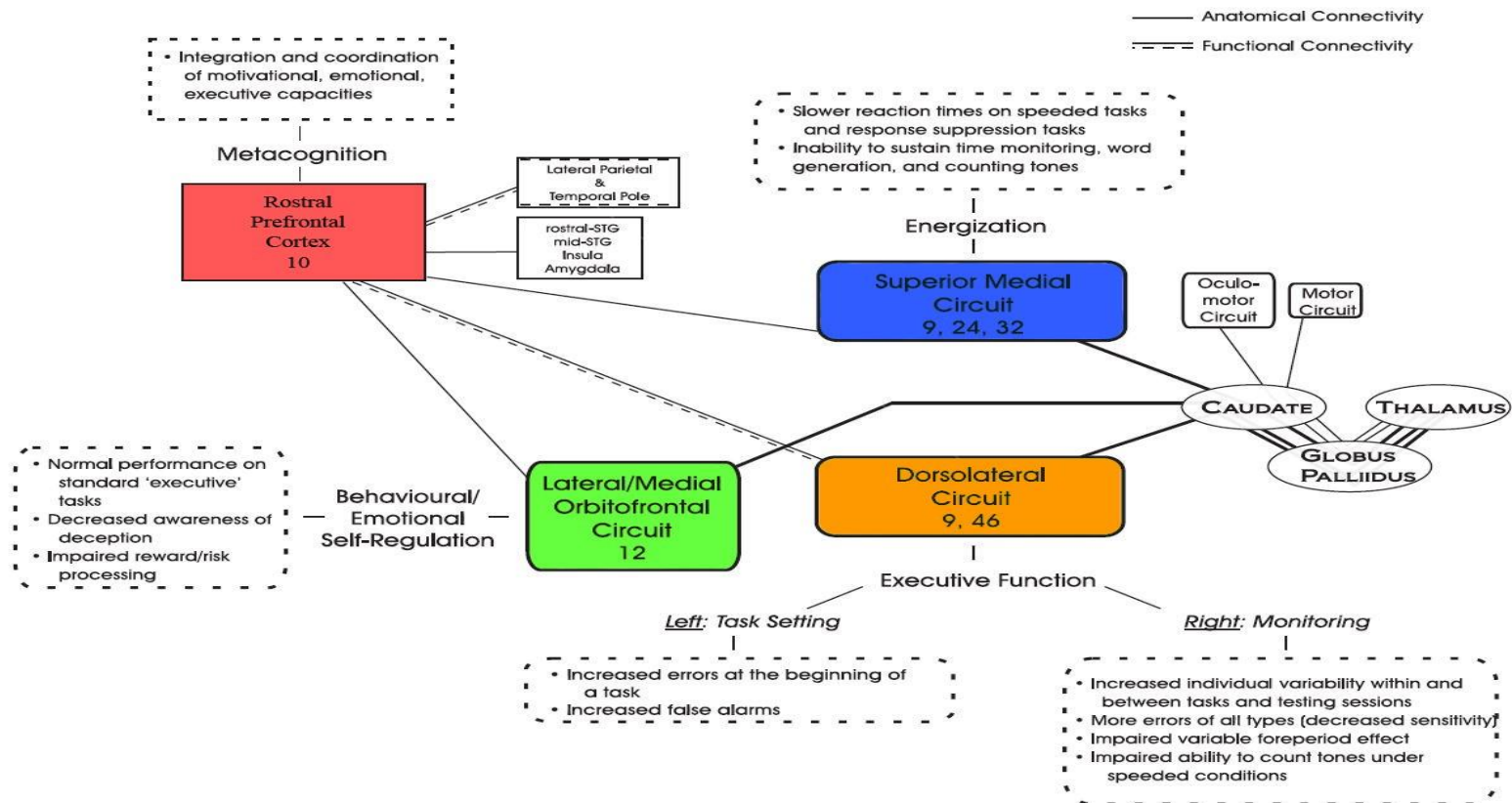


Figure 0.6. The frontal cortical-basal ganglia-thalamic circuits

This figure serves as an illustration of the frontal cortical-basal ganglia-thalamic circuits and in addition, supports the fractionation of the supervisory attentional system (SAS) of the frontal functional regions. Adapted from “Functions of the frontal lobes: Relation to executive functions,” by D.T. Stuss (2011), *Journal of the International Neuropsychological Society*, 17(5), p. 762. Copyright 2011 by International Neuropsychological Society.



Currently researchers accept that the prefrontal cortex is not the only brain area involved in executive functioning. They arrived at this conclusion because the frontal lobes are richly connected to various other brain regions. Almost all executive functioning processes rely on the integrity of complex neural networks instead than on a single frontal lobe area (Figure 0.7). Individual aspects of executive functioning are thus not viewed as easily localised. Working memory for example is not only dependent on the dorsolateral prefrontal cortex, but also on portions of the parietal lobe. In addition, response initiation is dependent on not only the left medial and ventral prefrontal cortices, but are also depended on the basal ganglia and the thalamus. In a similar vein, sustained attention relies on not only the superior medial prefrontal cortex, but also on the integrity of many regions within the right hemisphere and the thalamus (Aron, 2008; Tekin & Cummings, 2002).

There is consensus amongst researchers that the prefrontal cortex serves as the seat of high-level cognitive systems that receives input from specific lower-level systems and controls the activity of these lower-level systems. The prefrontal cortex is fundamentally complex with significant functional variation among the lateral, orbital, and medial prefrontal regions. As one progresses towards the fronto-polar regions a broader abstraction of function occurs. The dorsolateral prefrontal cortex is the most investigated region of the prefrontal cortex because of its association with executive functions.

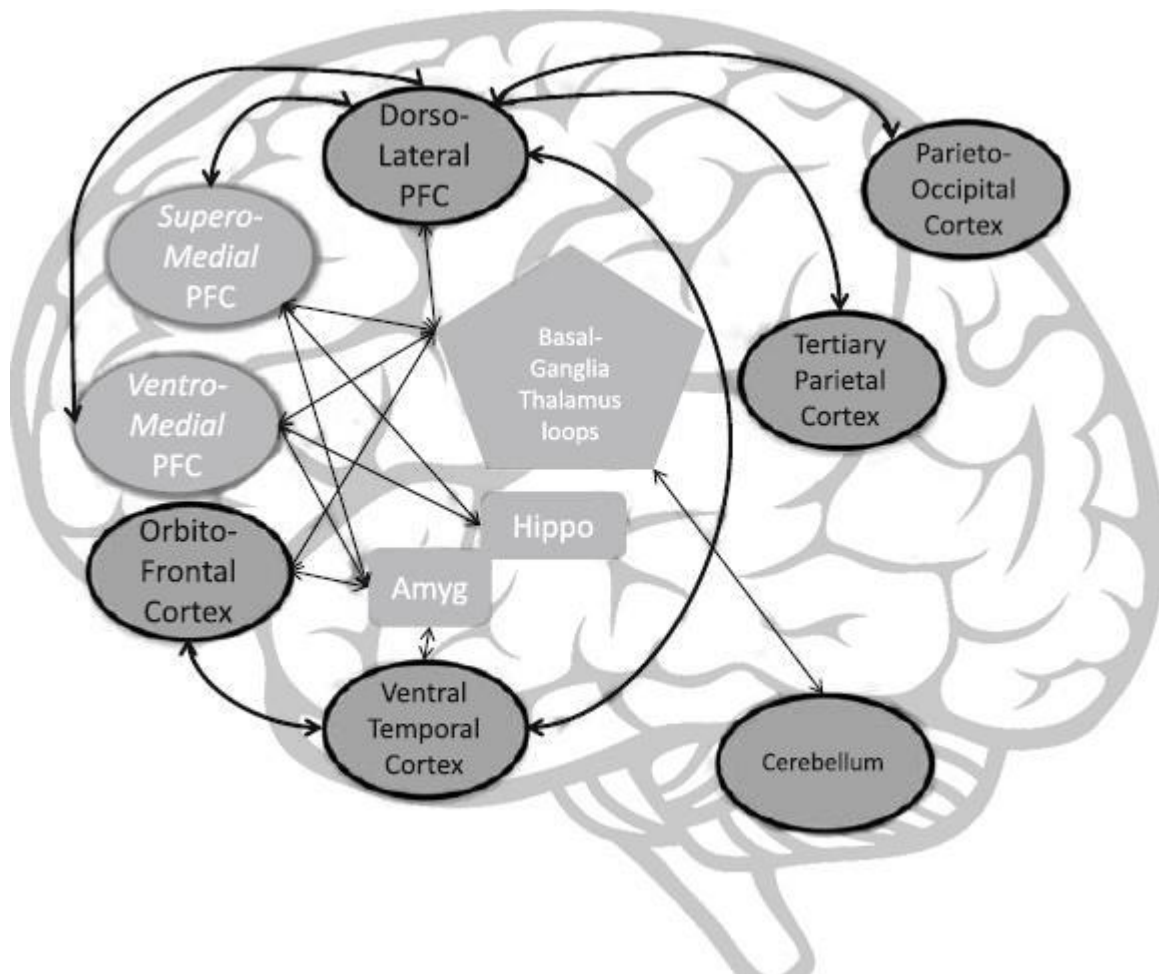


Figure 0.7. The reciprocal connection among the different convexities of the prefrontal cortex, limbic, and subcortical structures

Ovals that are outlined in black represent lateral cortical areas and cerebellum. Ovals outlined in gray represent medial cortical areas. Terms inside gray shapes without an outline represent subcortical or deep brain structures (amygdala, hippocampus, and basal ganglia/thalamus circuits). Curved thick lines represent cortico-cortical connections, whereas thin straight lines reflect cortico-subcortical connections. Adapted from “Executive functioning: Overview, assessment, and research issues for non-neuropsychologists”, by Y. Suchy, 2009, *Annals of Behavioural Medicine*, 37(2), p. 108. Copyright 2009 by The Society of Behavioural Medicine.

#### 1.4 Chapter Summary

To conclude this chapter, the researcher will provide a summary of the above by revisiting the definition of executive functioning as proposed by Suchy (2009). While being mindful that there is no universally accepted definition, Suchy (2009) proceeds to define

executive functioning from the following perspectives. All of which have been dealt with in this chapter:

1. The overarching evolutionary purpose of executive functioning. This allows an individual to choose and engage in purposeful, goal-directed, and future-oriented behaviour ensuring success and quality of life.
2. The clinical syndromes found in an individual who, resulting from damage to the brain loses the ability to engage in purposeful and goal-directed behaviour. Specifically, lesions found in the dorsolateral prefrontal cortex (dlPFC) and the related circuitry, lead to difficulty in reasoning, organising, planning, and solving problems—all these depend on an intact working memory capacity. This group of symptoms is referred to as the “dysexecutive syndrome”. Damage sustained to the orbitofrontal (OFC) and ventromedial prefrontal cortices (vmPFC) and their related circuitry, lead to impairment in the domain of social appropriateness and the making of adaptive choices, as well as depending on inhibition and response selection. This group of symptoms is referred to as the “disinhibited syndrome”. Damage sustained to the superior medial prefrontal cortex (mPFC) contribute to difficulties in the domain of motivation, initiation, and sustained attention. This group of symptoms is referred to as the “apathetic syndrome”.
3. The complex skills that make purposeful and goal-directed behaviour possible. This perspective addresses the ability to reason and solve problems, plan, and organise. Individuals with Posttraumatic Stress Disorder (PTSD) for example may find themselves incapable of producing solutions, because they suffer

from the disintegrating ability to reason, which resorts under the umbrella of executive functions.

4. Elemental neurocognitive processes forming the basis for specific executive functioning skills. From this perspective, these skills include inhibitory control, working memory, and cognitive flexibility.
5. Atheoretical approaches complicates the definition of executive functions. The variables, tasks, and populations of different studies drive these approaches to compile an oversimplified understanding of executive function. This perspective highlights that constructs such as self-regulation, self-control, emotion regulation, delay of gratification, attentional control, self-monitoring, and response modulation all depend on some or another aspect of executive function.
6. This perspective highlights the neuro-anatomic substrates that support complex skills and basic neurocognitive processes. Donald Stuss for example chose to stress the functional neuro-anatomy of the human frontal lobes (dorsolateral, superior medial, and ventral prefrontal cortices) above executive functioning constructs (Stuss & Alexander, 2000). Suchy (2009) concurs that the neuropsychological standpoint could be the most reasonable approach, because in this approach the physical boundaries are undisputed.
7. Constructivistic definitions. This is the last perspective proposed by Suchy (2009) by which to approach the definitions of executive functioning. This perspective acknowledges that, because of the complex nature of executive functioning and the struggles to define it, researchers proposed theoretical models that include new constructs or latent variables that could explain the

structure and function of executive functioning. One such model proposes a single unifying entity thereby hoping to capture the executive functioning construct of a central executive in its entirety. Although this (Baddeley's central executive model) and other similar models provide useful frameworks to study the constructs of executive functioning, they fail to provide researchers with executive functioning strengths and weaknesses among different populations. The reason for this shortcoming is that these models are unable to capture all clinically and theoretically relevant aspects of executive functioning.

A model that does provide a better understanding of executive functioning is the tripartite model of executive functioning (Miyake & Friedman, 2012; Miyake et al., 2000). This model consists of three entities: The ability to (1) form, (2) maintain, and (3) engage in shifting mental set (Suchy, 2009). These three entities correspond with (1) the ability to reason and solve problems (working memory), (2) maintain the motivation to continue with a response in the absence of external structures (inhibitory control), and (3) the ability to change goals and alter plans as necessary in response to changing environmental demands (cognitive flexibility). The advantage of this tripartite model (diversity model) is that it provides descriptions of varying profiles of the strengths and weaknesses amongst the three affirmed aspects of executive functioning. This may lead to predicting behavioural responses.

Finally, it can thus be stated that executive functioning is a multifaceted neuropsychological construct that consists of a set of higher-order neurocognitive processes that allow higher organisms to make choices and to engage in purposeful, goal-directed, and future-oriented behaviour. Therefore, no individual isolated part of the brain—no “homunculus”—can be delineated as the neural equivalent of a chief executive. Instead, various integrated brain systems act in concert to make flexible and goal-directed control of

behaviour possible. Executive functioning provides humans with an evolutionary advantage that release them from innate, hard-wired drives, and reflexes. It also frees them from over-practiced, over-learned, and reflexive actions—unlike animals that are solely driven by instinct or conditioning. Humans, because of their highly evolved executive functioning capacity, can consider multiple options and select specific responses to stimuli based on situational context, knowledge previously acquired, and long-term goals. From an energy consumption perspective, executive functioning is demanding and energy intensive. This costly process remains dormant for the greater part of the day, being activated only when odd and/or intricate situations rule out automatic or routine responses. Therefore, an automatic or routine response is not a reflection of executive functioning.

A wide range of frontal lobe symptoms may arise following selective damage to either the dorsolateral, medial, or orbitofrontal regions. Therefore, no single frontal lobe syndrome exists. Impairment in executive functions may follow when damage to the dorsolateral prefrontal circuit occurs, while critical affective and social changes may arise due to damage sustained to the orbitofrontal circuit. In addition, damage to the medial prefrontal circuit may cause a decrease in motivation and behavioural spontaneity. Therefore, damage to the frontal system brings about a disruption in most of the cognitive and behavioural abilities that have come to define humanity.

## CHAPTER 2

### STRESS

*“Sometimes even to live is an act of courage.” – Lucius Annaeus Seneca*

*(5 BCE–39CE)*

From the dawn of humanity, humans have experienced stress as part of their everyday life. The degree, however, to which they experience stress differ vastly, because each one of us exhibits a unique vulnerability or resilience to stressful challenges in our environment. In the following paragraphs, stress, although often studied from as a psychological construct, will be viewed from a biological perspective, and specifically how it activates neurobiological systems that preserve life through change or allostasis. Frequent neurobiological stress responses, although necessary for survival, may increase the risk of future physical and mental health problems, even more so when experienced during periods of rapid brain development (Gunnar & Quevedo, 2007).

In this chapter the classical definition of the normal stress response in all its stages, as developed by Hans Selye, will be provided. Thereafter, the physiology of the stress response will be provided in detail through a discussion firstly on how the amygdala reacts to threat and thereafter the role of the hypothalamic-pituitary-adrenal (HPA) axis response to stress will be addressed. An explanation will then be provided of the physiology of the stress response focussing on the different levels of physiological reaction in the face of a threatening situation. Here the emphasis will fall on the concept of allostasis and allostatic overload.

This chapter will also attempt to bring stress and trauma into relation with one another, while the emphasis will be placed on the biopsychological perspectives of traumatic stress responses and the development of posttraumatic stress disorder (PTSD). An overview of the

neuro-anatomy and biology of PTSD will be provided followed by an overview of the large-scale neurocircuitry models implicated in PTSD. The chapter will end with a discussion on the prevalence of PTSD in the world in general and South Africa in particular.

## **2.1 The Stress Response**

Human organisms have reacted to threat and danger in their environment from the earliest time of their existence to the present day by exhibiting stress. The psychic residue of such exposure has most commonly been approached by examining the stress response (Brannon & Feist, 2010; Thiel & Dretsch, 2011), which under certain circumstances may lead to experience the event as trauma. Hans Selye's view on stress is that a stressor (a stimulus) impacts on an organism which leads the organism to respond. He defined this response, how the body defends itself in stressful situations, as stress. According to Selye, stress is a general physical response caused by any of a number of environmental stressors. Selye (1998) conceptualised the body's attempt to defend itself against stressors as the General Adaptation Syndrome (GAS). The syndrome is divided into three stages (Figure 0.1) (Brannon & Feist, 2010):

The first stage, known as the alarm stage, is when the stressor sends the organism into a state of readiness for fight, flight, or freeze by activating the sympathetic nervous system (SNS). At the most basic level, the alarm stage involves a series of SNS and endocrine responses that aim to restore stability within the body and promote the ability of an organism to deal with a threat (Thiel & Dretsch, 2011). A critical feature of these systems is to mobilize energy resources for instant use while simultaneously inhibiting body functions that are nonessential for immediate survival. This results in an increase in heart rate, blood pressure, and blood glucose levels while digestive and reproductive processes are cut back.



Selye's second stage of the GAS is the resistance stage. In this stage, the organism attempts to adjust to the stressor by mobilizing various biological, psychological, and social resources. Continuing stress will cause continued neurological and hormonal changes. Despite its importance for survival, chronic and frequent physiological stress responses can lead to alteration in brain development, subsequently leading to dysregulation of neural circuitry (Acheson, Gresack, & Risbrough, 2012; Carrion & Wong, 2012; Karl et al., 2006; Liberzon, Britton, & Phan, 2003). Sapolsky (1996) reports that although adrenal steroid hormones, such as epinephrine, norepinephrine, and glucocorticoids are essential for the survival of acute physical stress, these adrenal steroid hormones may have the potential to cause adverse effects on the brain when their secretion is sustained over a long period of time.

In the third stage, the exhaustion stage, the organism exhausts its resources and becomes dysfunctional or collapses, due to what Selye termed adrenal exhaustion. This stage is characterized by an abnormally low level of functioning of the parasympathetic nervous system (PSNS) causing a person to become exhausted.

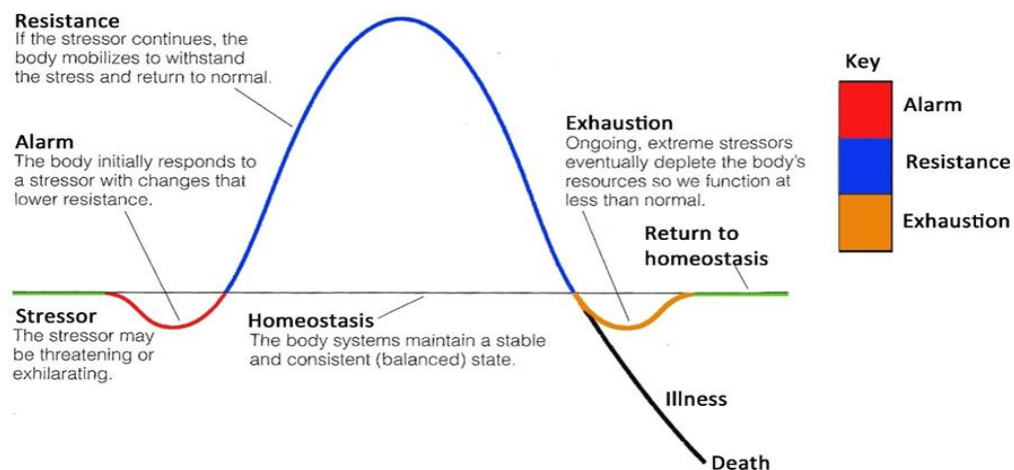


Figure 0.1. The three stages of Hans Selye's general adaptation syndrome (GAS)

Adapted from *Health Psychology: An Introduction to Behaviour and Health* (p. 103), by L. Brannon and J. Feist, 2010, Belmont, United States of America: Cengage Learning. Copyright 2010 by Wadsworth Cengage Learning.

Selye's model remains the basis of our biosocial understanding of stress as a process of adaptation and maladaptation. Recent scholarly thought, however is of the opinion that Selye's view of the physiology of stress was too simplistic. Selye largely ignored the situational and psychological factors that contributes to stress, including the emotional component and the individual interpretation of stressful events, which makes his view of stress incomplete in the view of psychologists like Brannon and Feist (2010), Christopher (2004), and Mason (1971; 1975). In contrast to Selye, Richard Lazarus deems the interpretation of a stressful event more important than the event itself. According to Lazarus, (Brannon & Feist, 2010), neither the environmental event nor the person's response defines stress; rather, the individual's perception of the psychological situation is the critical factor. This perception includes potential harms, threats, and challenges as well as the individual's perceived ability to cope with them. In other words, the person's feelings of threat, vulnerability, and ability to cope are more important than the stressful event itself.

This more complex view of the human stress response is part of a paradigm shift within psychology that interconnects biological, psychological, and sociocultural data. This shift is reflected in Lazarus and Folkman's definition of psychological stress as a "particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" (1984, p. 19). Brannon and Feist (2010) provides the following comments on Lazarus and Folkman's definition of psychological stress by stating that in the first instance the definition refers to a relationship between a person and his or her environment, it therefore takes a transactional position. Secondly, that the definition holds that the key to that transaction is the person's appraisal of the psychological situation, and thirdly Brannon and Feist (2010) emphasizes that the situation must be seen as threatening, challenging, or harmful.

Thus, Selye's general adaptation syndrome is no longer interpreted to mean that all types of stress evoke the same stereotyped response of the stress mediators. McEwen (2005), reports that the hypothalamic-pituitary-adrenal (HPA) axis and the noradrenergic and adrenergic nerves have different patterns of response that are related to the type of stressor. Furthermore, "the fight or flight" response does not apply equally to both sexes. The female response to non-life-threatening stress has been characterised by Taylor et al. (2000) as "tend-and-befriend," and not "fight or flight."

## **2.2 The Physiology of the Stress Response**

The physiology of the normal stress response will first be discussed at the hand of Christopher (2004), who places the emphasis first on the amygdala and threat recognition and secondly on the role of the hypothalamic-pituitary-adrenal (HPA) axis within the normal stress response. Thereafter, Brannon and Feist's (2010) overview of the physiology of the stress response will be given. Their contribution is relevant because they move beyond explaining the physiology of the stress response by focussing on the different levels of physiological reaction provided in the face of a threatening situation.

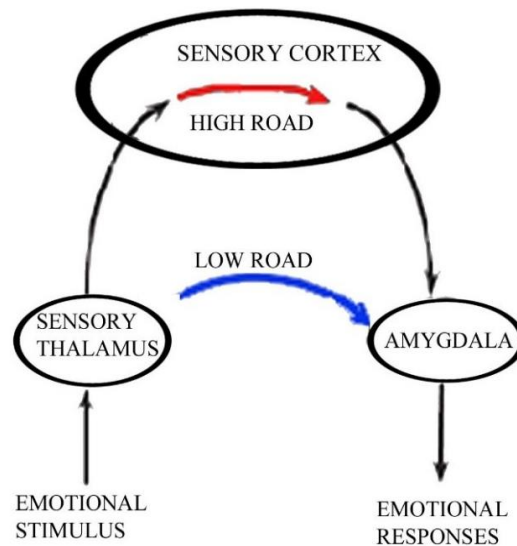
### **2.2.1 The amygdala and threat recognition.**

The psychological stress response, of which fear is considered the most primitive form, is regulated by the hypothalamic-pituitary-adrenal (HPA) axis, it begins however in the amygdala, which is an almond-shaped brain structure allocated in the medial temporal lobe of the brain. The amygdala processes stimuli from the senses to detect threats to the organism (LeDoux, Iwata, Cicchetti, & Reis, 1988) and control defensive responses with regard to threatening situations (Southwick, Rasmusson, Barron, & Arnsten, 2005). In essence, the amygdala is implicated in both the acquisition and expression of fear (Davis, 1992b). It receives

information about external stimuli and plays a key role in determining their significance (Davis, 1992a).

The amygdala has strong connections to the hypothalamus and the brainstem nuclei that are responsible for mediating fear responses, including freezing behaviours, variation in heart rate and blood pressure, sweat gland activity, and the release of stress hormones (Southwick et al., 2005). Threat-induced activation of the amygdala leads to alterations in stress hormones, particularly catecholamines (a family of neuromodulators consisting of dopamine, norepinephrine, and epinephrine) and glucocorticoids, which in turn triggers the appropriate emotional response. Bisson (2009) establishes that these responses are influenced by various other areas of the brain, with the hippocampus, medial prefrontal cortex, and perhaps the anterior cingulate gyrus in particular, being key. Bisson (2009) furthermore, is of the opinion that the hippocampus and medial prefrontal cortex weakens the response triggered by the amygdala.

There are two paths through which the amygdala's fear response can be triggered (Figure 0.2), a fast low road, that passes from the thalamus to the amygdala and a slower high road, that passes from the thalamus to the neocortex, and then to the amygdala.

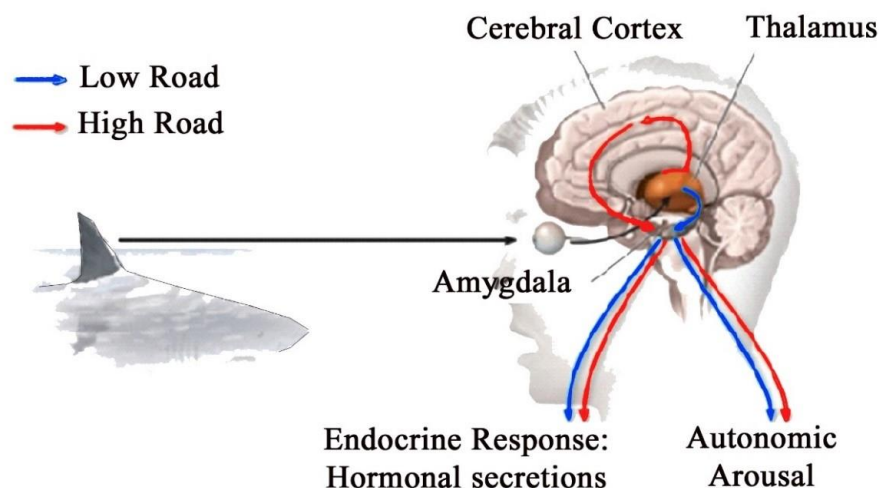


*Figure 0.2. The low road and the high road to the amygdala*

Reprinted from *Emotional networks in the brain* (p. 163), by J.E. LeDoux and E.A. Phelps, 2008, New York, United States of America: The Guilford Press. Copyright 2008 by The Guilford Press.

The second path or low road is very effective in situations of simple threat, when a rapid but rigid response such as fight or flight is adequate. The high road, unlike the low road, is affected by learning, as well as evolutionary inherited responses, because it is modulated by the cortex. Once the environmental stimuli are processed by the amygdala, neural projections from the amygdala transmit the information to the reticularis pontis, where the startle response and other defensive behaviours that do not require the direct action of the sympathetic nervous system are initiated (Davis, 1992a). Simultaneously, projections from the amygdala to the lateral hypothalamus, and then to the rostral ventral medulla, initiate the sympathetic nervous system and the release of catecholamines (LeDoux et al., 1988), while projections to the solitary tract stimulate a parasympathetic response (McGaugh, 2002). The discharge of the sympathetic nervous system produces an increase in heart rate and blood flow, accelerating the rate that glucose is pumped to the skeletal muscles, preparing them for flight from the threat or for some form of confrontation with the threat (Mountcastle, 1974). Other projections from the

solitary tract stimulate the parasympathetic response. The parasympathetic response initiates a negative feedback loop, which dampens the sympathetic discharge, and, finally, projections from the bed nucleus of the stria terminalis initiate the HPA axis (Roозendaal, Koolhaas, & Bohus, 1992). The HPA axis, the heart of the stress response, produces a number of changes that enable the organism to deal with a wide range of stressors by increasing attention, stamina, and reaction time. Ciccarelli and White (2015) provide the following example to illustrate the role of the amygdala in threat recognition and the neural pathways of signal transmission involved during fear (Figure 0.3).



*Figure 0.3. The role of the amygdala in threat recognition and the neural pathways of signal transmission involved during fear*

In this example the low road shouts “Danger!” and we react before the high road says, “It is a shark”. The low road is the pathway underneath the cortex and is a faster, simpler path, allowing for quick responses to the stimulus, sometimes before we are consciously aware of the nature of the stimulus. The high road uses cortical pathways and is slower and more complex, but it allows us to recognise the threat and, when needed, take more conscious control of our emotional responses. Reprinted from *Psychology* (p. 372), by S.K. Ciccarelli and J.N. White, 2015, Boston, United States of America: Pearson. Copyright 2015 by Pearson Education, Inc.

This two-road model of signal transmission demonstrates the way fear responses can be initiated before we are conscious of the eliciting stimulus.

### **2.2.2 The general characteristics of the HPA axis and the normal stress response.**

The hypothalamic-pituitary-adrenal (HPA) response to stress constitutes a fundamental aspect of the organism's reaction to stimuli that threaten homeostasis, such as stress (Diorio, Viau, & Meaney, 1993). The key elements of the HPA axis are depicted in (Figure 0.4). The HPA axis is triggered by the projections from the bed nucleus of the stria terminalis. This action begins with perception of a threatening situation, which prompts action in the hypothalamus. The paraventricular nucleus (PVN) of the hypothalamus is stimulated by brain neuropeptides that result in the secretion of corticotrophin-releasing factor (CRF), vasopressin, and a cascade of other regulatory neuropeptides (Rivier & Plotsky, 1986). CRF and the other neuropeptides stimulate the anterior pituitary gland to release adrenocorticotrophic hormone (ACTH). ACTH causes the adrenal cortex to release mineral corticoids, such as aldosterone, which helps control electrolyte levels, as well as glucocorticoids, in particular cortisol (Yehuda, 2009), which dampen the arousal effects, stimulate the immune system, and influence the metabolism of carbohydrates, protein, and fats. The secretion of cortisol mobilises the body's energy resources raising the level of blood sugar to provide energy for the cells and vital organs. In addition to mobilising and restoring energy stores, cortisol also suppresses growth and reproductive systems, holds back the immune response, and influences behaviour through acting upon various neurotransmitter systems and brain regions (Southwick et al., 2005). Cortisol also has an anti-inflammatory effect, giving the body a natural defence against swelling from injuries that might be sustained during a fight or flight.

The more the stress, more catecholamines and cortisol are released (Selye, 1976). The sympathetic branch of the autonomic nervous system activates the adrenal medulla, causing it to release a mixture of catecholamines, primarily epinephrine and norepinephrine. Epinephrine is secreted solely by the adrenal glands, while norepinephrine is produced at numerous sites throughout the body and the brain. The catecholamines facilitate arousal and the availability of

energy to the body's vital organs, while cortisol helps to shut down sympathetic activation and other arousal and defence mechanisms (Munck, Guyre, & Holbrook, 1984). In other words, cortisol acts as an antistress hormone by playing an important role in the termination of the stress reaction and by stopping the further release of more cortisol (Keller-Wood & Dallman, 1984). Once the acute stressor is no longer detected by the amygdala, cortisol initiates a negative feedback inhibitory response on the pituitary gland, hypothalamus, hippocampus, and amygdala (McEwen, De Kloet, & Rostene, 1986; Yehuda, 2009), each of which contains high concentrations of cortisol and glucocorticoid receptors (Jacobson & Sapolsky, 1991; McEwen et al., 1986; Reul & De Kloet, 1985). In doing so, it returns the hormones of the HPA axis to their basal levels.

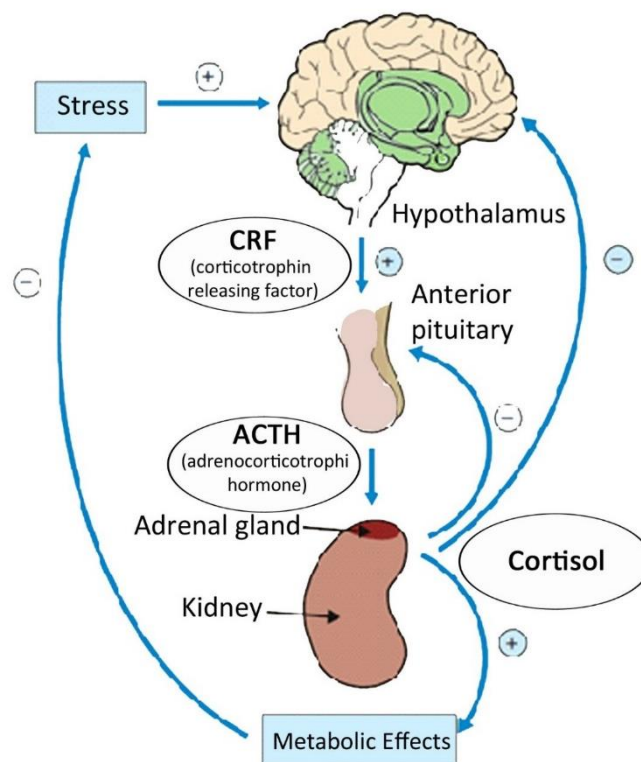


Figure 0.4. The hypothalamic-pituitary-adrenal (HPA) axis

Adapted from “The neurobiology of posttraumatic stress disorder,” by J.I. Bisson, 2009, *Psychiatry*, 8(8), p. 289. Copyright 2009 by Elsevier Ltd.



### **2.2.3 The physiology of the stress response: Different levels of activation.**

According to Brannon and Feist (2010), the physiological reactions to stress begin with the perception of stress. That perception results in activation of the sympathetic division of the autonomic nervous system, which mobilizes the body's resources to react in emotional, stressful, and emergency situations. Sympathetic activation prepares the body for intense motor activity, the sort necessary for attack, defence, or escape (Walter Cannon's (1932) idea of fight or flight). This mobilisation occurs through two pathways and affects all parts of the body.

One pathway is through direct activation of the sympathetic division of the ANS (called the adrenomedullary system), which activates the adrenal medulla to secrete epinephrine and norepinephrine. The effects occur throughout the body, affecting the cardiovascular, digestive, and respiratory systems. The other pathway is through the hypothalamic-pituitary-adrenal (HPA) axis, which involves all of these structures. This pathway has already been discussed above.

Brannon and Feist (2010) move beyond explaining the physiology of the stress response when they focus on what the different levels of physiological reaction provides in the face of a threatening situation. For them the point of the physiological reaction to stress is to provide a mixture of responses that allow remodelling to a threatening situation. The process leading to adaptation and thereby maintaining an appropriate level of activation is referred to as allostasis. During this process glucocorticoids and epinephrine as well as other mediators, promote adaptation to the stressor (McEwen, 2005). The wide range of circumstances that people encounter requires different levels of physiological activation. Activation of the sympathetic nervous system is the body's attempt to meet the needs of the situation during emergencies. At its optimum in maintaining allostasis, the autonomic nervous system adapts smoothly adjusting to normal demands by parasympathetic activation and rapidly mobilising resources for threatening or stressful situations by sympathetic activation. However, prolonged

activation of the responses of the sympathetic nervous system creates allostatic load, which can overcome the body's ability to adapt. In other words, if the body's alarm response is sustained and the adrenal output of glucocorticoids and catecholamines is repeatedly elevated over many days, an allostatic state may ensue, leading to allostatic overload. A state of allostatic overload represents the almost inevitable wear and tear produced by exposure to mediators of allostasis. Such a state may result in the exacerbation of pathophysiological change (Gunnar & Quevedo, 2007; McEwen, 2005).

In the following paragraphs, stress and trauma will be brought into relation to one another. The emphasis will be on the biopsychosocial perspectives of traumatic stress responses.

### **2.3 Stress versus Trauma**

The general belief is that trauma is simply an extreme form of stress. This belief is however complicated by the fact that, because of the high road to the amygdala, where an event that is located on this continuum has as much, and often more, to do with subjective experiential factors, than it does with the objective nature of the event (Christopher, 2004). The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) acknowledges that psychological distress following exposure to a traumatic or stressful event is quite diverse. The DSM-5 does not distinguish between a traumatic or stressful event. However, not all stressful events are experienced as traumatic. Research shows that in contemporary society, trauma is quite common and, in many environments, most people experience one or more traumatic events during a lifetime. In addition to natural disasters and war, trauma include accidents, criminal violence, domestic violence, disease within the family and community, and all sorts of forms of neglect and emotional abuse. Data obtained from the Institute for Security Studies (ISS), a leading African policy research and training organisation based in Pretoria, containing

crime statistics released by the South African Police Service (SAPS) for the period 2004 to 2016, illustrates the many and varied sources of manmade trauma present in South African society. During 2016 for example, 169 319 cases of assault intended to cause grievous bodily harm, 17 766 murders, and 47 731 sexual crimes cases, to name but three of the 29 crime categories, had been reported in South Africa (“ISS Crime Hub,” 2016). It must be remembered that violence, accidents, and natural disasters have been a regular part of human existence from its beginning, which is why evolutionary psychologists like Michael Christopher (2004), argues that humans have developed a specific response mechanism to cope with stress and trauma. He believes traumatic experiences are not only life-threatening or just bodily threatening events, but also includes threats to the self. Christopher (2004), under the influence of biological and evolutionary research, focuses on the distinction between chronic and acute stress as opposed to the specific content of the stressor. He is however aware that this focus on the negative consequences of stress continues to oversimplify the nature and significance of the response to traumatic and non-traumatic stress.

From the biopsychosocial perspective, a traumatic stress response is a normal evolutionary inherited response to extreme states of arousal that may have negative or positive consequences for adaption, adjustment, and well-being, depending on factors including genetic inheritance, individual experience, and sociocultural conditions. This perspective correlates with the overwhelming body of evidence that shows that only a minority of persons (10-35%) exposed to a traumatic event will develop some sort of pathological disorder (DSM-5: Trauma- and Stressor-Related Disorders) and that the majority of those who develop PTSD will be symptom free within 6-16 months without treatment (Baum & Fleming, 1993; Green & Lindy, 1994), while the majority, if not everyone, will experience some positive or adaptive effects in the form of Post Traumatic Growth (PTG). Positive personal change after adverse events (PTG) was shown to be a scientifically valid construct in a group of earthquake survivors by Eren-

Koçak and Kiliç (2014). They found that growth was predicted by executive functions and not by memory or processing speed, showing that the correlation between cognitive functions and growth is a specific one and that personal growth was related to cognitive functions, whereas relational growth was not.

Christopher (2004) supports the research that claims that the positive effects of trauma are universal. These effects are not restricted by the content of a traumatic event, but instead appear to be a function of the acute experience of powerlessness. Now that the distinction between stress and trauma has been unpacked, trauma developing into posttraumatic stress disorder as contained in the DSM-5 will be addressed.

#### **2.4 Trauma and Posttraumatic Stress Disorder (PTSD)**

The root of the word *trauma* lies within a Greek word meaning ‘to tear’ or ‘to puncture’ (Kaminer & Eagle, 2010). In the case of psychological trauma, this understanding is reflected in a notion of psychological wounding and the penetration of unwanted thoughts, emotions, and experiences into the psyche or the being of the person. Evidence of psychological suffering in response to trauma can be traced back to at least 1300BC (Gallagher, 2015). Trauma has become a keyword through which clinicians and scholars from various disciplines approach the experience of violence and its aftermath. Although psychological suffering in response to traumatic events has always been with us, it was not until 1980 that the term Posttraumatic Stress Disorder (PTSD) was officially introduced into the psychiatric literature (Joseph, Williams, & Yule, 1997). The term PTSD provided a common language, which has succeeded in bringing together research from various fields under one, unifying, theoretical umbrella. As a result, the concept of posttraumatic stress has infiltrated the public domain to such an extent that this terminology has become commonly used within society (Kaminer & Eagle, 2010). Damir and Toader (2014) pose the question whether trauma is an event or a feeling. They

choose to describe posttraumatic stress disorder initially as an event that has already ended when the disorder starts to manifest itself. In other words, the disorder follows the trauma. Damir and Toader (2014) continue to expand their definition, to not only include the “event” point of view in their understanding, but also to acknowledge the subjective processes involved. For them trauma has thus an objective and subjective side. It is thus fair to say that, being bodily injured through trauma (an event), also affects the victim’s dignity (feeling). Recent research done at the Freiburg Institute for Psychotraumatology found that psychological trauma should be treated as a moral injury with its consequences being understood as a “wound of the soul” (Damir & Toader, 2014, p. 277). Stress is thus acknowledged as a daily occurrence easily dealt with by most humans, whereas psychological trauma, is suffering or illness.

Litz (2004, p. 1) describes traumatic events as being “unpredictable, uncontrollable, and devastating.” Suddenly, individuals or groups are exposed to unthinkable suffering and threats to themselves or others, which lead to panic, terror, frailty, and vulnerability. As a result, these traumatic events defy core tacit beliefs and assumptions, which promote safety, stability, well-being, purposefulness, and personal and collective agency (Litz, 2004). It is not only that what happens to people which is considered as important, but also what it means to those people in relation to their sense of who they are, the world they live in, and what their expectations are of the future (Joseph et al., 1997). Thus, the exposure to traumatic events may challenge the whole meaning of a person’s life and his or her sense of purpose. It is during the time of the event and for a varying period afterwards, that trauma cripples’ normal functioning and overpowers consciousness, physiology, and coping resources (Kaminer & Eagle, 2010; Litz, 2004). Therefore, trauma as a metaphor draws the attention to the ways in which extremes of violence breaks bodies and minds, leaving ingrained marks even after healing and recovery (Kirmayer et al., 2007). An example hereof is found in a poem depicting the battle experience

of a soldier during The South African War, penned in the poem: “I killed a man at Graspan” (Pyke, 1904).

On 25 November 1899, Montague Macgregor Grover, an Australian soldier and a member of the Bushmen’s Contingent, wrote a poem called “I killed a man at Graspan” in which he depicts the impact of a traumatic event, which he experienced during The South African War that took place in South Africa between the forces of Great Britain and the two Boer Republics—the South African Republic (Transvaal) and the Orange Free State between 1899 and 1902.

### **I Killed a Man at Graspan**

I killed a man at Graspan,  
I killed him fair in fight;  
And the Empire’s poets and the Empire’s priests  
Swear blind I acted right.  
The Empire’s poets and the Empire’s priests  
Make out my deed was fine,  
But they can’t stop the eyes of the man I killed  
From starin’ into mine.

I killed a man at Graspan,  
Maybe I killed a score;  
But this one wasn’t a chance-shot home,  
From a thousand yards or more.  
I fired at him when he’d got no show;  
We were only a pace apart,  
With the cordite schorchin’ his old worn coat  
As the bullet drilled his heart.

I killed a man at Graspan,  
I killed him fightin’ fair;  
We came on each other face to face,  
An’ we went at it then and there.  
Mine was the trigger that shifted first,  
His was the life that sped.  
An’ a man I’d never a quarrel with  
Was spread on the boulders dead.

I killed a man at Graspan;  
I watched him squirmin’ till  
He raised his eyes, an’ they met with mine;  
An’ there they’re star’n still.

Cut of my brother Tom, he looked,  
Hardly more'n a kid;  
An' Christ! he was stiffenin' at my feet  
Because of the thing I did.

I killed a man at Graspán;  
I told the camp that night;  
An' off all the lies that I ever told  
That was the poorest skite.  
I swore I was proud of my hand-to-hand,  
An' the Boer I'd chanced to pot,  
An' all the time I'd ha' given my eyes  
To never ha' fired that shot.

I killed a man at Graspán;  
An hour ago about,  
For there he lies with his starin' eyes,  
An' his blood still tricklin' out.  
I know it was either him or me,  
I know that I killed him fair,  
But all the same, wherever I look,  
The man that I killed is there.

I killed a man at Graspán;  
My first an, God! my last;  
Harder to dodge than my bullet is  
The look that his dead eyes cast.  
If the Empire asks for me later on  
It'll ask for me in vain,  
Before I reach to my bandolier  
To fire on a man again.

This poem reflects poignantly the classic symptoms attributed to someone who is experiencing posttraumatic stress disorder. Kirmayer et al. (2007) explain that trauma can be viewed simultaneously as a socio-political event, a psychophysiological process, a physical and emotional experience, and a narrative theme in explanations of individual and social suffering. Thus, the concept of trauma has been extended to include a multitude of situations of extremity and equally varied individual and collective responses. The following distinctions are made which contribute to the various forms of catastrophic stress: Natural disasters, technological disasters, combat, criminal victimization, sexual assault, childhood sexual abuse, political violence, refugees, road traffic injuries, burn injuries, genocide, war, terrorist attack,

and widespread diseases (American Psychiatric Association, 2013; Danckwerts & Leathem, 2003; Joseph et al., 1997; Kaminer & Eagle, 2010; Wilson, 1994).

It is estimated that at some point of their lives, 50-80% of individuals will be exposed to a serious trauma, whether it is the result of war, sexual assault, extreme accidents, or other real-life horrors (Gillespie et al., 2009; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Roberts, Gilman, Breslau, Breslau, & Koenen, 2011). However, it is also predicted that only 5-10% of individuals who had been exposed to a traumatic life event will go on to develop symptoms, which will qualify them for the diagnosis of posttraumatic stress disorder (Aupperle, Melrose, et al., 2012). These findings have led researchers to examine which factors other than the trauma itself may contribute to, or protect against the development and maintenance of PTSD symptoms.

Both the characteristics of a traumatic event and that of the person involved determines the person's psychological and biological responses to such an event. Interwoven in the following discussion, which will endeavour to establish the relationship between an individual's normal reaction to trauma and him/her developing PTSD, the reader will find the psychological as well as the biological responses to traumatic events.

## **2.5 Normal Trauma Reactions versus PTSD**

Christopher (2004) argued that the normal trauma response is better understood as an evolutionary inherited mechanism for metalearning, which destroys and reform the metaschema (i.e., concepts of self, society, and nature), in which learning normally takes place. Although the focus is orientated towards the biological dimensions of this mechanism, the inherent assumption of the biopsychosocial model is that this process is always at the same time biological, psychological, and social. According to the perspective of evolutionary psychology, the self and the brain are resulting properties of the sociobiological interaction of



the brain's individual subsystems, each of which evolved and expresses itself, in adaptation with the environment (Cosmides & Tooby, 1997).

Following a traumatic event, almost every individual will experience some degree of distress as an effort to try and adapt to that what has happened. Kaminer and Eagle (2010) state that common reactions to a traumatic event include feelings of anxiety and mild depression, the experiencing of distressing thoughts and memories of the traumatic event, difficulty sleeping, and feeling hyper-alert to any signs of danger. Christopher (2004) ascribes these negative outcomes to trauma as a failure to adequately adjust to the normal adaptive trauma response. To cope with these symptoms, countless trauma survivors may wish to avoid talking about what happened, may withdraw from interacting with others, and they may experience emotional numbness when thinking of the trauma (O'Brien, 1998). These reactions to the trauma can last for a few days, weeks or even months after the traumatic event had taken place and then gradually fade, without severely affecting the survivor's ability to continue with their normal daily functioning (Kaminer & Eagle, 2010).

For some trauma survivors however, the symptoms mentioned above do not gradually diminish over time and continue to create significant impairment in their work and social environments. Posttraumatic Stress Disorder (PTSD) is therefore the psychiatric diagnosis that has been developed to describe such a response to trauma (Kaminer & Eagle, 2010). The current diagnostic criteria for PTSD is set out by the American Psychiatric Association (2013) described in the DSM-5.

PTSD symptoms according to the DSM-5 criteria varies, but can be summarised as persistent intrusive recollections, avoidance of stimuli related to the trauma, negative alterations in cognition and mood, and hyperarousal. A diagnosis can be made in someone

who's ability to function normally has been noticeably impaired for one month according to the DSM-5 criteria.

Yehuda, Pratchett, and Pelcovitz (2012), report that there appear to be multiple trajectories following exposure to a traumatic event. They mention four trajectories: First, people who do not experience significant distress following either immediately after the event or later. Second, there are people who initially experience symptoms of “PTSD” but whose symptoms gradually disappear within a relative short period. Thirdly, Yehuda et al. (2012) mentions a smaller group of people who initially experienced symptoms of PTSD and who then developed a chronic illness, and lastly, the smallest group who developed a delayed expression of PTSD. Yehuda et al. (2012) could not indicate under which trajectory an individual will resort from the onset of trauma. For examples of the many different trajectories of PTSD symptoms after exposure to trauma see Figure 0.5 below. Delayed presentation (the more the social support the longer the delay) is common, including where the effects are severe (Andrews, Brewin, Philpott, & Stewart, 2007; Horesh, Solomon, & Ein-Dor, 2013).

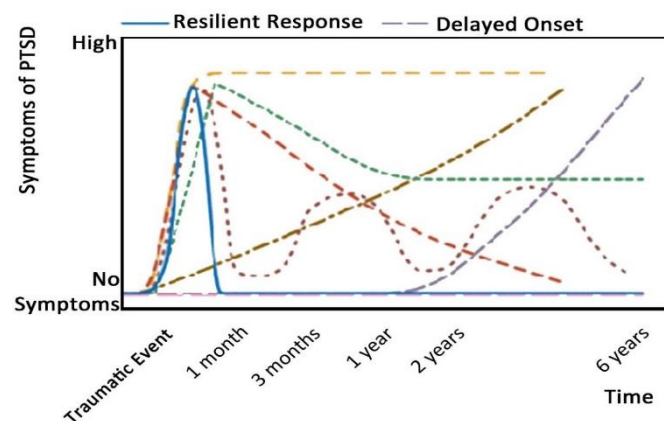


Figure 0.5. Examples of the many different trajectories of PTSD symptoms after exposure to trauma

Reprinted from “Post-traumatic stress disorder,” by J.I. Bisson, S. Cosgrove, C. Lewis, & N.P. Roberts, 2015, *The BMJ*, 351, p. 7. Copyright 2015 by the British Medical Journal Publishing Group.

Various life trajectories may develop in an attempt to cope with a traumatic event. These trajectories may range from surrendering and sheer surviving through recovery up to personal growth, which is not only a form of adaptive coping with a traumatic experience, but provides an additional benefit to the life and personality of the survivor (Hašto et al., 2013). Figure 0.6 below illustrates the different variations of coping with a traumatic event.

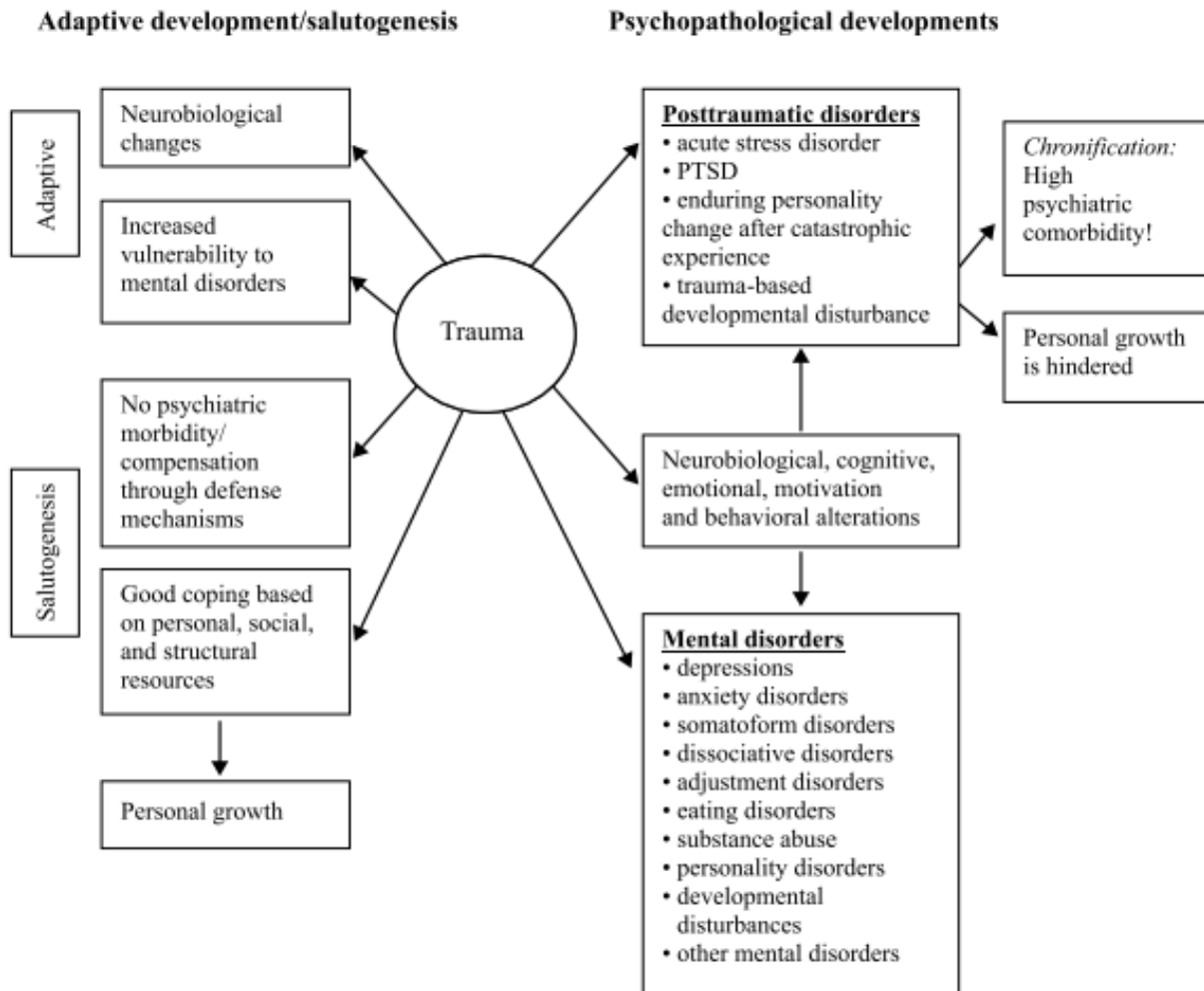


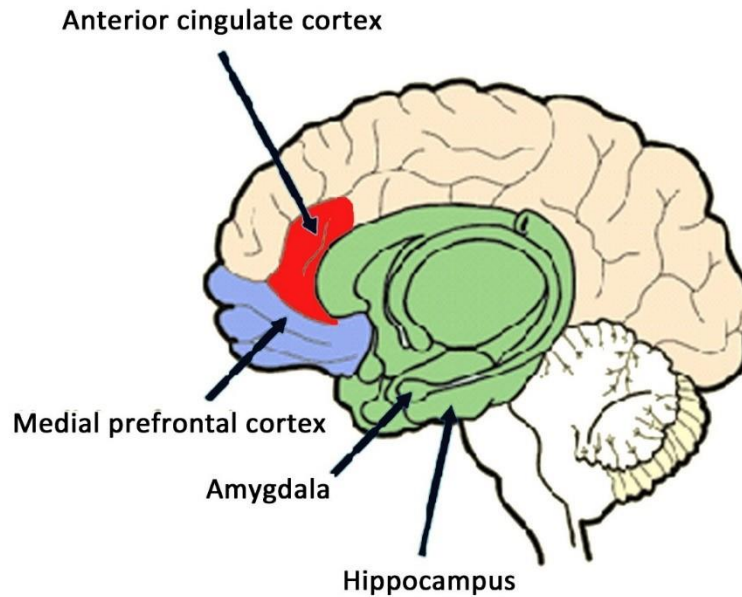
Figure 0.6. Possible developmental trajectories after a traumatic event

Reprinted from “Biopsychosocial approach to psychological trauma and possible health consequences,” by J. Hašto, H. Vojtová, R. Hrubý, & P. Tavel, 2013, *Neuroendocrinology Letters*, 34(6), p. 467. Copyright 2013 by Neuroendocrinology Letters.

## **2.6 Neuro-anatomy and Biology of PTSD**

According to Bremner (2011), traumatic stressors have the potential to contribute to several chronic psychiatric disorders. These disorders include not only posttraumatic stress disorder (PTSD), but also disorders such as depression (Franklin & Zimmerman, 2001; Prigerson, Maciejewski, & Rosenheck, 2001), substance abuse (Kessler et al., 1995), dissociative disorders (Hornstein & Putnam, 1992), borderline personality disorder (Yen et al., 2002), as well as other health problems (Dube, Felitti, Dong, Giles, & Anda, 2003).

Despite the limitations in neurobiological understanding of PTSD, scientists think that the symptoms of PTSD and other trauma- and stressor-related disorders may be represented in the behavioural manifestation of stress-induced changes in the structure and function of the brain. Until 20 years ago, there had been no brain imaging studies performed in patients who had PTSD or other stress-related psychiatric disorders (Bremner, 2007). During the past decade however, there had been a proliferation of research relying on brain imaging to assess the impact of traumatic stress on the brain (Garfinkel & Liberzon, 2009; Liberzon & Martis, 2006; Robinson & Shergill, 2011; Shin, Rauch, & Pitman, 2006; Villarreal & King, 2004). All of these studies have indicated structural changes within the brain (for review see Bremner, 2007; Shin et al., 2006). Changes relating specifically to the amygdala, hippocampus, and medial prefrontal cortex, including the anterior cingulate cortex have been observed (Figure 0.7). Apart from the structural changes associated with PTSD, functional neuroimaging studies in PTSD have also shown to be consistent with dysfunction in three regions of the brain, namely the amygdala, medial prefrontal cortex, and the hippocampus (Liberzon et al., 2003; Liberzon & Martis, 2006). The neuroimaging research provided evidence that led to the hypothesis that PTSD represents a failure of the medial prefrontal-anterior cingulate networks to regulate amygdala activity, resulting in hyper-reactivity to threat (Bisson, 2009; Bremner, 2007; Shin et al., 2006).



*Figure 0.7. Brain areas implicated in PTSD*

Adapted from “The neurobiology of posttraumatic stress disorder,” by J.L. Bisson, 2009, *Psychiatry*, 8(8), p. 288. Copyright 2009 by Elsevier Ltd.

In the following paragraphs, the relationship between the various brain regions that are critically involved in the fear response (e.g., the amygdala, medial prefrontal cortex, and hippocampus), as well as the neurotransmitter/neurohormone system (e.g., the hypothalamic-pituitary-adrenal (HPA) axis that is known to be dysregulated in many individuals with PTSD), will be discussed. Extensive attention will be given to the role of the critical aspect of neural context and situational context within brain functioning, because cognitive operations are only possible through the inter relatedness of the different brain areas. Thereafter an overview of the large-scale neurocircuitry models implicated in PTSD will follow. On its own, this review is largely limited and does not reflect the complete vast complexity of the neurobiological responses to danger or the neurobiological dysregulation associated with PTSD.

### **2.6.1 Abnormal stress response: Dysregulated energy systems.**

Although the view of Walter Cannon and Hans Selye that the physiological role of the stress response is to regulate the autonomic, neuroendocrine, as well as the immune responses to potential homeostatic threats is still generally accepted, Myers, McKlveen, and Herman (2014), Dallman et al. (2006), and Nederhof and Schmidt (2012) suggest that the primary role of the stress response is to mobilize energy to encourage survival instead of only maintaining homeostatic systems at the level before the challenge occurred. This emerging concept proposes that an individual's response to acute, as well as chronic stress, must be considered adaptive to a certain point, while preparing the individual for current and future demands. Myers et al. (2014) thus defines stress as “a stimulus that mobilizes energetic systems to respond to an ongoing or anticipated challenge” (p. 181). According to this definition an individual's response to stress involves purposeful engagement of various interacting central stress regulatory systems to provide energy for the individual. This activation may occur either as a consequence of, or in anticipation of, a threat (Myers, McKlveen, & Herman, 2012).

The neuroendocrinological response to stress comprises a well-regulated, temporal process that involves the integration of sensory information from numerous modalities to quickly activate or inhibit glucocorticoid secretions. This activity needs to be tightly regulated throughout the system from cellular to behavioural levels, because in response to stress, glucocorticoid secretion enhances an individual adaptation to environmental demands, thereby helping to obtain the necessary energetic requirements (Herman et al., 2003). This process demands the integration of multiple systems and activates vital limbic-neuroendocrine circuits (HPA axis), which coordinates the individuals physiological and behavioural outputs. In the event where this energetic drive does not appropriately correspond to the environmental challenge, or when an individual chronically activates these systems, risk factors arise for the development of numerous stress-related pathologies, for example PTSD. Myers et al. (2014)

therefore believe that the glucocorticoid-mediated energetic drive provides an individual with an adaptive capacity to overcome challenges in the environment. When conditions of elevated environmental stress occur, which could be either acute or chronic, the cost of repeated or excessive activation of the energetic system however, may compromise the individual's performance. Stress promotes adaptation, but at a cost. An individual's response to stress cause a redistribution of energy resources to meet immediate or perceived needs through a process by which glucocorticoid signalling affects cellular functions, neurocircuits, and the individual's behaviour. This glucocorticoid signal activates the physiological system and continue to adjust until the adaptive cost becomes more than the individual's adaptive capacity. According to Myers et al. (2014) this adaptive capacity is the degree to which an individual deploys energy mobilising systems (e.g., the HPA axis) to adequately meet the current or expected demands. In other words, energy depletion increases adaptive cost. When adaptive cost exceeds adaptive capacity, a breaking point is reached in the process of adaptation resulting in the emergence of psychopathologies such as PTSD. The appropriate regulatory control of the hypothalamic–pituitary–adrenocortical (HPA) axis stress response is thus vital for an individual's health and survival (Herman et al., 2003). Southwick et al. (2005) and Yehuda (2009) found changes in the functioning of the HPA axis in patients diagnosed with PTSD.

The biologic changes noticed in individuals who suffer from PTSD do not equally mirror those associated with other types of stress (Figure 0.8). For example, cortisol levels have been found to be lower than what is normally expected within individuals suffering from PTSD, even decades after a traumatic event had occur (Yehuda, 2009; Yehuda, Kahana, et al., 1995; Yehuda et al., 2012). Paradoxically, Baker et al. (1999) found that the levels of corticotropin-releasing factor in cerebrospinal fluid also seem to be increased. This pattern is different from those associated with brief and sustained periods of stress and with that of major depression,

which are most commonly associated with increased levels of both cortisol and corticotropin-releasing factor (Figure 0.8).

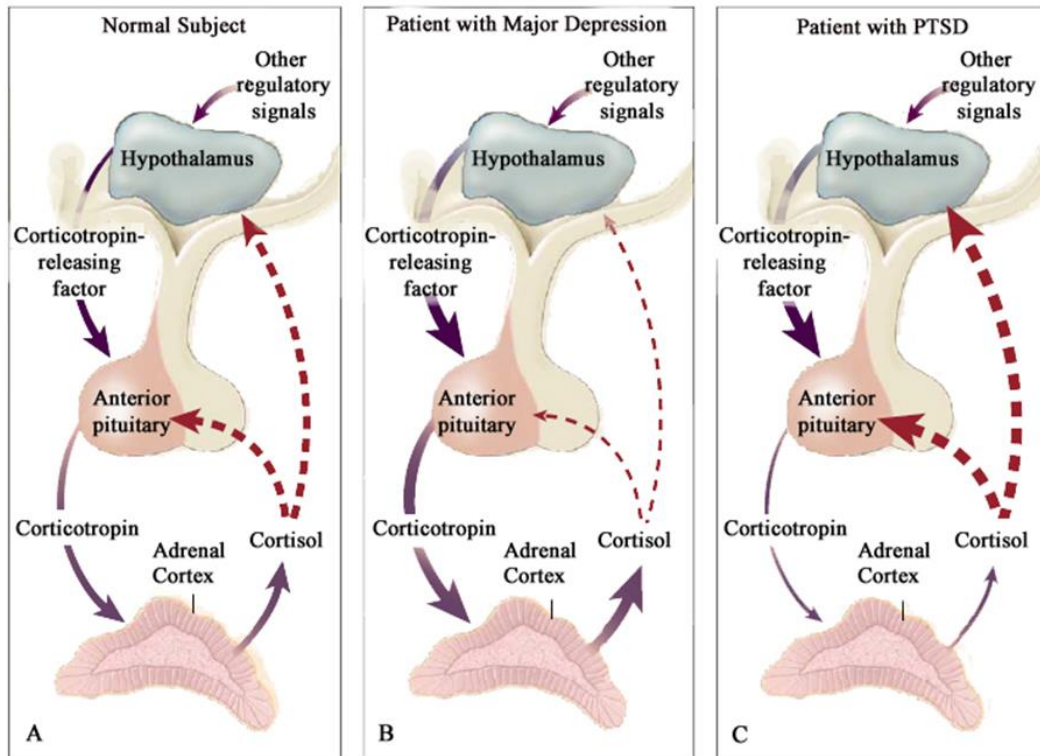


Figure 0.8. Neurohormone response to stress

Response to stress in a Normal Subject (Panel A), a patient with Major Depressive Disorder (Panel B), and a patient with PTSD (Panel C).

In normal subjects (Panel A) and in patients with major depression (Panel B), brief or sustained periods of stress are typically associated with increased levels of both cortisol and corticotropin-releasing factor. In each panel the thickness of the interconnecting arrows denotes the magnitude of the biologic response. Corticotropin-releasing factor stimulates the production of corticotropin, which in turn stimulates the production of cortisol. Cortisol inhibits the release of corticotropin from the pituitary and the release of corticotropin-releasing factor from the hypothalamus. It is also responsible for the containment of many stress-activated biologic reactions.

In patients with PTSD (Panel C), levels of cortisol are low and levels of corticotropin-releasing factor are high. In addition, the sensitivity of the negative-feedback system of the hypothalamic–pituitary–adrenal axis is increased in patients with PTSD rather than decreased, as often occurs in patients with major depression. Reprinted from “Post-traumatic stress disorder,” by R. Yehuda, 2002b, *The New England Journal of Medicine*, 346(2), p. 112. Copyright 2002 by the Massachusetts Medical Society.



The sensitivity of the negative-feedback system of the HPA axis is increased within PTSD, as reflected by the exaggerated suppression of cortisol in response to dexamethasone administration (Goenjian et al., 1996; Yehuda et al., 1993) as well as the increased sensitivity of lymphocyte glucocorticoid receptors (Yehuda, Boisoneau, Lowy, & Giller, 1995).

Yehuda (2002b) supports the psychological and biological data that presents the hypothesis that the onset of PTSD is facilitated by a failure to contain the biologic stress response at the time of the trauma resulting in an outpouring of changes that lead to intrusive recollections of the traumatic event, avoidance of reminders of the event, and symptoms of hyperarousal. This breakdown may illustrate an alternative path to the normal process of adaptation and recovery following a traumatic event. Yehuda (2002b) refers to research with patients in whom PTSD or symptoms of PTSD developed a diminishing increase in cortisol levels in the immediate aftermath of a traumatic event. This could be attributed to the patient being previously exposed to a traumatic event or other risk factors. Patients with PTSD could therefore have more activation of the sympathetic nervous system. Consequently, decreased levels of cortisol at the time of a traumatic event could delay the availability of norepinephrine to the synapses in both the periphery and the brain, resulting in an impaired consolidation of the memory of the traumatic event. Such an impairment would most likely aid the development of altered perceptions and thoughts in the aftermath of the traumatic episode. Thoughts relating to the perception of danger or the ability to cope with a threat will thus be severely affected, delaying the recovery by leading to an inability to subdue fearful responses. This reaction will further strengthen flawed cognitive responses to trauma and fear, maintaining the symptoms of PTSD and triggering an array of secondary biologic changes (Yehuda, 2002a). During normal situations, it is beneficial to have mechanisms that enable us to rely on previous experiences to avoid current or future risks. However, in PTSD these mechanisms are pathologically

hyperactive, resulting in the inhibition, rather than the support of adaptive reactions (Hašto et al., 2013).

In PTSD, the most important biological dysregulation seems to be associated with an abnormal stress response implicating the HPA axis. This view is congruent with the idea, supported by Southwick et al. (2005) and Yehuda (2009), of an enhanced negative feedback where the primary deficit involves an increased activation of glucocorticoid receptors at numerous sites across the HPA axis. It appears that individuals who suffer from PTSD are not able to put their traumatic experiences in the past, because the biological stress responses they experienced have not yet been resolved. Yehuda (2009) believes that this could explain the re-experiencing of undesirable reminders as retraumatisations that extend the biological dysregulation in individuals with PTSD. In the past numerous studies focused on the size of important brain areas coupled to PTSD. Currently, neuropsychologists are shifting their attention toward investigating the function rather than the shape of these key areas. It appears that changes in function, including changes in the genes, may add to the development and maintenance of this crippling stress disorder (Yehuda, 2002a). An overview regarding the functional and structural alterations found in key brain areas commonly associated with PTSD, in particular the amygdala, medial prefrontal cortex, and hippocampus, will be provided in the subsequent paragraphs.

### **2.6.2 Amygdala.**

As previously indicated, threat activates the amygdala. The amygdala is involved in the assessment of threat-related stimuli and/or biologically relevant ambiguity and is necessary for the process of fear conditioning. Individuals with PTSD presents with hypervigilance in potential threatening environments and have shown relatively heightened acquisition of conditioned fear in laboratory studies. This have led researchers to believe that the amygdala is hyper responsive in individuals with PTSD (Liberzon et al., 1999; Rauch et al., 1996; Shin

et al., 2006) and that this increased activity of the amygdala may be associated with symptom severity in PTSD (Shin et al., 2004).

The functioning of the amygdala is enhanced by high levels of catecholamines and glucocorticoids that are released during stress, which promotes fear conditioning as well as the consolidation of emotionally relevant memories (Southwick et al., 2005). Equally important is the amygdala's ability to significantly influence the neurochemical environment of the prefrontal cortex. Even the slightest psychological stressors may result in high levels of catecholamine release within the prefrontal cortex, including an increase in circulating cortisol (Goldstein, Rasmusson, Bunney, & Roth, 1996). Lesions to the amygdala cause these neurochemical responses to be terminated (Goldstein et al., 1996). However, despite their effect in the amygdala, high levels of catecholamines and glucocorticoids can also considerably impair the cognitive functioning of the prefrontal cortex (Southwick et al., 2005). The prefrontal cortex is known to be responsible for the regulation of behaviour, thought, and affect and plays a significant role in the planning, guidance, and organisation of behaviour (Funahashi, 2017; Miller, 2000). Therefore, damage to the prefrontal cortex may result in the inability to have voluntary control over behaviour, increased motor activity, impaired attention, and a reduced ability to inhibit distracting stimuli (Southwick et al., 2005).

### **2.6.3 Medial prefrontal cortex.**

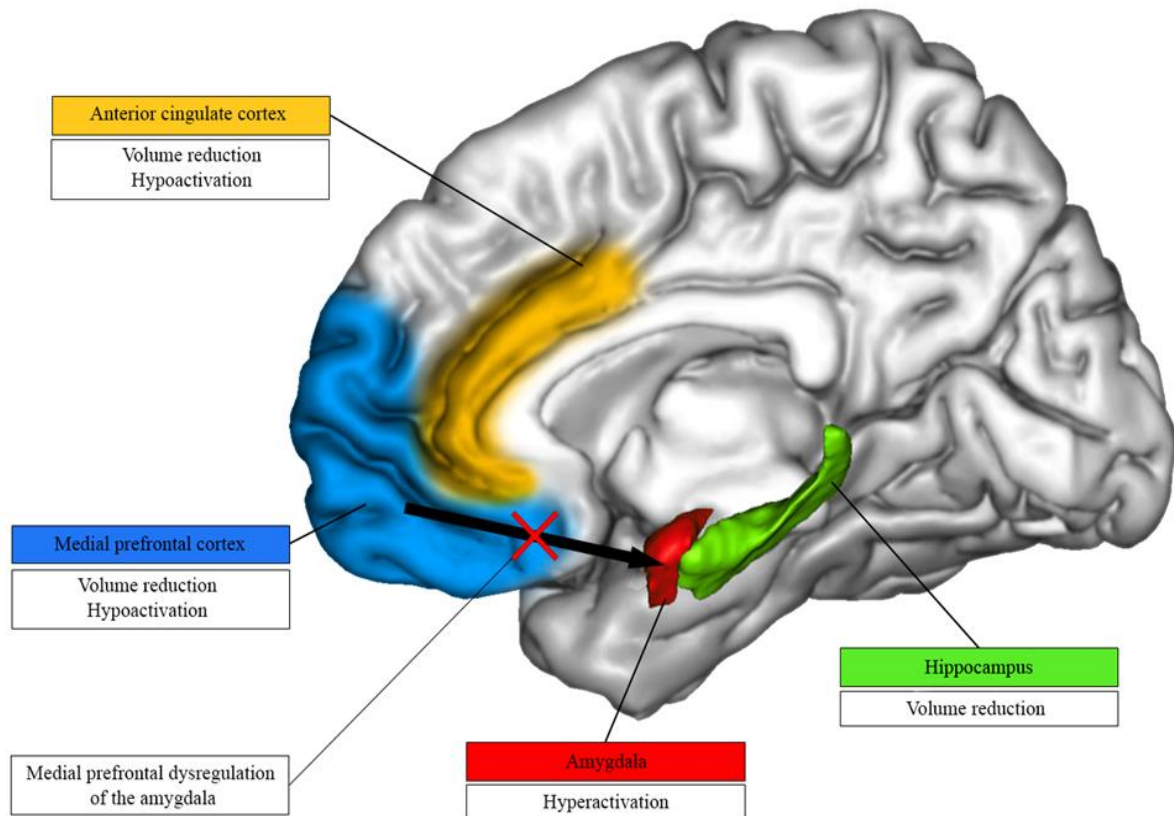
The second brain region of interest involved in the pathophysiology of PTSD is the medial prefrontal cortex (mPFC), which includes the anterior cingulate cortex (ACC), subcallosal cortex, and medial frontal gyrus. Shin et al. (2006) confirms that individuals with PTSD exhibit persistent inappropriate fear responses in daily life and diminished extinction of conditioned fear in the laboratory. These responses led Shin et al. (2006) to hypothesise that the medial prefrontal cortex may be impaired in individuals suffering from PTSD. Shin et al. (2006) list studies that report that there is not only a reduction in anterior cingulate volumes,

but also shape differences in the anterior cingulate in individuals with PTSD. Kim and colleagues have demonstrated that there are new non-invasive neuroimaging methods such as diffusion tensor imaging (DTI) that provide evidence that the white matter structural integrity of the cingulum bundle is compromised in PTSD patients compared to healthy individuals (Kim et al., 2011). Lower N-acetylaspartate (NAA)/creatinine ratios in the pregenual anterior cingulate cortex have been found, suggesting a possibility of decreased neuronal integrity in that region. Functional neuroimaging studies found further consistent evidence suggesting a decrease and/or failure to activate the medial prefrontal cortex including the anterior cingulate cortex and medial frontal gyrus (Shin et al., 2006). They furthermore, report that neuroimaging studies have showed reduced cortical volumes and neural integrity, together with decreased function in the medial prefrontal structures of the brain. Decreased activity in the medial prefrontal cortex has also been found to be associated with symptom severity in PTSD (Williams et al., 2006), and, importantly, has been negatively linked with amygdala activation in response to threat (Shin et al., 2005), supporting the hypothesis that there is a disconnect in the normal modulation of the amygdala by the medial prefrontal cortex.

#### **2.6.4 Hippocampus.**

The third region of interest is the hippocampus. This brain region is involved in explicit memory processing and in the encoding of context during fear conditioning. In his meta-study Bremner (2007) reports on numerous studies that found that, individuals suffering from PTSD exhibited deficits in verbal declarative memory. This can be contributed to changes in the volume of both the left and the right sides of the hippocampus. Other studies of PTSD also found smaller hippocampal volumes or reductions in N-acetylaspartate (NAA), a marker of neural integrity (Bremner, 2007). Bremner (2007) concluded that stress-induced hippocampal dysfunction may mediate many of the symptoms of PTSD that are related to memory dysfunction. Shin et al. (2006) mentions studies where greater PTSD symptom severity has

been associated with smaller hippocampal/parahippocampal blood flow. Figure 0.9 below provides an overview of the structural and functional changes of the amygdala, medial prefrontal cortex, anterior cingulate cortex, and the hippocampus as associated with PTSD.



*Figure 0.9. Structural and functional changes associated with PTSD relevant brain areas*

The arrow represents the disconnect in the normal modulation of the amygdala by the medial prefrontal cortex. Adapted from *Posttraumatic Stress Disorder*, In Center, n.d., Retrieved June 11, 2018, from <http://mindfulhealthcenter.com/project/post-traumatic-stress-disorder>. Copyright 2018 by Mindful Health Centre.

## 2.7 Neural networks.

Over the past few years there has been a change in the understanding of how dysfunction of cognitive and emotional regulation processes, which rely on distributed brain regions stretching over multiple lobes, contribute to major psychopathologies, including PTSD. The idea that cognition results from the interaction of large-scale neural networks, has been present in different forms throughout the history of neuroscience (Bressler, 1995; 2002). By

examining the parallel architecture within the brain, it provides an intricate view of system processing by revealing that each brain area may influence various other brain areas, through direct or indirect connection (Bressler & McIntosh, 2007). Research on the link between large-scale brain networks and psychopathology over the past few years seem to indicate that a greater unlikelihood exists that the dysfunction of cognitive and psychological processes that is associated with psychiatric disorders, like PTSD, could be attributed to dysfunction in structure or function in one individual brain area. That is, a brain area can only contribute to a cognitive operation through the interactions that it has with other areas to which it is connected. In other words, a brain area plays a specialized role in any cognitive function by its unique position within the overall connectional framework of the brain (Bressler & McIntosh, 2007). This does not necessarily mean that for the same brain area to be involved in many different functions, that it exercises the same functional role in each. On the contrary, different cognitive functions appear to be associated with different neural contexts, and individual areas may contribute in a differentially specialized manner within each neural context. An example would be the way in which the frontal cortical areas typically interact with the parietal cortical areas to perform central executive functions (Collette & Van der Linden, 2002). The frontal or parietal areas may be considered as playing a specialized role in each function based on their contribution within the unique neural context that is associated with that function. Therefore, this researcher aligns himself with Bressler and McIntosh (2007), who views neural context (defined as “the local processing environment of a given neural element that is created by modulatory influences from other neural elements” (Bressler & McIntosh, 2007, p. 403) as a general effect that modulates the processing which takes place in any part of the brain. Neural context is only an indication of the context that arises within the brain because of the interplay between the different neural elements.

Bressler and McIntosh (2007) further distinguish situational context apart from neural context as a “related form of context” (p. 404). Situational context, according to Bressler and McIntosh (2007), serves as many interrelated environmental factors, which includes the aspects of the sensory scenes and the related responses demanded by both the external and internal milieus. When researchers examine “contextual effects” on the brain, they have situational contexts in mind. Under normal circumstances, neural context is dependent upon the situational context. In the environment where people must survive, there is a high degree of structural complexity. There is therefore a high degree of uncertainty in individuals’ perceptual-motor interactions with the environment. People are constantly uncertain about the state of their environment, because of the magnitude of information that must be processed to set the most appropriate course of action. The ability to use and manipulate stimuli coming from the individual’s context, can greatly reduce uncertainty and thereby improving the individual’s interactions with the environment, providing a significant advantage to the person’s survival.

Bressler and McIntosh (2007) found that situational context affects all types of cognitive function, because of the complexity of the environment in which the individual lives. The types of cognitive functions mentioned by Bressler and McIntosh (2007) includes sensation, perception, emotion, memory, planning, decision-making, and action generation. They conclude that just as situational context can have effects at various scales and across various behaviours, so too is neural context expected across all spatial and temporal scales in the brain and across all behaviours.

Therefore, the neural context of processing in any area is firstly dependent on its connectivity with other brain areas, and secondly on the processes occurring in those areas. This researcher joins Bressler and McIntosh (2007) in their view that the prefrontal cortex contributes to working memory by forming the neural context of distributed sensory and motor networks through the regulatory influences it exerts on them. The prefrontal cortex’s own

neural context is further shaped by the regulatory influences it receives back from the sensory and motor networks. During this process, the prefrontal cortex collaborates with other brain areas to support different aspects of the situational context by interacting for example with the basal ganglia to maintain cross-temporal context. The prefrontal cortex's neural context, by its different interactions, depends on the situational context. With the above mentioned in mind, the logical progression of this study would now be to investigate the neurocircuitry models relevant to PTSD.

### **2.7.1 Neurocircuitry models of PTSD: Explaining interacting brain networks.**

As already mentioned, the past two decades brought forth many studies investigating the structural and functional neuroanatomy of posttraumatic stress disorder (PTSD). These findings are often inconsistent, thus challenging traditional neurocircuitry models of PTSD. A broad range of experimental paradigms, including those related to symptom provocation, emotional processing, and cognitive activation have been used to assess the neural underpinnings of PTSD. Three neurocircuitry models of PTSD explaining the interaction between the different brain networks will be reviewed in the following paragraphs.

#### ***2.7.1.1 The traditional neurocircuitry model of PTSD: The Hypersensitivity-to-Threat Model.***

Alterations in regional activation are thought to underlie behavioural, cognitive, or emotional symptomatology. The traditional neurocircuitry model of PTSD, first proposed by Rauch, Shin, Whalen, and Pitman in 1998, suggested hypoactivation of the medial prefrontal cortex (including the anterior cingulate cortex (ACC), ventromedial prefrontal cortex, subcallosal cortex, and orbitofrontal cortex) resulting in an inability to effectively control attention and response to trauma-related stimuli. Combined with this loss of top-down inhibitory control, amygdalar hyperresponsivity promotes the vivid nature of trauma



recollections and symptoms of hyperarousal (Shin et al., 2006). The model further proposes that abnormal functioning of the hippocampus underlies PTSD-related deficits in learning and memory (e.g., the inability to extinguish a fear response) (Patel, Spreng, Shin, & Girard, 2012).

The traditional neurocircuitry model of Rauch and colleagues has been useful in the understanding of PTSD. It has however been found to contain inconsistencies. The level of hippocampal activation in a few studies for example varies—some report lower, while others report greater hippocampal activation in response to both threat-related and non-threat-related stimuli. Patel et al. (2012) furthermore refers to studies that report less activation while others report greater activation in the rostral anterior cingulate cortex. Another factor mentioned by Patel et al. (2012), is that the traditional neurocircuitry model might be constrained by its focus on threat. That is, while some PTSD symptoms may stem from deficits in threat-related processing, other symptoms (e.g., emotional numbing and avoidance behaviours) are unexplained by this model.

### ***2.7.1.2 Liberzon and Garfinkel's Contextualization Model.***

In Liberzon and Garfinkel's (2009) contextualization model, they emphasize the role of the medial prefrontal cortex in contextualization. During this process, stimuli in different situational contexts are interpreted, represented, and used to guide behavioural action. Since a number of processes that rely on contextualization, including extinction, emotion regulation, social cognition, and self-referential processing (below Figure 0.10), all implicate the medial prefrontal cortex. Liberzon and Garfinkel (2009) proposed that the altered functioning of this region could explain a number of problems (e.g., re-experiencing phenomena and emotional numbing) that are characteristic of PTSD. They further emphasise the importance and complex roles of the medial prefrontal cortex afforded by its high connectivity with other areas such as the anterior insula.

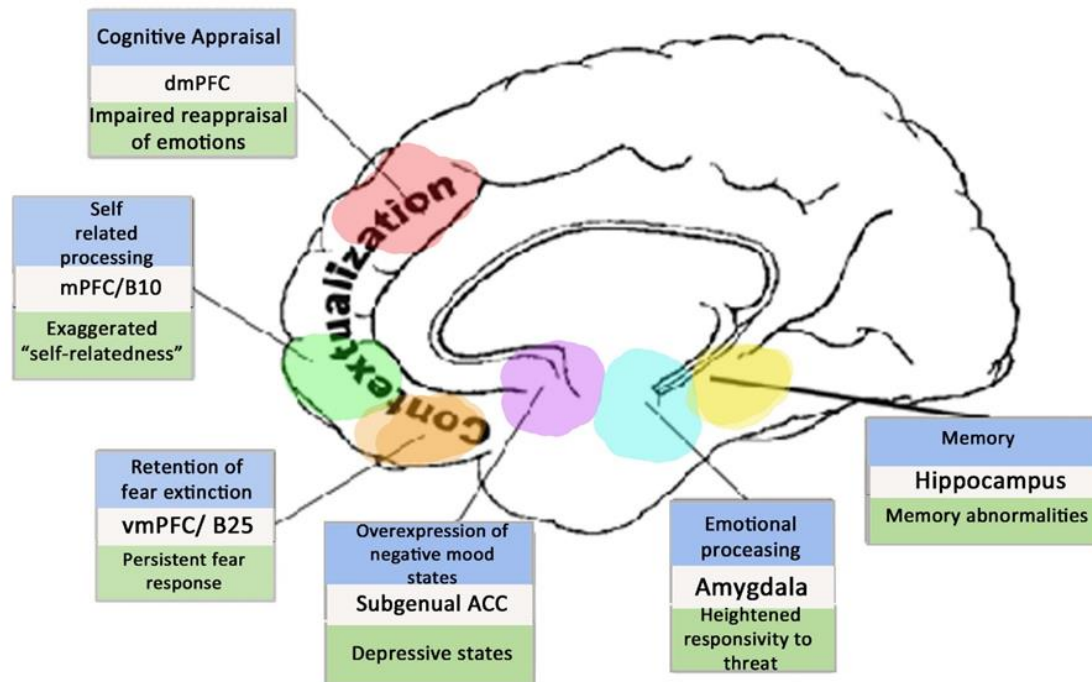


Figure 0.10. Neural regions underlying core processes and PTSD symptomatology

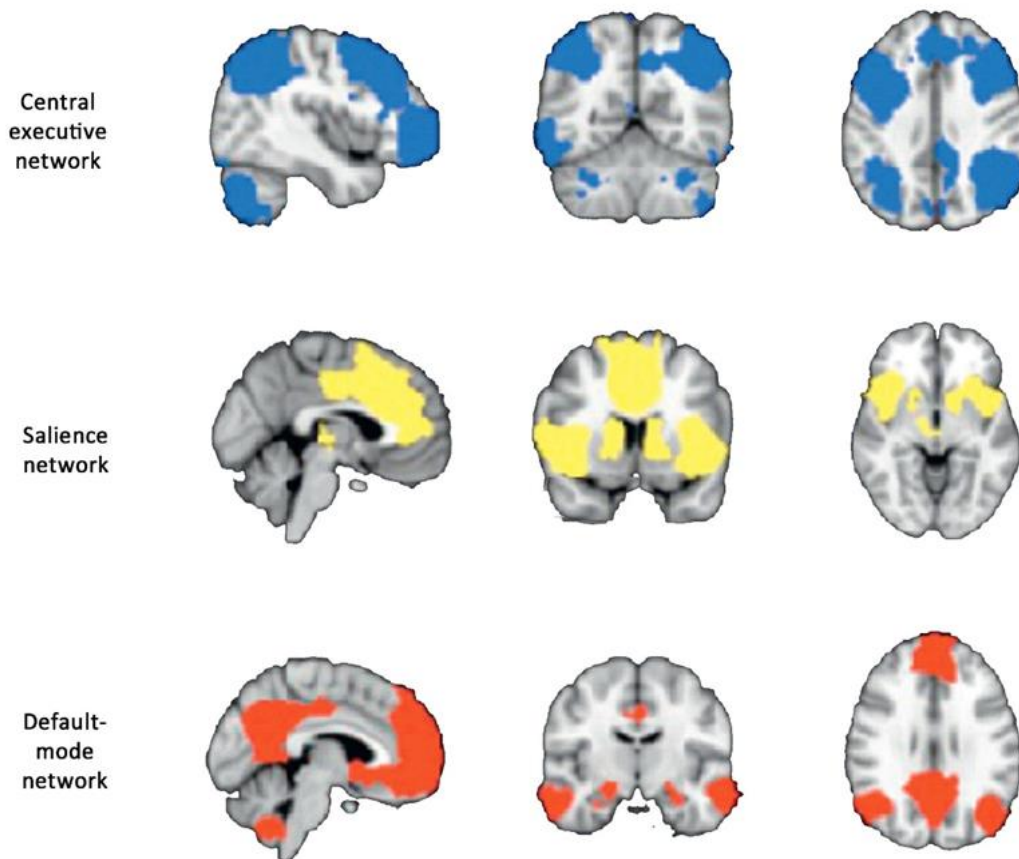
These include the dorsal medial prefrontal cortex (dmPFC), medial prefrontal cortex (mPFC), ventral medial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), amygdala, and hippocampus. Adapted from *Functional neuroimaging in post-traumatic stress disorder* (p. 302), by I. Liberzon and S.N. Garfinkel, 2009, New York, United States of America: Human Press.

### 2.7.1.3 Menon's Unifying Triple Network Model.

The two models reviewed thus far have implicated the insula, the amygdala, and hippocampus, as well as the medial prefrontal regions in the pathophysiology of PTSD. To facilitate the characterization and interpretation of reliable brain regions, Patel et al. (2012) adopted the unifying triple-network framework of psychopathology put forth by Menon (2011).

Bressler and McIntosh (2007) provided evidence supporting human behaviour and cognition as an emergent property of interacting, large-scale brain networks. Menon (2011) has proposed that a broad range of neurological and psychiatric disorders can be understood by evaluating dysfunction in three core neurocognitive networks: (1) The default mode network

(DMN), (2) the frontoparietal central executive network (CEN), and (3) the salience network (SN) (Figure 0.11). Patel et al. (2012) provide the following summary of these networks: The default network comprises a set of interconnected brain regions, including the medial prefrontal cortex, posterior cingulate cortex, lateral and medial temporal lobes, and posterior inferior parietal lobule that are suppressed during externally oriented, attention-demanding tasks relative to when participants are at rest. In contrast, activation of the default network has been linked to various processes of internal mentation, such as autobiographical memory, self-referential thinking, and social cognition. The central executive network is anchored in the dorsolateral prefrontal cortex and anterior inferior parietal lobule, and subserves processes related to working memory and attentional control (Menon, 2011). The third network, or salience network is anchored in the fronto-insular cortex and dorsal ACC, with extensive connectivity to subcortical regions including the amygdala, thalamus, ventral striatopallidum, and substantia nigra/ventral tegmental area. Patel et al. (2012) concludes their summary of Menon's model by stating that together, these regions subserve processes related to autonomic and emotion regulation, conflict monitoring, and reward-processing.



*Figure 0.11. Three core neurocognitive networks*

The fronto-parietal CEN (shown in blue) which is anchored in the dorsolateral prefrontal cortex and the posterior parietal cortex plays an important role in working memory and attention. The SN (shown in yellow) is important for detection and mapping of salient external inputs and internal brain events. The SN is anchored in the fronto-insular cortex and dorsal anterior cingulate cortex and features extensive connectivity with subcortical and limbic structures involved in reward and motivation. The DMN (shown in red) which is anchored in the posterior cingulate cortex and medial prefrontal cortex is important for self-referential mental activity. Adapted from “Large-scale brain networks and psychopathology: A unifying triple network model,” by V. Menon, 2011, *Trends in Cognitive Sciences*, 15(10), p. 494. Copyright 2011 by Elsevier Ltd.

Patel et al. (2012) describe the working of these three networks as normally interacting with one another in a dynamic and complementary manner. Regions of the central executive and salience networks become engaged (i.e., show greater activation) during stimulus-oriented cognitive and affective information processing, whereas the default network disengages when demands for external attention are high and internally focused processes are minimized. Aberrant organisation or dysfunction in any part of these networks can lead to dysfunction in

the remaining networks and a unique constellation of clinical symptoms (Menon, 2011). Given the comprehensive scope of Menon's model, Patel et al. (2012) applied and adapted the triple-network approach by harnessing the traditional neurocircuitry model of PTSD as a guiding framework in their understanding of the neurobiological dysfunction in PTSD.

### **2.7.2 Integrating the neurocircuitry models of PTSD.**

The three neurocircuitry models of PTSD does not exclude one another. There is ample evidence that the Traditional Neurocircuitry Model, Liberzon and Garfinkel's Contextualization Model, and Menon's Unifying Triple Network Model are all helpful in understanding the pathophysiology of PTSD. These models consistently confirmed the diminished activation of the medial prefrontal cortex and exaggerated activity in the limbic regions, such as the amygdala, as being the hallmark features of PTSD. The triple network model suggests that a loss of prefrontal inhibition is the result of abnormal functioning of the anterior insula. These models demonstrated exaggerated activity in the anterior insula as the neural locus for heightened interoceptive and emotional awareness. This perspective is in line with the belief in an overactive salient network because of enhanced salience signalling from the amygdala in PTSD. The triple network model posits that the anterior insula acts as a critical hub that facilitates access to other large-scale neurocognitive networks. Patel et al. (2012) found further that the abnormal functioning of the anterior insula in PTSD might disrupt the control signals that facilitate proper engagement of the networks responsible for higher order cognitive functions that are reliant on the prefrontal cortex and that are involved in normal fear extinction and emotion regulation.

As reported by Patel et al. (2012), the areas of the traditional neurocircuitry model are key nodes of the networks implicated in the triple network model. They came to this conclusion because the hyper-activation of the amygdala in PTSD is consistent with a hyper responsive salience network. Heightened activity in the temporal cortex on the other hand, interacting with

less activity in the medial prefrontal cortex might reflect functional dissociations within the default network. Abnormal connectivity between regions of the default network, according to Patel et al. (2012), is the result of deficits in self-referential processing manifesting as symptoms of depersonalisation for example. A breakdown in the functions within and between the neurocircuitry networks could lead to a unique set of PTSD symptoms. To support this statement, Patel et al. (2012) refers to the interactions between an overactive salience network and an increased suppression of the default regions could form a basis for intrusive trauma recollections and an impairment of autobiographical recall. Patel et al. (2012) lastly caution against the effects of the altered functioning of areas implicated in the neurocircuitry models. Changes in these models may have downstream effects within the context of their respective neurocognitive networks and thereby impacting a range of cognitive and affective functions in PTSD.

Patel et al. (2012) concluded that they could generally support the traditional neurocircuitry model of PTSD in terms of lower activation in medial prefrontal regions and hyperactivation of the amygdala including abnormal activation in the hippocampus, which was characterised, by hyperactivation. They finally concluded that individuals with PTSD might over-engage the salience network, while being unsuccessful in properly recruiting the central executive network, and demonstrating diverse changes in the activation of the default network.

## **2.8 Conclusions and implications of the neuroanatomy and biology of PTSD.**

The clarification of the biological changes related to PTSD has clarified the issue why some people recover from traumatic events while others do not. These changes may go a long way in explaining some of the somatic symptoms of PTSD. Individuals suffering from PTSD, decreased prefrontal cortex functioning may compromise executive functioning and impair inhibitory control of the amygdala resulting in the increase in fear-related behaviour. High-

levels of stress-induced glucocorticoids were coupled to the impairment of working memory as well as the augmentation of synaptic catecholamine levels. In the preceding paragraphs, information was presented demonstrating that high levels of stress-induced catecholamines and cortisol have significant effects on the amygdala by amplifying fear conditioning and consolidation of emotional memories. In addition to coordinating the fear response, the amygdala also regulates the neurochemical environment of the prefrontal cortex. Increased disinhibition by the prefrontal cortex, may leave the amygdala in an activated state. In the presence of impaired frontal lobe executive functioning, the activation of the amygdala may trigger the release of the neurotransmitters, norepinephrine, dopamine, and acetylcholine. Thus, leading to an impaired capacity for rational problem-solving and rational influence on behaviour and thought, as well as the amplifying of the startle response, enhancing fear-conditioning, increase consolidation of emotional memory, and raise vigilance, insomnia, impulsivity, intrusive memories, flashbacks, and other fear-related behaviours (Southwick et al., 2005).

Yehuda (2002b) refers to neuroanatomical studies, which identified adjustments in two of the three major brain structures mentioned above, namely the amygdala and the hippocampus in patients with PTSD. In addition, positron-emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies indicated an increase in the reactivity of the amygdala and anterior paralimbic region to trauma-related stimuli (Liberzon et al., 1999; Rauch et al., 2000), whereas a decrease in the reactivity of the anterior cingulate and orbitofrontal regions of the prefrontal cortex were found (Shin et al., 1999). These areas of the brain are involved in fear responses. Intrusive recollections and other cognitive problems associated with PTSD may be the result of differences in hippocampal function and in memory processes thought to be dependent on the hippocampus.

Despite the relationship between neurobiology and behaviour being exceptionally complex, research over the past 30 years has made it increasingly clearer that the dysregulation of various neurobiological systems plays an important role in the pathophysiology of PTSD. During situations of threat, parallel activation of different brain regions and neurotransmitter systems enables an individual to assess and appropriately respond to situations of potential danger. Short term, this process serves a protective role and facilitates fleeing from or actively confronting danger. However, enduring neurobiological responses to threat and stress may prove to be maladaptive and contribute to the development of PTSD, in some individuals.

This ends the focus of the neuroanatomy and biology of PTSD. In the subsequent section, the researcher will address the prevalence of PTSD in the world in general and South Africa in particular.

## **2.9 Prevalence of PTSD**

The DSM-5 defines a traumatic event (TE) as exposure to threatened death, serious injury, or sexual violence. Such exposure may occur directly or indirectly by witnessing the event, learning of the event occurring to a loved one, or repeated confrontation with aversive details of such event (American Psychiatric Association, 2013). Exposure to TEs is a prerequisite for the diagnosis of PTSD and is also associated with a wide range of other adverse mental and physical health outcomes. In the following paragraphs the researcher will endeavour to understand who is at risk for exposure to TEs.

Benjet et al. (2016) conducted 26 surveys in 24 countries to estimate the prevalence, and examine the sociodemographic correlates, of a wide range of TEs. Members of the general public participated in the World Health Organisation's (WHO) World Mental Health (WMH) Surveys (Kessler & Üstün, 2008) where they reported on their exposure to 29 traumatic event types. A summary of their findings depicting traumatic event exposure worldwide appears in



Figure 0.12. Exposure varied by country of residence, sociodemographic characteristics, and history of previous traumatic event exposure. In addition, it was found that traumatic event exposure does not occur randomly in the populations consulted. It is furthermore insightful that over 70% of participants reported a lifetime traumatic event of which 30,5% were exposed to four or more traumatic events. Five types of traumatic events, namely: Witnessing death or serious injury, the unexpected death of a loved one, being assaulted, being in a life-threatening motor vehicle accident, and experience a life-threatening illness or injury, were responsible for over half of all exposures. The most consistent protective factor was found to be in a marital relationship. Contact with interpersonal violence had the strongest association with subsequent traumatic events. Rather than identifying a particular group of vulnerable individuals Benjet et al. (2016) opted to give a nuanced picture in which individual life circumstances of those subjected to traumatic events were exposed. The results from the WMH Surveys finally concluded that traumatic event exposure is very common and therefore suggests that experiencing a traumatic event is not outside the normal range of human experience.

The first national representative study of mental disorders in Africa, using the random event method opposed to the worst event method of identifying potentially traumatic events (PTEs), was done in 2004 by Williams and colleagues. They reported on the prevalence of trauma exposure and risk of PTSD associated with PTEs utilizing the South African Stress and Health Survey (SASH). The SASH is a national representative survey of South African adults using the WHO's Composite International Diagnostic Interview (CIDI) to assess exposure to trauma and the presence of mental disorders.

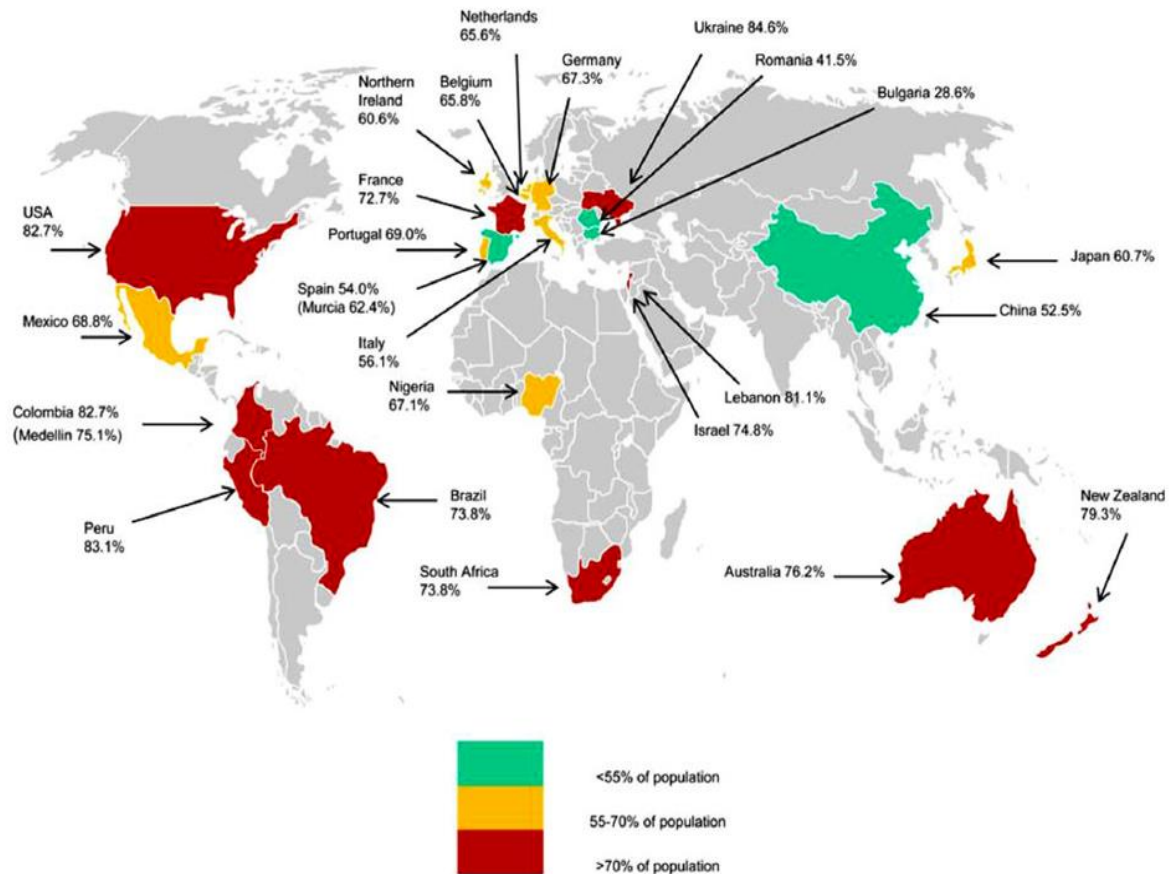


Figure 0.12. Prevalence of exposure to traumatic events in 24 countries

Reprinted from “The epidemiology of traumatic event exposure worldwide: Results from the “World Mental Health Survey Consortium,” by C. Benjet et al., 2016, *Psychological Medicine*, 46(2), p. 334. Copyright 2015 by Cambridge University Press.

The WHO CIDI identifies up to 29 different types of traumatic events categorised into eight classes: War events (combat, relief worker in a war zone, civilian in a war zone, civilian in a region of terror, refugee and purposely injured, tortured or killed someone); physical violence (physical abuse by caregiver, physical assault by spouse or romantic partner, physical assault by someone else, assaulted or threatened with a weapon, and kidnapped); sexual violence (raped, sexually assaulted and stalked); accidents (toxic chemical exposure, motor vehicle accidents, other life-threatening accident, natural disaster, man-made disaster and a life-threatening illness); unexpected death of a loved one; network events involving others in one’s social network (having a child with a serious illness, traumatic event occurring to a loved

one and accidentally causing serious injury or death); and witnessing trauma (witnessing a death, seeing a dead body, or someone seriously hurt, seeing atrocities, and witnessing domestic violence). The final category included other trauma comprising other traumatic events not included in the CIDI list and private events that respondents did not report because of embarrassment (Atwoli et al., 2013). This study found that reported lifetime traumatic event prevalence rate for South Africa is 73.8%, similar to that for Brazil. This is however higher than surveys conducted in Europe or Japan (prevalence rate of 54-64%) and Nigeria (prevalence rate 67.1%), but lower than the USA (prevalence rate 82.7%), Australia (prevalence rate 76.2%), and New-Zeeland (prevalence rate 79.3%) (Atwoli, Stein, Koenen, & Mclaughlin, 2015).

This study found that the most common traumatic events experienced in South Africa is the unexpected death of a loved one caused by injuries sustained through assault or motor vehicle accidents (Norman, Matzopoulos, Groenewald, & Bradshaw, 2007) and witnessing trauma occurring to others (Figure 0.13). Lifetime and 12-month prevalence rates of PTSD were found to be 2.3% (Atwoli et al., 2015) and 0.7% respectively, while the conditional prevalence of PTSD after trauma exposure was 3.5% (Atwoli et al., 2013). PTSD conditional risk after trauma exposure and probability of chronicity after PTSD onset were both highest for witnessing trauma (Figure 0.14). Inquiry done into the witnessing of infliction of pain on others and on groups such as war journalists by Feinstein, Owen, and Blair (2002), and rescue workers (ambulance personnel) during disasters (Berger et al., 2012), supports the assumption that witnessing trauma can be just as harmful, or even more than the direct experience of any traumatic event.

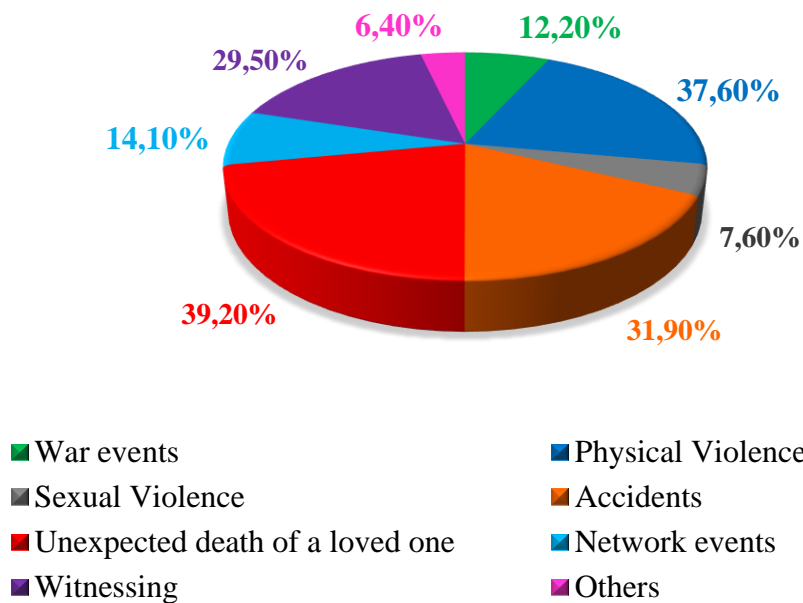


Figure 0.13. Prevalence of potential traumatic event (PTE) exposure in South Africa

Adapted from “Trauma and posttraumatic stress disorder in South Africa: Analysis from the South African Stress and Health Study,” by L. Atwoli et al., 2013, *BMC Psychiatry*, 13(1), p. 185. Copyright 2013 by Atwoli et al.

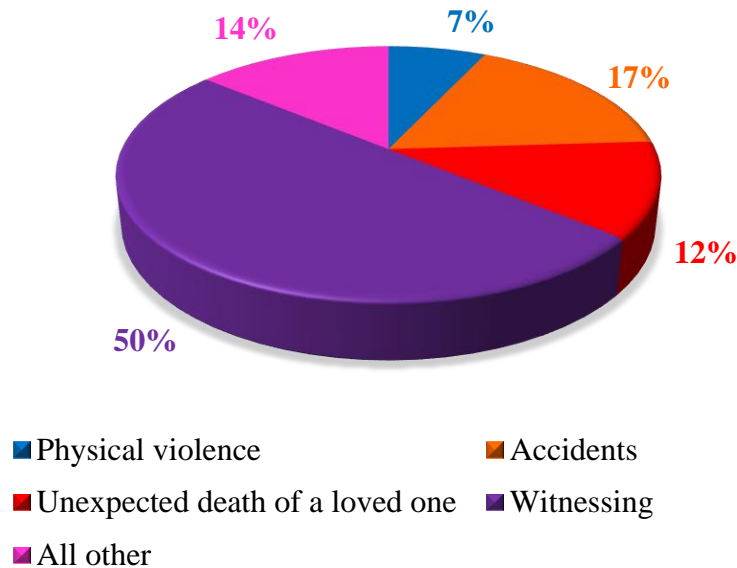


Figure 0.14. Relative PTSD burden associated with specific events in the South African populations

Reprinted from “Trauma and posttraumatic stress disorder in South Africa: Analysis from the South African Stress and Health Study,” by L. Atwoli et al., 2013, *BMC Psychiatry*, 13(1), p. 187. Copyright 2013 by Atwoli et al.

Atwoli et al. (2015) recognises the role of culture in determining mental health outcomes of traumatic event exposure. The importance of witnessing events for PTSD burden in South Africa maybe related to the cultural philosophy of *Ubuntu*, which has been described as a comprehensive ancient African world view that emphasises the values of “intense humanness, caring, sharing, respect, compassion and associated values, ensuring a happy and qualitative human community life in a spirit of family” (Broodryk, 2002, pp. 13–14).

Sociodemographic factors such as gender, age, and education were found to be unrelated to PTSD risk in South Africa (Atwoli et al., 2013). This equalizing trend can be attributed to the high levels of traumatic event exposure in communities.

The unique role of witnessing a trauma in causing PTSD in South Africa has been attributed to the history of political and criminal violence that often occurs in public settings in South Africa (Kaminer, Grimsrud, Myer, Stein, & Williams, 2008). Following the end of *apartheid* in 1994 many South Africans continued to experience high levels of criminal interpersonal violence fuelled by rapid urbanization and ongoing socio-economic disparities.

Kaminer et al. (2008) assessed a portion of the South African population to study the relative risk for PTSD associated with political, domestic, criminal, sexual, and other miscellaneous forms of assault. Their findings differed from the survey done by Williams et al. (2004), because Kaminer et al. (2008) used the worst traumatic event scenario as criteria for their study. Kaminer et al. (2008) indicated that over a third of the South African population had been exposed to some form of violence—amongst the highest rates in the world. The most common form of violence experienced by men were criminal and miscellaneous assaults, while physical abuse by an intimate partner, childhood physical abuse, and criminal assaults were most common for women. Among men, political detention and torture were the forms of violence most strongly associated with a lifetime diagnosis of PTSD, while rape had the

strongest association with PTSD among women. At a population level, criminal assault and childhood abuse were associated with the greatest number of PTSD cases among men, while intimate partner violence was associated with the greatest number of PTSD cases among women. In addition, Seedat, van Nood, Vythilingum, Stein, and Kaminer (2000) estimated that the lifetime prevalence of PTSD among adolescents in South Africa may be as high as 12% to 22%, while 99% of teenagers reported being exposed to some form of violent trauma.

Professor Lindiwe Zungu (2013) undertook a uniquely South African study where she researched the prevalence of PTSD in the South African mining industry. She consulted PTSD data submitted over five years (2006–2010) by 451 mining employees to the Rand Mutual Assurance Company. She found that the overall prevalence for PTSD was 0.09%, with 0.085% among males and 0.143% among females. These results confirmed her premise that mine accidents are traumatic stressors with the potential to cause severe mental health problems like PTSD long after physical injuries sustained by survivors of mine accidents had healed.

The prevalence of PTSD in South Africa was further investigated by Connell (2011) who distributed a quantitative, anonymous, internet-based questionnaire among 109 former South African National Servicemen who matriculated between 1975 and 1988 at a Johannesburg high school. Connell (2011) achieved a response rate of 49.5% and concluded that the PTSD level in the sample was 33%. This result was statistically significantly associated with combat exposure. Connell (2011) concluded that the PTSD prevalence in the population of former National Servicemen is higher than in comparable international studies.

From the above it is clear that to determine a nation's exact national lifetime prevalence rate for PTSD remains elusive. The present study however, could determine that according to the WMH Surveys traumatic event exposure is very common. Experiencing traumatic events is part and parcel of human experience. The SASH determined that the lifetime traumatic event

prevalence rate for South Africa is 73.8%. The most common traumatic events experienced in South Africa is the unexpected death of a loved one and witnessing trauma. Lifetime and 12-month prevalence rates of PTSD were found to be 2.3% and 0.7% respectively, while the conditional prevalence of PTSD after trauma exposure was 3.5%.

## **2.10 Chapter Summary**

This chapter provided a broad overview of the concept of stress by taking a closer look at the normal stress response in all its stages. The physiology of the stress response was addressed by focusing on the role of the amygdala in threat recognition and how the HPA axis is involved in regulating stimuli that threaten homeostasis. The importance of perception was discussed by highlighting the different levels of activation of the sympathetic nervous system to provide the body with the recourses to respond to emotional, stressful, and emergency situations. Here the discussion particularly focused on the concepts of allostasis and allostatic overload. Stress was also brought into relation to trauma and explained from a biopsychosocial approach. The development of Posttraumatic Stress Disorder (PTSD) was unpacked by examining the differences between normal trauma reactions versus a clinical psychological condition such as PTSD. This chapter provided a broad overview of the neuro-anatomy and biology associated with PTSD by launching an investigation into the abnormal stress response as a dysregulated energy system and the large-scale neurocircuitry models associated with PTSD. Chapter 2 concluded with a brief discussion regarding the prevalence of PTSD in the world in general and South Africa in particular. In the following chapter the link between PTSD and frontal lobe executive functions will be investigated.

## CHAPTER 3

### BRINGING IT ALL TOGETHER

*“It is abundantly obvious here that for longer than we can tell, the truth is immeasurably greater than all the tiny fragments we have so far been able to discover.” – Alexander Luria (1902–1977)*

The purpose of this chapter is to provide a synthesis of the knowledge acquired thus far about the impact of posttraumatic stress disorder on frontal lobe executive functions. This chapter will start with a discussion about the relationship between prefrontal executive functioning, stress, and PTSD. Thereafter the impact of stress on executive functions will be addressed by investigating the effect that acute stress has on executive functioning, followed by the impact that chronic stress exerts on frontal lobe executive functions. Here the focus will be on examining the influence of chronic stress on specific executive functioning processes such as working memory, attention, response inhibition, and cognitive flexibility.

#### **3.1 The Link between Prefrontal Executive Functions, Stress, and PTSD.**

The anatomy of the cerebral cortex is divided in a hierarchical manner. Located at the bottom of the cortical organisation one finds the sensory and motor areas supporting distinctive sensory and motor functions. Moving increasingly higher towards the later phylogenetic and ontogenetic developmental areas, one finds regions that support functions that are progressively more integrative. Situated at the hierarchical top is the prefrontal cortex (Fuster, 2001). The prefrontal cortex (PFC), often referred to as the most evolved brain region, is responsible for the highest-order cognitive abilities (Arnsten, 2009a). This means that the PFC is involved in the mediation of several top-down cognitive processes. The operations governed by the PFC enable the flexible regulation of behaviour that allow individuals to respond



appropriately to changes in their environment. This ability of the PFC includes the ability to shift attentional set to other dimensions and change decision-making to allow for advantageous rewards (Lee & Seo, 2007; Robbins, 2007). The PFC further monitors errors, providing insight into incorrect behaviour and therefore enable us to change our approach (Modirrousta & Fellows, 2008). The cognitive processes mediated by the PFC, generally referred to as executive functions, develop throughout the course of life and decline with age. These processes include attention, planning, working memory, cognitive flexibility, and response inhibition and demand effort and conscious engagement (Girotti et al., 2018). From the aforementioned, it is clear that executive functions are essential for everyday adaptive behaviour. These include the ability to concentrate and pay attention, plan a course of action, adjust to unanticipated events, or manage and control impulsive behaviours which are considered inappropriate to a particular situation (Barnes, Dean, Nandam, O'Connell, & Bellgrove, 2011; Diamond, 2013; Leh, Petrides, & Strafella, 2010; Logue & Gould, 2014; Robbins & Arnsten, 2009). These abilities rely on highly sensitive intact prefrontal cortical neural network connections, as well as a healthy balanced neurochemical environment (Arnsten, 2009a; Logue & Gould, 2014).

The PFC is however also the brain region that is extremely susceptible to the adverse effects of stress exposure. Even moderate acute uncontrollable stress can bring about a swift and significant loss of prefrontal cognitive abilities, whilst drawn-out exposure to stress causes structural changes in the prefrontal regions. Change so profound, that it affects the length, branching, and density of dendritic material (Arnsten, 2009a). Throughout the lifespan of an individual, acute and executive stress can challenge executive functions. The inability to regulate these processes leads to a reduction in the quality of life and impairment of appropriate daily conduct of otherwise healthy individuals. Research demonstrated that both intense stressful life events and prolonged stressors are significant risk factors for developing various

mental illnesses. These may include mood disorders (Beck, 2008) as well as anxiety and addictive disorders (Kessler, Davis, & Kendler, 1997). Dysfunction of executive processes is further a common symptom of various psychiatric conditions such as depression, generalised anxiety disorder, obsessive-compulsive disorder, attention-deficit hyperactivity disorder, and important for this study, posttraumatic stress disorder (PTSD) (Baler & Volkow, 2006; Carvalho et al., 2014; Ferreri, Lapp, & Peretti, 2011; Polak, Witteveen, Reitsma, & Olf, 2012). Girotti et al. (2018) acknowledges that in certain cases, executive dysfunction can be a direct consequence of an underlying disease process that are not caused by stress. They furthermore indicate that executive impairment in itself can cause a stressful experience, which could lead to the worsening of underlying psychiatric illnesses, such as in the case of depression (Jaeger, Berns, Uzelac, & Davis-Conway, 2006).

Sufficient scientific evidence exists supporting the idea that intact prefrontal cortical regions (anterior cingulate cortex, orbitofrontal cortex, dorsolateral prefrontal cortex, and the medial prefrontal cortex) are necessary for optimal executive control (Baier et al., 2010; Barbey, Koenigs, & Grafman, 2013; Colvin, Dunbar, Colvin, Dunbar, & Grafman, 2001; Drevets, Price, & Furey, 2008; Levens et al., 2014; Müller, Machado, & Knight, 2002). It should however be noted, according to Holmes and Wellman (2009), that the PFC does not function independently and that an extensive network of connectivity with other brain regions such as the hippocampus, amygdala, striatum, and posterior parietal cortex is necessary for mediating numerous aspects of executive functioning (As demonstrated in Figure 0.7 in Chapter 1 page 46). Human fMRI studies of large-scale interconnectivity networks identified by Menon (2011) indicated the central executive network (CEN) as one of three core neurocognitive networks. The neurocognitive networks consist of brain areas that are intrinsically connected. These networks are systematically co-activated during tasks requiring higher-order cognitive processes, such as executive control, working memory, and decision-

making tasks in goal-oriented behaviour (Menon, 2011). Behavioural outcomes associated with these processes originate from the coordinated activation and/or inactivation of numerous brain circuits integrating the PFC top-down control. The merging functions of the PFC perform within the broad neurobiological context of the sensory-motor cycle of interactions connecting individuals with their environments at all the levels of their nervous system. The PFC completes that cycle at the top through integrating temporal cognitive representations of perception and executing actions necessary for goal-directed behaviour (Fuster, 2001).

Diamond (2013) states clearly that executive functions and the PFC are the first to deteriorate and suffer disproportionately when something disturbs an individual's life. The deterioration occurs primarily when an individual is stressed (Arnsten, 2009a; Liston, McEwen, & Casey, 2009; Oaten & Cheng, 2005), sad (Hirt, Devers, & McCrea, 2008; Von Hecker & Meiser, 2005), lonely (Baumeister, Twenge, & Nuss, 2002; Campbell et al., 2006; Tun, Miller-Martinez, Lachman, & Seeman, 2013), sleep deprived (Barnes, Gozal, & Molfese, 2012), or not physically fit (Best, 2010; Chaddock, Hillman, Buck, & Cohen, 2011; Hillman, Erickson, & Kramer, 2008). The effects of the mentioned stressors are visible not only at the physiological or neuroanatomical level, but especially at behavioural level where impaired executive functioning present as poor reasoning and problem-solving ability, forgetfulness, and the inability to exercise discipline and self-control (Diamond, 2013).

This chapter sets out to investigate the impact of stress on executive functioning processes. A review of what is reported in literature regarding the prefrontal cortex's role in regulating the stress response and the subsequent compromised executive functions, including their subcomponent processes reflecting the effect of a stressful condition such as PTSD, will be provided.

### **3.2 Impact of stress on executive function.**

The first studies aimed at investigating the impact of stress on cognition in humans began following the end of the Second World War. This investigation between the relationship of stress and cognition was inspired by the observation of the behaviour of pilots during the war. Researchers observed that highly skilled pilots had a greater tendency to crash their aeroplanes during stressful combat situations largely due to mental errors compared to peacetime flights when stressful situations were absent (Broadbent, 1971). After recognising the effect of stress on the cognitive abilities of pilots, research began to experimentally manipulate stress levels to observe changes within the relationship between performance and cognitive abilities. Early studies, such as those conducted by Broadbent (1971) and Hartley and Adams (1974) demonstrated that exposure to stress indeed negatively affect an individual's ability to perform tasks that demand complex and flexible thinking. However, in contrast to its debilitating effect on cognitive performance, research found that stress also seem capable of improving an individual's performance on simple or well-rehearsed tasks (Hartley & Adams, 1974), including functions that are particularly associated with the hippocampus and amygdala (memory consolidation) (Cahill & McGaugh, 1996; McEwen, 2006). Today, there is consensus amongst researchers that the type of tasks negatively affected by stress are mostly the tasks that depend on PFC operations (Arnsten, 1998), while deep-rooted habits which depend on basal ganglia circuits, are preserved, or even amplified (Elliott & Packard, 2008).

During non-stressful situations (Figure 0.1) the vast connections of the PFC orchestrate the activities of the brain for astute regulation of behaviour, cognition, and emotion. In the event of psychological stress (Figure 0.2) the amygdala initiates stress pathways in the hypothalamus and brainstem, which in turn activates high levels of norepinephrine and dopamine. This causes impairment of PFC regulation while strengthening the functioning of the amygdala. The result is that attentional regulation switches from thoughtful top-down

control by the PFC what is based on the current situation to bottom-up control by the sensory cortices capturing our immediate attention. Thus, when we experience stress, the brain's response pattern changes from a slow thoughtful regulation by the PFC to a reflexive immediate emotional response by the amygdala and its related subcortical structures. In other words, acute uncontrollable stress negatively affects the functions mediated by the PFC, switching the control of behaviour and emotion to more primitive brain circuits (Arnsten, 2009a).

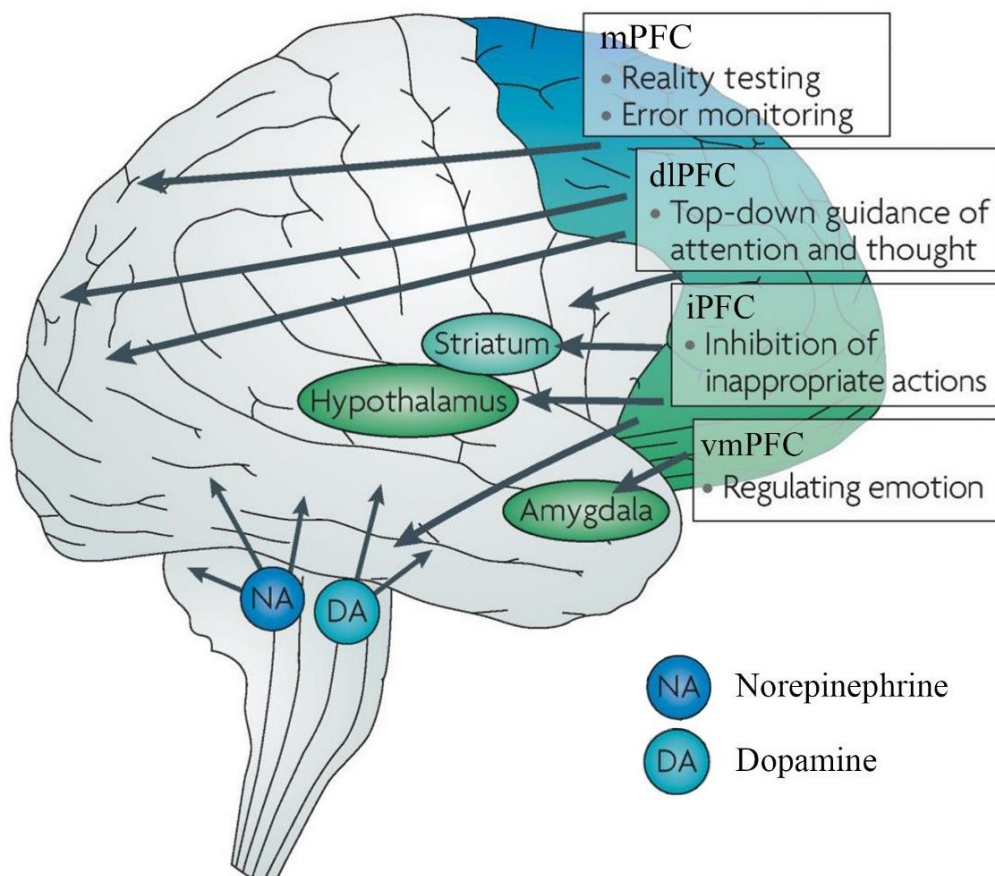


Figure 0.1. Top-down prefrontal cortex regulation during alert, non-stressful situations

Adapted from “Stress signalling pathways that impair prefrontal cortex structure and function,” by A.F.T. Arnsten, 2009a, *Nature Reviews Neuroscience*, 10(6), p. 414. Copyright 2009 by Macmillan Publishers Limited.

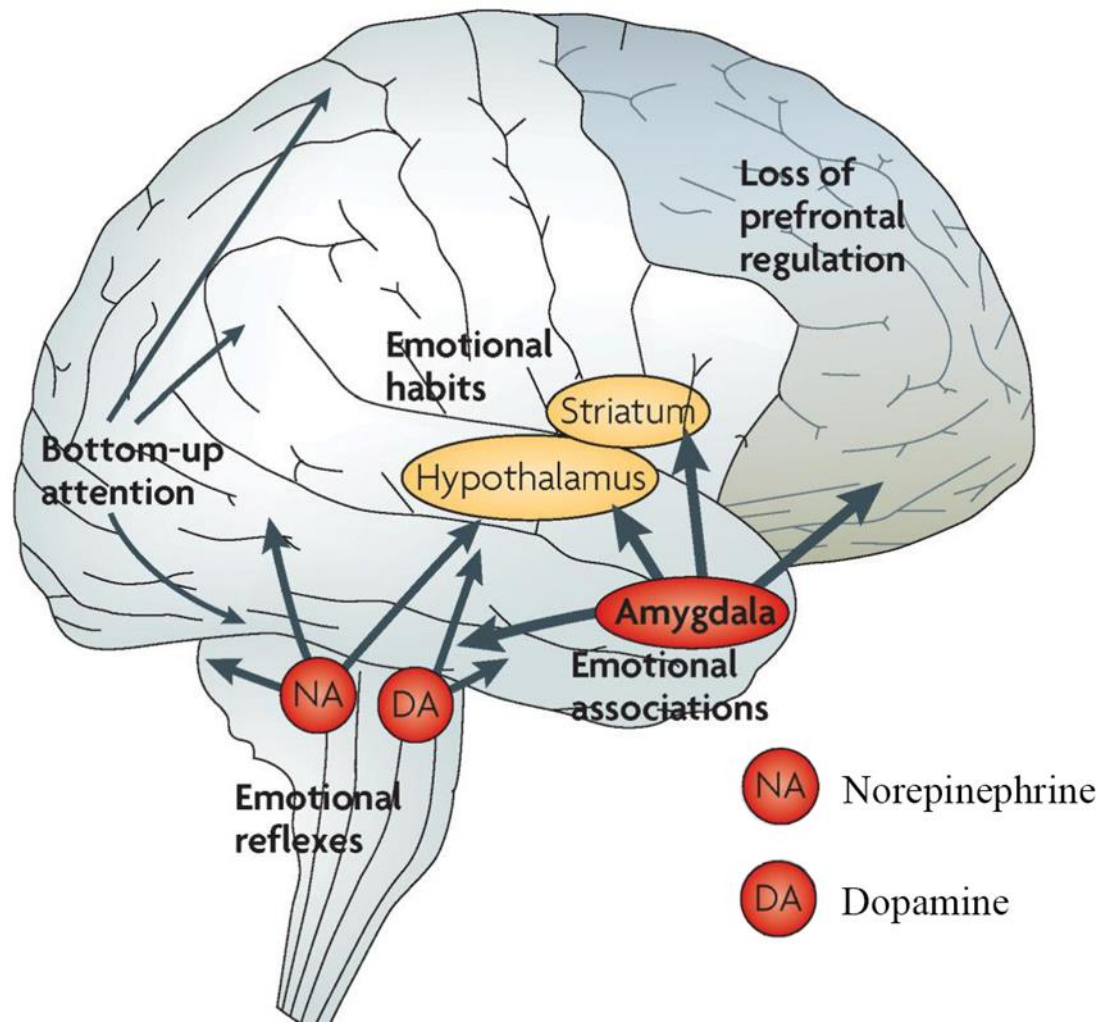


Figure 0.2. Bottom-up control of the amygdala during stressful situations

Adapted from “Stress signalling pathways that impair prefrontal cortex structure and function,” by A.F.T. Arnsten, 2009a, *Nature Reviews Neuroscience*, 10(6), p. 414. Copyright 2009 by Macmillan Publishers Limited.

An important aspect highlighted by those earlier studies mentioned above is the critical role of the subject’s sense of control over the stressor. Individuals who perceived themselves to be in control of the situation (despite this being an illusion) were usually not negatively affected when exposed to stress, while those individuals who experienced a sense of loss of control, were however impaired (Glass, Reim, & Singer, 1971).

In the following paragraphs, the effects of stress on executive functions with specific reference to working memory, attention, response inhibition, and cognitive flexibility will be dealt with. The discussion will commence with an overview of the impact of acute stress on executive function, followed by a review of the impact of chronic stress on executive function.

### **3.2.1 The impact of acute stress on executive function.**

The acute stress response exhibits a powerful impact on cognitive function. It activates saliency networks situated around the amygdala, anterior cingulate cortex, hypothalamus, insula, striatum, and the locus coeruleus. It is also responsible for furthering sensory gain and environmental scanning leading to improved performance (Cousijn et al., 2010; Oei et al., 2012; Van Marle, Hermans, Qin, & Fernández, 2010). On the other hand, cognitive processes underlying working memory, problem-solving, and cognitive flexibility are all adversely affected by acute stress (Oei, Everaerd, Elzinga, Van Well, & Bermond, 2006; Plessow, Fischer, Kirschbaum, & Goschke, 2011; Plessow, Kiesel, & Kirschbaum, 2012; Schoofs, Preuss, & Wolf, 2008; Schoofs, Wolf, & Smeets, 2009; Steinhäuser, Maier, & Hübner, 2007). This allocation of resources to cognitive functions are in line with an adaptive strategy for a short period. This strategy guarantees the allocation of resources to cognitive functions that enhance sensory hypervigilance, scanning attention, and immediate, but rigid behavioural responses. This happens at the expense of higher-order cognitive involvement. To achieve these effects, the actions of catecholamines and the immediate effects of glucocorticoids are necessary (Girotti et al., 2018).

Herman et al. (2016) and Hill and Tasker (2012) report that the adaptive value of the stress response depend on the swift clearance of the acute effects through the negative feedback mechanism. The effect thereof is that within one hour after the exposure to a stressful stimulus,

when the levels of catecholamines are low, but the levels of glucocorticoids are still elevated, the neurocognitive processes linked with the salience network decreases (Henckens et al., 2010; Henckens et al., 2012). Simultaneously, working memory and the ability to execute cognitive tasks improve together with a decline in anxious behaviour. Executive cognitive processes improve following exposure to an acute stressor, thereby confirming the effects of genomic corticosteroid actions (Henckens et al., 2011; Het & Wolf, 2007; Maheu, Joobert, & Lupien, 2005; Oei, Tollenaar, Spinhoven, & Elzinga, 2009; Putman, Hermans, Koppeschaar, van Schijndel, & van Honk, 2007).

### **3.2.2 The impact of chronic stress on executive function.**

When an individual's physiological response to stress is unable to return to homeostasis after acute activation, or with prolonged and/or excessive stress exposure (as associated with conditions of chronic stress) the physiological and cognitive outcome for the individual, can be detrimental. Stenfors, Marklund, Magnusson Hanson, Theorell, and Nilsson (2013) found, for example, that where individuals had difficulty with concentration, memory, and decision-making because of their exposure to chronic stress, they performed poorly on tests of attentional shifting and working memory. In their study among healthy individuals, Soares et al. (2012) found through using fMRI techniques, that prolonged exposure to stress causes an imbalanced activation within the networks responsible for governing decision-making processes. Although they found that stress-induced changes in human decision-making are reversible, chronic stress did alter individuals' decision-making strategies to favour habitual responding. Landrø, Rund, Lund, and Sundet (2001), as well as Rose and Ebmeier (2006), confirm the effect of chronic stress on individuals with mood and anxiety disorders who demonstrated deficits in executive function, particularly impairments in working memory amongst individuals with major depressive disorder. Merriam, Thase, Haas, Keshavan, and



Sweeney (1999) also provides neuropsychological evidence for significant prefrontal cortical dysfunction in individuals suffering from depression, therefore highlighting the adverse effect of chronic stress. Soares et al. (2012), in their study mentioned above, further found that chronic stress exhibits an atrophic effect on the morphology of the medial prefrontal cortex (mPFC) and the caudate. Surprisingly, they found that the volume of the putamen increased and dendritic arborisation occurred following exposure to chronic stress.

From the above and previous discussions (Chapter 2), it is evident that from the literature that chronic stress produces structural and functional changes in the fronto-striatal circuitry. These changes decrease the ability of an individual to shift from automated behaviour to goal-directed behaviour. Chronic stress may also cause maladaptive behaviour in situations that demand change.

It is evident that traumatic stress has a broad range of effects on the functioning of the brain. The medial prefrontal cortex, amygdala, and the hippocampus, the areas discussed in Chapter 2, play a critical role in memory, highlighting the important interplay between memory and the traumatic stress response (Bremner, 2007). Horner and Hamner (2002) indicate that there is evidence for deficits in attention and immediate memory associated with PTSD.

As indicated earlier the frontal areas of the brain are especially vulnerable to the effects of PTSD. The literature provides various studies showing impairment in distinctive domains of executive functioning because of PTSD. These include the domains of attention and working memory (Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Meewisse et al., 2005; Samuelson et al., 2006), inhibitory functions (Jenkins, Langlais, Delis, & Cohen, 2000; Koso & Hansen, 2006), and flexibility and planning (Beckham, Crawford, & Feldman, 1998; Jenkins et al., 2000; Stein, Kennedy, & Twamley, 2002). Despite the fact that impairment in executive functioning was not consistently found across studies (Crowell, Kieffer, Siders, &

Vanderploeg, 2002; Twamley, Hami, & Stein, 2004), Polak et al. (2012) point to an overall dysfunction in executive functioning in PTSD when compared to trauma-exposed and healthy controls. Neuropsychological approaches may contribute to the essential understanding of the susceptibility and resiliency factors by pinpointing pre-trauma cognitive functions that might relate to the subsequent development of posttraumatic stress disorder, as well as the posttraumatic cognitive processes that may influence the development or maintenance of the disorder. The main objective of this study is to determine whether a condition such as posttraumatic stress disorder has any significant influence on the executive functioning abilities of those individuals suffering from PTSD. This will be addressed by investigating the influence of PTSD on the following processes of executive functioning: (1) Working memory, (2) attention, (3) inhibitory control, and (4) cognitive flexibility.

#### **3.2.2.1 Working memory.**

Working memory as previously mentioned is the temporally storage of information used to perform a wide variety of cognitive tasks (Baddeley, 1992). The role of the PFC in working memory, especially in tasks with a delay component, is observed in individuals with focal brain damage involving both the ventral and dorsal parts of the lateral PFC. This specific damage in the PFC regions leads to impairment in tasks that requires the maintenance and monitoring of object and spatial information (Müller et al., 2002). Impairment in working memory sustained due to frontal lobe damage may bring about a wide range of problems regarding the control of attention, planning, and switching between tasks (Humphreys & Samson, 2004).

The impact of stress on working memory in individuals can be either positive or negative. The effects depend on the intensity and duration of the stressor. Prolonged and/or high intensity stressors negatively affect an individual's performance in working memory tasks

(Luethi, Meier, & Sandi, 2008; Schoofs et al., 2008). Evans and Schamberg (2009) report that working memory impairments in adults correlate with childhood poverty and are most likely the result of chronic stress experiences in early life. Working memory deficits were also found to be present in a broad range of stress-related psychiatric illnesses. For the aim of this study, the work of Veltmeyer et al. (2006) on brain stability and working memory in individuals with PTSD is relevant. Veltmeyer et al. (2006) investigated the influence of PTSD on working memory amongst 34 PTSD patients and 136 gender and age-matched controls. They found that the PTSD group demonstrated impairment on a number of measures of working memory performance. In particular, the members of this group demonstrated delayed reaction times and missed more targets as opposed to their counterparts. This group's reaction time served as a strong indicator of impaired working memory ability. Samuelson et al. (2006) confirmed the work of Veltmeyer and colleagues (2006) by finding a decreased performance in working memory amongst 128 veteran participants diagnosed with PTSD. These findings correspond with previous research conducted by Vasterling et al. (2002) who also found cognitive deficits in working memory tasks among 26 Vietnam veterans.

### **3.2.2.2 Attention.**

Sarter, Givens, and Bruno (2001) defines attention as the preparedness to notice rare or unexpected stimuli over an extended period. The emotional and motivational state of an individual can affect an individual's attentional capacity (Goetz, Robinson, & Meier, 2008) and is regularly impacted by stress. Injury sustained to the mPFC and a compromised connectivity of the inferior prefrontal cortex (iPFC) to striatal, cerebellar, and parietal areas lead to deficits in attention in humans (Arnsten, 2009b; Arnsten & Rubia, 2012; Aron & Poldrack, 2005).

Stress compromises different modalities of attention. Liu and Raine (2006) found that prenatal stress brought about by malnutrition is associated with a predisposition to attention deficits. Acute stress is also linked to attention deficit. Students' ability to pay attention are poorer during examination periods than during non-exam periods (Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000). Compared to newly recruited soldiers combat veterans who have experienced combat related trauma and demonstrate PTSD symptoms have decreased attention during cognitive tasks (Uddo, Vasterling, Brailey, & Sutker, 1993).

There is evidence suggesting that individuals who suffer from PTSD perform poorly on measures of both auditory and visual sustained attention when compared to individuals without PTSD and non-trauma controls (Brandes et al., 2002; Gilbertson et al., 2001; LaGarde, Doyon, & Brunet, 2010; Shucard, McCabe, & Szymanski, 2008; Vasterling et al., 2002; Wu et al., 2010). These attentional deficits are also found to closely correspond with PTSD symptom severity (Burriss, Ayers, Ginsberg, & Powell, 2008). Equally important to mention is that there are studies who have failed to identify impairments in auditory and visual sustained attention (Jenkins et al., 2000; Leskin & White, 2007; Neylan et al., 2004; Samuelson et al., 2006).

Although numerous neurocognitive impairments have been associated with chronic PTSD, LaGarde et al. (2010) investigated whether neurocognitive deficits involving emotionally neutral stimuli are also associated with acute PTSD. Their investigation relied on specific and standardised tasks such as the Trail Making Test, Stroop task, Concentration-Endurance Test (D2), as well as the Tower of London Test to examine attentional and executive functions among 21 trauma exposed individuals with acute PTSD, 16 trauma exposed individuals without PTSD, and a normal comparison group of 17 individuals. While the trauma exposed group without PTSD and the normal comparison group did not differ markedly in their performances, LaGarde et al. (2010) did however found that the performance for the acute PTSD group was significantly inferior on domains of explicit memory functioning, higher-

level attentional processes, working memory, and executive functions when compared to the other two groups.

Shucard et al. (2008) believe that the attentional problems associated in PTSD are connected to slowed central processing during instances when response inhibition is required as well as the inability to screen irrelevant information. Moreover, Bressan et al. (2009) are of the opinion that the dysfunction of higher-level attentional resources is responsible for affecting the activity in other systems concerned with memory and thought in PTSD. It seems that attention and concentration difficulties are the core deficits in PTSD while memory impairment may in fact be secondary to inadequate attention. Bressan et al. (2009) believes that the breakdown in attentional processes impede the accurate registration of information, subsequently preventing the consolidation and retrieval of memory.

Taken together these studies suggest that the effects of stress on attention may depend on the amount of time one is subjected to stress (i.e., chronic, acute, and experimentally-induced brief stress) and the emotional valence of the stimuli presented during the task. Thus, there seems to exist evidence, although inconsistent, that deficits in auditory and visual sustained attention may constitute an important aspect of the cognitive profile associated with PTSD. However, conclusive evidence is still lacking to fully support the notion that PTSD is linked to primary deficits in attention, or a result of difficulties in coping with and inhibiting unintentional distractors from an individual's internal (e.g., emotions or cognitions) or external stimuli (e.g., sounds and sights in the environment).

### ***3.2.2.3 Inhibitory control.***

An additional executive function mediated by the PFC and negatively affected by stress is the ability to inhibit a response. Inadequate response inhibition leads to impulsivity, which is referred to as an individual's tendency to act prematurely while lacking foresight

(Dalley, Everitt, & Robbins, 2011). This tendency includes actions that are not fully conceived, rashly expressed, immensely risky, or unsuitable to a situation that generally leads to undesirable outcomes (Chamberlain & Sahakian, 2007). Impulsivity stretches across various cognitive domains and consist of diversified behaviours. These behaviours include for instance (1) reflection impulsivity, which is the inability to sufficiently gather and assess information prior to making decisions, (2) impulsive choice, which is the inability to delay gratification by opting for a smaller and more immediate reward as opposed to a larger delayed reward, and (3) response inhibition, which refers to an individual's inability to suppress a response that has been "primed" to occur through reinforcement, repeated use, habit, or reflex (Chamberlain & Sahakian, 2007; Dalley et al., 2011; Fineberg et al., 2010). Given response inhibition's diverse role in executive functioning, the review will particularly focus on response inhibition in the following paragraphs.

Along with other cognitive functions mediated by the PFC, response inhibition also seems to be differently affected by acute and chronic stressors. Surprisingly, Schwabe, Höffken, Tegenthoff, and Wolf (2013) found that acute stress improves response inhibition while a mineralcorticoid receptor antagonist called spironolactone abolished this enhancement. Conversely, chronic exposure to stress impedes response inhibition in individuals. Rahdar and Galván (2014) found within in a population of 23 adults and 22 adolescents that reported high stress levels performed significantly worse on a response inhibition task versus low stress states. This effect was considerably stronger in the adolescent group highlighting an important age by stress interaction. At a neural level, adolescents displayed less recruitment of the dorsal lateral prefrontal cortex (dlPFC) under high stress versus low stress when engaged in inhibition, while adults demonstrated the opposite pattern of activation. Such findings may suggest that a developing brain may be more susceptible to the neurobiological and cognitive effects of daily stress (Rahdar & Galván, 2014). In addition, impaired inhibitory functioning has frequently

been reported among individuals diagnosed with PTSD (Bressan et al., 2009; Casada & Roache, 2005; Cottencin et al., 2006; Falconer et al., 2008; Koso & Hansen, 2006; Leskin & White, 2007; Shucard et al., 2008; Wu et al., 2010).

PTSD has been linked to deficits in information processing including hypervigilance to noticeable threat-related stimuli (Buckley, Blanchard, & Neill, 2000). Bryant et al. (2005) suggested that these deficits are associated with an increase in bottom-up hyperarousal together with a disruption in inhibitory functions that are essential for attentional control and working memory. Falconer et al. (2008) set out to investigate the hypothesis whether executive inhibitory control networks are jeopardised in PTSD. By using fMRI techniques, Falconer et al. (2008) examined the performances of individuals who were diagnosed with PTSD on a Go/No-Go inhibition task. Their results indicated that individuals diagnosed with PTSD made significantly more inhibition-related errors in comparison to controls without trauma exposure. In addition, their findings also illustrated a difference in the recruitment of inhibitory networks. Participants without trauma exposure activated a right-lateralised cortical inhibitory network, whereas participants diagnosed with PTSD only recruited the left lateral frontal cortex. PTSD is therefore linked to a cut back in right cortical activation coupled with an increase in activation in striatal and somatosensory areas. The increase in inhibitory errors as well as the reduced activation in the right frontal cortical areas, are in line with impaired inhibitory control in PTSD. The greater activation of brain areas associated with sensory processing together with an increasing demand on inhibitory control may be indicative of enhanced stimulus processing in individuals suffering from PTSD, which may weaken cortical control mechanisms (Falconer et al., 2008). The findings obtained by Falconer et al. (2008) do not only support previous models of PTSD by highlighting that PTSD is associated with enhanced stimulus processing (Bryant et al., 2005) and reduced cortical control (Bremner et al., 2004; Clark et al., 2003; Liberzon et al., 2003; Shaw et al., 2002; Veltmeyer et al., 2006; Weber et al., 2005), but also

expand these models by indicating that PTSD is subject to unique neural changes during tasks that require the careful control over executive inhibitory processes responsible for the processing of emotionally neutral information.

According to Cottencin et al. (2006), the capacity to retrieve memories relies on the individual's ability to inhibit others. Cottencin et al. (2006) studied 30 individuals who were diagnosed with PTSD and compared them to 30 healthy individuals based on their performance on the Direct Forgetting Task (DFT). The DFT examines an individual's ability to forget recently processed information as well as the retention of applicable information. Their results indicated that individuals with PTSD found it increasingly more difficult to remember and forget specific words when compared to their counterparts. They found that individuals with PTSD struggled to ignore words they were instructed to forget and recalled fewer words which they were instructed to remember, compared to their control group. Cottencin et al. (2006) believe that this discrepancy in the performance between the two groups can be attributed to a decline in directed forgetting in individuals who suffer from PTSD, which results in a struggle to inhibit irrelevant information from the overall information. Their findings highlighted the deficit in the inhibitory processes in memory consolidation in PTSD.

Furthermore, Swick, Honzel, Larsen, Ashley, and Justus (2012) set out to explore the effects of PTSD on motor response inhibition among 40 combat veterans with mild traumatic brain injury (mTBI). Their results showed that veterans with PTSD demonstrated an impaired ability when it came to inhibit inappropriate motor responses by committing more errors on No-Go trials than their controls. Their performance on the Go/No-Go task also significantly correlated with the severity of PTSD including depressive symptoms with higher levels of PTSD and depressive symptoms linked to higher error rates. Given that the co-morbidity of mTBI and PTSD was high in their population group, Swick et al. (2012) used secondary analyses to compare PTSD veterans with mTBI to PTSD veterans without mTBI during which



their results showed that the two groups did not differ on any measure. These results correspond with earlier findings in civilian populations (Falconer et al., 2008; Wu et al., 2010) as well as Gulf War Veterans (Vasterling, Constans, Brailey, & Sutker, 1998).

Poor performance on the colour-word Stroop task, which examines individuals' response time to name the ink colour of a colour-related word (e.g., "blue" printed in red), which is also accepted as a measure of inhibitory function, were documented for many different PTSD populations. Researchers such as Lindauer, Olf, Van Meijel, Carlier, and Gersons (2006) and Vasterling et al. (1998) linked PTSD with an increase of intrusions during memory recall which could possibly reflect difficulty to inhibit related, but irrelevant internally-generated stimuli. Vasterling et al. (1998) discovered that an individual's tendency to intrude information over a wide range of cognitive tasks, such as commission on attentional tasks and intrusions on memory measures, were associated with the severity of re-experiencing and hyperarousal symptoms in PTSD. Impaired inhibitory control could have potentially negative effects on an individual's ability to effectively engage in daily-living activities such as driving (Lew, Amick, Kraft, Stein, & Cifu, 2010) and may impede the process of recovery from potentially traumatic events (Aupperle, Melrose, et al., 2012). Research propose that impaired response inhibition results from compromised functionality in the inferior prefrontal cortex (iPFC) and its connections to striatal, cerebellar, and parietal regions (Arnsten, 2009b; Arnsten & Rubia, 2012; Aron & Poldrack, 2005).

There are increasing evidence that support the link between PTSD and inhibitory dysfunction that is specifically connected to re-experiencing and hyperarousal symptoms. It is possible that heightened arousal and re-experiencing symptoms may lead to greater distraction during situations in which an individual try to concentrate on a specific task, through which working memory, sustained attention, and inhibitory functions are interrupted. The possibility also exist that impaired inhibitory control may not only lead to poorer performance on cognitive

tasks, but may also compromise an individual's ability to inhibit emotional memories including physiological arousal in response to triggers (Aupperle, Melrose, et al., 2012). Casada and Roache (2005) suggests that the presence of disinhibition along with increased behavioural activation may explain the impulsivity and aggression generally associated with PTSD.

#### ***3.2.2.4 Cognitive flexibility.***

The power to alter one's behaviour in response to a changing environment—cognitive flexibility—is frequently associated with dysregulation in neuropsychological disorders such as PTSD (Aupperle, Melrose, et al., 2012; Jenkins et al., 2000; Stein et al., 2002). For example, in individuals who experience depressive mood states, the attentional bias towards negative information, including the maladaptive perseverative cognition and behaviours, continue regardless of a changing environment (Disner, Beevers, Haigh, & Beck, 2011; Peckham, McHugh, & Otto, 2010). A proliferation of evidence points to the negative effects that stress and anxiety have on various aspects of cognitive flexibility (McKlveen, Myers, & Herman, 2015; Park & Moghaddam, 2017). In the following review, the focus will be on two cognitive flexibility dimensions, namely reversal learning and set-shifting.

##### ***3.2.2.4.1 Reversal learning.***

Reversal learning is defined as a type of discrimination learning (Girotti et al., 2018). After acquiring the rule associated with a specific sensory discriminatory stimulus leading to an associated reward, the reinforcement outcomes are swapped (Izquierdo, Brigman, Radke, Rudebeck, & Holmes, 2017). Lesions sustained to the ventromedial prefrontal cortex (vmPFC) are associated with deficits in reversal learning (Fellows & Farah, 2003; Hornak et al., 2004). Other brain regions essential for reversal learning are the striatum (Cools, Clark, Owen, & Robbins, 2002; Rogers, Andrews, Grasby, Brooks, & Robbins, 2000), amygdala (Wassum & Izquierdo, 2015), and the hippocampus (Levy-Gigi & Richter-Levin, 2014).

The effects of stress on reversal learning were demonstrated by Levy-Gigi and Richter-Levin (2014) through the impaired performance of individuals who had been repeatedly exposed to traumatic events, even without a diagnosis of PTSD, in a cue-context reversal learning paradigm. Levy-Gigi and Richter-Levin (2014) compared the performance of active-duty firefighters who were regularly exposed to trauma as part of their occupation and a matched civilian control group without any exposure to traumatic events. They found that although the active firefighters as well as the unexposed control group could obtain and recall stimulus-outcome associations, the firemen had difficulty learning that a previous associated negative context could later be associated with a positive outcome. The research conducted by Levy-Gigi and Richter-Levin (2014) suggest that both individuals with PTSD and individuals that were highly exposed to similar traumatic events, are unable to link traumatic outcomes with their appropriate context.

#### 3.2.2.4.2 *Set-shifting.*

Unlike reversal learning, set-shifting demands shifting an attentional response towards alternative stimuli across various dimensions (Girotti et al., 2018). This shift appears when the relative reinforcement value of the stimulus changes. The Wisconsin Card Sorting Test, as well as the Trail Making Test (Aupperle, Melrose, et al., 2012), are generally used to explore an individual's set-shifting capacity as an indicator of prefrontal cortical activity (Miller & Cohen, 2001; Stuss et al., 2000). A decline in performance on set-shifting, reflected in the results of the Wisconsin Card Sorting Test, is indicative of the breakdown in the integrity of the prefrontal cortex, specifically the dorsolateral regions (Manes et al., 2002). Orem, Petrac, and Bedwell (2008) found that 81 undergraduate students with high self-reported chronic stress required more time to complete the set-shifting component of the Trail Making Test, thereby confirming the effects of stress on set-shifting.

Liston et al. (2009) confirmed the results of Orem et al. (2008) by using fMRI on 20 healthy young adults who had been exposed to psychosocial stress over a period of one month. These young adults were required to perform a prefrontal cortical dependent attention-shifting task. After a month, the subjects returned for a second scanning session following a period of reduced stress. Liston et al. (2009) found that psychosocial stress selectively impaired the test subjects' attentional control capacity. The psychosocial stress also affected the subjects' functional connectivity in their frontoparietal networks which are responsible for mediating attentional shifts. It is interestingly to note that Liston et al. (2009) reported that these effects were reversible. After a period of one month of reduced stress, the young adults presented no significant differences from the control group. Their results emphasise the pliability of prefrontal cortex networks in healthy individuals and indicate a mechanism by which disrupted plasticity may add to cognitive deficits attributes of stress-related neuropsychiatric disorders in vulnerable individuals. Through this study Liston et al. (2009) demonstrated that, although psychosocial stress may have long-lasting effects on the functioning of the prefrontal cortex in humans, the detrimental effect caused, are to an extent reversible for set-shifting.

The capacity to switch between various tasks is a fundamental aspect of executive control (Aupperle, Melrose, et al., 2012). Determining an individual's capability to be mentally flexible and to switch between tasks is measured by means of the Trail Making Test, which involves the connecting of dots while switching between letter and number sets, and verbal fluency switching, which involves the production of words while switching between categories (Delis et al., 2001). Executive functioning measures which include the added dimensions of planning and strategy application, includes tests such as the Tower of London Test as well as the Wisconsin Card Sorting Test (Aupperle, Melrose, et al., 2012). While investigating cognitive deficits in PTSD some inconsistent findings are reported using these measuring instruments. Kanagaratnam and Asbjørnsen (2007) found that individuals with PTSD required

larger number of trails to complete the first category of the Wisconsin Card Sorting Test (which is indicative of deficits in initial problem-solving), but failed to find impairments on overall performance. Likewise, Twamley et al. (2009) also reported a similar finding in which PTSD is associated with an escalation in the number of trails that it takes to complete the first category, but with an increase in learning efficiency. Aupperle, Melrose, et al. (2012) argue that this discrepancy could be because the Wisconsin Card Sorting Test involves cognitive switching and flexibility similar to that of the Trail Making Test. However, the Trail Making Test is a timed test that demands quick attentional switching between already defined tasks. On the other hand, the Wisconsin Card Sorting Test is untimed and demands the production of a strategy, as well as switching between developed strategies, as opposed to flexibility and quick switching of attention as in the Trail Making Test. Neuropsychological research aimed at investigating cognitive flexibility provides evidence of deficits in speed-reliant and attentional switching. It furthermore demonstrates that rule-learning, planning, and untimed strategy switching are largely still intact in individuals who suffer from PTSD.

### **3.3 Chapter Summary**

When linking the prefrontal executive functions with stress and any stress-related condition such as PTSD it must be noted that the prefrontal cortex is the brain region that is extremely susceptible to the adverse effects of stress exposure. The resources consulted in this chapter is adamant that even moderate stress can bring about significant impairment in prefrontal cognitive abilities, while drawn out-exposure may cause structural changes in the prefrontal regions. An individual's inability to regulate these stressors may lead to a reduction in the quality of life and impairment in everyday activities. Compounding the effect of stress, executive impairment may furthermore lead to the worsening of a stressful experience eventually developing into a psychiatric illness. The effects of stressors are not only visible on the physiological or neuroanatomical levels, but especially on the behavioural level where

impaired executive functioning presents as poor reasoning and problem-solving abilities, forgetfulness, as well as the inability to exercise discipline and self-control. However, it has also been mentioned that stress is able to improve an individual's performance on simple or well-rehearsed tasks, especially tasks that are associated with the hippocampus and the amygdala. Thus, when we experience stress the brain's response pattern changes from a slow thoughtful regulation by the prefrontal cortex to a reflexive immediate emotional response by the amygdala and its related subcortical structures.

The frontal areas of the brain are especially vulnerable to the effects of PTSD. The domains affected include working memory, attention, inhibitory control, cognitive flexibility, and planning. Thus, an overall dysfunction in executive functioning occurs in individuals diagnosed with PTSD. The aim of this chapter has therefore been reached in determining that a condition developed following exposure to chronic or acute traumatic stress has a significant influence on the executive functioning abilities of those suffering from PTSD. To test if such a relationship exists in the context of South Africa, the next chapter will set out to investigate the hypothesis whether there are statistically significant differences between two groups (PTSD+ and PTSD-) of adult participants with different levels of trauma exposure in respect of their frontal lobe executive functioning performance on an Executive Functioning Battery.

## CHAPTER 4

### THEORETICAL OR PARADIGMATIC POINT OF DEPARTURE

*“We shall not cease from exploration and the end of all our exploring will be to arrive where we started and know the place for the first time.” – T.S. Eliot (1888 – 1965)*

In the preceding chapters a theoretical base for the current study is systematically provided by focussing on the main concepts such as the general functions associated with the frontal lobes, executive functioning, and stress, specifically the interface between posttraumatic stress disorder and frontal lobe executive functioning. The objective of this chapter in the first instance, is to provide the research design and research questions. Thereafter, the rationale for the way in which the research question is investigated, will be discussed, followed by the aim, objectives, and justification for the current study, including the hypotheses on which the study is built. Hereafter, a discussion on the empirical methodology used in this investigation, will follow. The methodology will be presented in the discussion about the research participants and sample. This discussion will include the procedures followed during the data collection, as well as the measuring instruments employed. This chapter will conclude with an overview and justification of the methods employed for the analysis of the statistical data. In the subsequent paragraphs, a discussion regarding the research design and the research questions for the current study will follow, after which the researcher will provide a rationale for the research methodology.

#### **4.1 Research design and research questions**

The research design implemented is an ex post facto comparative research design according to a positivistic/post-positivistic methodological paradigm during which two groups of participants are compared in terms of various domains of frontal lobe executive functioning.

The overall research question deals with the relationships between posttraumatic stress disorder and frontal lobe functioning, specifically those functions encapsulated under the umbrella term—executive functions. These frontal lobe functions are measured by the Executive Functioning Battery, which consists of two specific measuring instruments:

- Executive Function Index (EFI) (Miley & Spinella, 2006) consisting of five subscales namely Motivational Drive (MD), Organisation (ORG), Strategic Planning (SP), Impulse Control (IC), and Empathy (EM).
- Delis Kaplan Executive Function System (D-KEFS) (Delis et al., 2001), during which three subtests with high validity and reliability coefficients are used (Tower Test, Colour–Word Interference Test, and the Trail Making Test). The three D-KEFS subtests also consist of five subscales namely the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio.

Collectively, the subscales from both the EFI and the D-KEFS constitute the 10 subscales of the Executive Functioning Battery.

The specific questions that the current study aim to answer, apart from the questions that relate to the measuring instruments (e.g., reliability, validity, and normal distributions), are typically positivistic in nature and includes questions about comparisons. These questions are as follow:

Comparisons:

- Comparisons in terms of level of trauma exposure:



1. Are there differences in the mean scores between two groups of participants with different levels of trauma exposure with respect to frontal lobe executive functioning as measured by the ten subscales of the Executive Functioning Battery?
- Comparisons in terms of gender:
    1. Are there differences in the mean scores between male and female participants?
    2. Are there differences in the mean scores between male and female participants diagnosed with PTSD?
    3. Are there differences in the mean scores between male and female participants without the diagnoses of PTSD?
  - Comparisons in terms of education:
    1. Are there differences in the mean scores between participants in terms of the level of academic qualification?

## **4.2 Rationale for the methodology**

The discussion of the rationale for the methodology of the current study is introduced by the argument stating that gaps exist within the field of knowledge regarding the variables that are under investigation and that the variables are also poorly integrated. It thus assumes a research question that revolves around causal relationships, which can be accommodated by a positivist/post-positivist methodological paradigm, taking into consideration that reality can be studied, but never fully apprehended, only approximated (Lincoln & Guba, 2000). This methodological paradigm has implications on the selection of the sample and the way in which data is gathered and analysed. This research study therefore aims to determine, through the use of a scientific method, if there is a significant difference amongst the executive functioning abilities in individuals suffering from posttraumatic stress disorder. In other words, this study

will make use of systematic observation and description of phenomena contextualised within a model or theory, the presentation of hypotheses, the execution of tightly controlled assessments, the use of inferential statistics to test hypotheses, and the interpretation of the statistical results in light of the original theory. It is important to mention one of the main criticisms of positivistic approaches, is that this approach is reductionistic in nature. This criticism could be levelled against the current study. However, the complex nature of the current field of study is not denied in the research process. The findings of this study focus on a comprehensive view.

The chosen paradigm is also explained by means of a macro-model, namely, the Biopsychosocial Model. This model serves as a theoretical framework by which posttraumatic stress disorder is explained. This model provides the researcher with a framework for understanding the very complex relationship between psychological development and pathology (Christopher, 2004). This study will also try to interpret and contextualize its findings within the complementary models of the functioning of the prefrontal cortex as set forth by Luria in his classical view of problem-solving and the frontal lobes and Norman and Shallice's framework of frontal supervision (Grafman, 1999).

From the above, it is clear that an empirical research method is appropriate to answer the research questions within this study. The criticism against the empirical research method employed was also addressed. In the following section, the purpose for the current study will be explained.

### **4.3 Aim, Objectives, and Justification**

The aim of this research study is to do theoretical, empirical, and quantitative research into the neurocognitive basis of posttraumatic stress disorder with specific reference to frontal lobe executive brain functioning. This study falls within the biopsychosocial domain, which

attempts to explain psychological phenomena in terms of their biological and social foundations (Hergnhahn, 1997). The current research study is basic in nature, but has potential implications for the practice of psychology.

The overarching research question for the proposed research study is: Does posttraumatic stress disorder affect frontal lobe executive functioning? This research question can be divided into specific comparative research questions as evidenced by the specific objectives of the study that will be discussed next.

#### **4.3.1 Specific objectives**

The main objective of the study is to determine whether there are statistically significant differences in the Executive Functioning Battery test scores between two groups of participants with different levels of trauma exposure.

#### **4.3.2 General objectives**

The general objective of this study is to undertake an extensive literature study focusing on frontal lobe executive functioning, posttraumatic stress disorder, and the relationship between these two constructs.

On an empirical level, the current study aims to contribute to already existing knowledge regarding traumatic stress and its influence on frontal lobe executive functioning amongst the adult population in the South African context. This investigation will also attempt to identify gaps within the research field and possible suggestions will also be provided to how these gaps can potentially be addressed.

#### **4.3.3 Motivation**

The rationale for the central research question is motivated by the following:

Despite the fact that PTSD is just one of many clinically recognisable responses to trauma that often occur it has come to occupy center stage in research, writing, and clinical

interventions (Kirmayer et al., 2007). In the field of psychiatry, current work regarding the psychological impact of trauma has mostly focused on the diagnostic criteria of PTSD. Moreover, most of the neuropsychological research in PTSD has focused on learning and memory; therefore, the current research study justifies this investigation to provide new information and contribute to the dearth in existing research dealing with the effect of PTSD on frontal lobe executive functioning. This study specifically aims to investigate frontal lobe executive functioning within individuals who are suffering from PTSD. According to the knowledge of this researcher, such research does not currently exist in South Africa and may further contribute to the understanding of how cognitive dysfunction is associated with PTSD symptomatology (e.g., hyperarousal, avoidance, and re-experiencing) or to other factors (e.g., aspects relating to trauma itself). The reason for this investigation is motivated by already existing evidence within the literature, which indicates that executive dysfunction that leads to maladaptive behaviour and is a symptom of psychiatric pathology, may be instigated or exacerbated by stress (Girotti et al., 2018). Examples are provided and discussed in the literature section of this study. Numerous research studies demonstrate that the frontal lobes are involved in executive functioning, which plays an important role in physical and mental health. Clinically, the emphasis on PTSD reflects the fact that specific treatment interventions based on learning theory are effective in helping some sufferers (Kirmayer et al., 2007). It is further argued that the investigation into the executive functioning deficits associated with PTSD is as important to the treatment of PTSD as it is to the refinement of the psychological theories associated with PTSD (Kristensen, Parente, & Kaszniak, 2006).

An improved understanding and recognition regarding the distinctive manner in which various types of counselling approaches affects the brain is needed. This study is thus further motivated by the hope that by incorporating knowledge from cognitive and neuroscientific research, efficient treatment plans can be developed that will make it possible for us to treat

those who suffer from PTSD more successfully. In the next section, the hypotheses of the current study are formulated.

#### **4.4 Hypotheses of the Current Study**

For the purpose of the current study, the null hypothesis will not be set, but a non-directional alternative hypothesis. The null hypothesis states that there is no difference or correlation within the general population. It therefore holds that the independent variable has no effect on the dependent variable. In contrast, the alternative hypothesis suggests that there is a change, difference, or correlation in the sample that was drawn from the larger population that is currently under investigation. It therefore states that the independent variable (PTSD) will have an effect on the dependent variable (frontal lobe executive functioning) (Pietersen & Maree, 2007; Salkind, 2008).

All the hypotheses will be formulated in two ways, because it is not clear in which direction the effect will be observed. There are mostly contradictory research findings about the differences and relationships between the effects that PTSD has on frontal lobe executive functioning. Among the cognitive deficits found in PTSD, attentional and executive dysfunction have been associated with negative outcomes in the fields of mental health, career success, and marital harmony (see the discussion presented in Table 0.3 in Chapter 1 page 33); in other words, daily living activities and social relations. As discussed in Chapter 3, evidence exist linking attention and executive dysfunction to re-experiencing and avoidance symptoms associated with PTSD. Brewin and Beaton (2002) as well as Vasterling et al. (1998) state that the re-experiencing symptoms and avoidance symptoms may be due to a lack of inhibitory control. For example, they found that individuals failed to suppress involuntary thoughts and direct their attention to stimuli besides those related to the trauma. It is possible that this breakdown in cognitive control processing may be linked to each individual's capability to

regulate subjectivity (Shiffrin & Schneider, 1984). It has also been suggested that hyperarousal symptoms are linked to over stimulated attention, in such a way that selective attention (e.g., the ability to distinguish which information is relevant and should be remembered) is compromised (McFarlane, Weber, & Clark, 1993). Notwithstanding these hypotheses, it must be noted that no agreement exist amongst researchers regarding which aspects of attention and executive functions (e.g., working memory, inhibitory control, shift of attention, self-monitoring, mental flexibility, attention span, concentration, or abstraction) are more impaired. Put differently, there appear to be no general agreement concerning the order of attentional and executive dysfunction that correlate with PTSD.

As indicated earlier, two groups are identified for the current research study:

- Group 1: Consist of 44 trauma exposed individuals who had been diagnosed with PTSD by a professional clinician. These participants will serve as the posttraumatic stress disorder positive group (PTSD+).
- Group 2: Consist of 44 trauma and non-trauma exposed individuals who do not meet the criteria for PTSD and who will serve as the posttraumatic stress disorder negative healthy comparison control group (PTSD-).

The main hypothesis for the current study is as follows:

There are statistically significant differences between two groups of participants with different levels of trauma exposure regarding their mean scores on an Executive Functioning Battery.

$$H_1: \bar{X}_1 \neq \bar{X}_2$$

where

$H_1$  represents the symbol for the main research hypotheses.

$\bar{X}_1$  represents the mean scores for PTSD+ group with respect to the frontal lobe executive functioning as measured by the ten subscales of the Executive Functioning Battery.

$\bar{X}_2$  represents the mean scores for PTSD– with respect to the frontal lobe executive functioning as measured by the ten subscales of the Executive Functioning Battery.

The main hypothesis is further divided to represent the ten subscales of the Executive Functioning Battery, specifically the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio; and the EFI the EFI Motivational Drive, Organisation, Strategic Planning, Impulse Control, and Empathy subscales. One example is formulated below:

There are statistically significant differences between the PTSD+ and the PTSD– group regarding their mean scores on D-KEFS Trail Making Test – Condition 4: Number Letter Switching subscale of the Executive Functioning Battery.

Hypotheses with respect to comparisons are formulated separately, followed by their respective additional sub-hypotheses that bear reference to the ten subscales of the Executive Functioning Battery. Where appropriate, a rationale for the hypothesis from the literature will briefly be provided. Comparative hypotheses with respect to the level of trauma exposure, gender, and education pertaining to the two groups are as follow:

#### **4.4.1 Hypotheses with respect to differences in level of trauma exposure.**

*Hypothesis 1: The mean scores between the PTSD+ group and the PTSD– group in respect of the different subscales of the Executive Functioning Battery (D-KEFS and the EFI) differ significantly.*

The subscales of the Delis Kaplan Executive Function System (D-KEFS) include scores for the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio questions. The subscales of the Executive Function Index (EFI) include scores for Impulse control, Strategic planning, Organisation, Motivational drive, and Empathy questions.

There are statically significant differences in the mean scores between the PTSD+ group and the PTSD– group with respect to the following frontal executive functioning subscales:

Hypothesis 1.1: Mean scores for D-KEFS Trail Making Test – Condition 4: Number Letter Switching.

Hypothesis 1.2: Mean scores for D-KEFS Colour – Word Interference Test – Condition 3: Inhibition.

Hypothesis 1.3: Mean scores for D-KEFS Colour – Word Interference Test – Condition 4: Inhibition/Switching.

Hypothesis 1.4: Mean scores for D-KEFS Tower Test – Total Achievement Score.

Hypothesis 1.5: Mean scores for D-KEFS Tower Test – Move Accuracy Ratio.

Hypothesis 1.6: Mean scores for Motivational Drive.

Hypothesis 1.7: Mean scores for Organisation.

Hypothesis 1.8: Mean scores for Strategic planning.



Hypothesis 1.9: Mean scores for Impulse Control.

Hypothesis 1.10: Mean scores for Empathy.

Evidence exist in the literature reflecting impaired performance in distinctive domains of executive functioning as a result of PTSD when compared to trauma-exposed and healthy controls. Jenkins et al. (2000) for example found that rape survivors with PTSD performed significantly worse than trauma-exposed and healthy controls without PTSD diagnoses on measures of sustained and divided attention. In addition to their findings, Stein et al. (2002) also found that female victims of intimate partner violence, regardless of PTSD status, demonstrated statistically significant differences on tasks of speeded, sustained auditory attention, and working memory, including response inhibition. Female victims of intimate partner violence with the diagnosis of PTSD, especially performed worse than normal controls on a set-shifting task. The literature provides numerous findings highlighting executive dysfunction in areas of attention and working memory (Gilbertson et al., 2001; Samuelson et al., 2006), inhibitory functions (Koso & Hansen, 2006) and, flexibility and planning (Jenkins et al., 2000; Beckham et al., 1998). Based on these findings, it is therefore possible that we can suspect that an overall dysfunction in executive functioning associated with PTSD may exist when compared to trauma-exposed and healthy controls.

#### **4.4.2 Hypotheses with respect to gender differences.**

*Hypothesis 2: The mean scores between male and female participants in respect of the different subscales of the Executive Functioning Battery (D-KEFS and the EFI) differ significantly.*

The subscales of the Delis Kaplan Executive Function System (D-KEFS) include scores for the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference

Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio questions. The subscales of the Executive Function Index (EFI) include scores for Impulse control, Strategic planning, Organisation, Motivational drive, and Empathy questions.

There are statically significant differences in the mean scores between male and female participants with respect to the following frontal lobe executive functioning subscales:

Hypothesis 2.1: Mean scores for D-KEFS Trail Making Test – Condition 4: Number Letter Switching.

Hypothesis 2.2: Mean scores for D-KEFS Colour – Word Interference Test – Condition 3: Inhibition.

Hypothesis 2.3: Mean scores for D-KEFS Colour – Word Interference Test – Condition 4: Inhibition/Switching.

Hypothesis 2.4: Mean scores for D-KEFS Tower Test – Total Achievement Score.

Hypothesis 2.5: Mean scores for D-KEFS Tower Test – Move Accuracy Ratio.

Hypothesis 2.6: Mean scores for Motivational Drive.

Hypothesis 2.7: Mean scores for Organisation.

Hypothesis 2.8: Mean scores for Strategic planning.

Hypothesis 2.9: Mean scores for Impulse Control.

Hypothesis 2.10: Mean scores for Empathy.

*Hypothesis 3: The mean scores between male and female participants diagnosed with PTSD in respect of the different subscales of the Executive Functioning Battery (D-KEFS and the EFI) differ significantly.*

The subscales of the Delis Kaplan Executive Function System (D-KEFS) include scores for the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio questions. The subscales of the Executive Function Index (EFI) include scores for Impulse control, Strategic planning, Organisation, Motivational drive, and Empathy questions.

There are statically significant differences in the mean scores between male and female participants diagnosed with PTSD with respect to the following frontal executive functioning subscales:

Hypothesis 3.1: Mean scores for D-KEFS Trail Making Test – Condition 4: Number Letter Switching.

Hypothesis 3.2: Mean scores for D-KEFS Colour – Word Interference Test – Condition 3: Inhibition.

Hypothesis 3.3: Mean scores for D-KEFS Colour – Word Interference Test – Condition 4: Inhibition/Switching.

Hypothesis 3.4: Mean scores for D-KEFS Tower Test – Total Achievement Score.

Hypothesis 3.5: Mean scores for D-KEFS Tower Test – Move Accuracy Ratio.

Hypothesis 3.6: Mean scores for Motivational Drive.

Hypothesis 3.7: Mean scores for Organisation.

Hypothesis 3.8: Mean scores for Strategic planning.

Hypothesis 3.9: Mean scores for Impulse Control.

Hypothesis 3.10: Mean scores for Empathy

*Hypothesis 4: The mean scores between male and female participants without the diagnosis of PTSD in respect of the different subscales of the Executive Functioning Battery (D-KEFS and the EFI) differ significantly.*

The subscales of the Delis Kaplan Executive Function System (D-KEFS) include scores for the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio questions. The subscales of the Executive Function Index (EFI) include scores for Impulse control, Strategic planning, Organisation, Motivational drive, and Empathy questions.

There are statically significant differences in the mean scores between male and female participants without the diagnosis of PTSD with respect to the following frontal executive functioning subscales:

Hypothesis 4.1: Mean scores for D-KEFS Trail Making Test – Condition 4: Number Letter Switching.

Hypothesis 4.2: Mean scores for D-KEFS Colour – Word Interference Test – Condition 3: Inhibition.

Hypothesis 4.3: Mean scores for D-KEFS Colour – Word Interference Test – Condition 4: Inhibition/Switching.

- Hypothesis 4.4: Mean scores for D-KEFS Tower Test – Total Achievement Score.
- Hypothesis 4.5: Mean scores for D-KEFS Tower Test – Move Accuracy Ratio.
- Hypothesis 4.6: Mean scores for Motivational Drive.
- Hypothesis 4.7: Mean scores for Organisation.
- Hypothesis 4.8: Mean scores for Strategic planning.
- Hypothesis 4.9: Mean scores for Impulse Control.
- Hypothesis 4.10: Mean scores for Empathy.

Existing literature demonstrates that females perform better on verbal and memory tasks in comparison to men (Boghi et al., 2006; Fenson et al., 1994; Goldstein et al., 1998; Kramer, Delis, Kaplan, O'Donnell, & Prifitera, 1997). In addition to performing cognitive tasks, females tend to from a young age to be superior to males in social skills, demonstrating greater empathy, sensitivity to facial expressions, and a higher developed theory of mind (Baron-Cohen, 2002; Dunn, Brown, Slomkowski, Tesla, & Youngblade, 1991). There are also indications that females are better at verbal fluency tasks than males (Reader, Harris, Schuerholz, & Denckla, 1994). Men on the other hand, tend to outperform women on spatial tasks which includes activities comprising of spatial cognition and spatial learning (Astur, Ortiz, & Sutherland, 1998; Geary, Saults, Liu, & Hoard, 2000; Saucier et al., 2002; Waller, Hunt, & Knapp, 1998). Boghi et al. (2006) argue that females tend to employ executive strategies more and rely more on working memory during planning tasks, while males rely more on the use of visuospatial reasoning abilities. Males also appear to outperform females in multitasking because of their superior ability in spatial ability tasks (Mäntylä, 2013). Polak et al. (2012) also found that males diagnosed with PTSD performed significantly worse than

females with PTSD on a Digit Span Test. Given these findings, it is therefore possible that we can suggest that gender differences do exist in frontal lobe executive functioning.

#### **4.4.3 Hypotheses with respect to differences associated with education.**

*Hypothesis 5: The mean scores between participants in terms of their level of academic qualification in respect of the different subscales of the Executive Functioning Battery (D-KEFS and the EFI) differ significantly.*

The subscales of the Delis Kaplan Executive Function System (D-KEFS) include scores for the D-KEFS Trail Making Test – Condition 4: Number Letter Switching; D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching; D-KEFS Tower Test – Total Achievement Score; and the D-KEFS Tower Test – Move Accuracy Ratio questions. The subscales of the Executive Function Index (EFI) include scores for Impulse control, Strategic planning, Organisation, Motivational drive, and Empathy questions.

There are statically significant differences in the mean scores between participants in terms of their level of academic qualification with respect to the following frontal executive functioning subscales:

Hypothesis 5.1: Mean scores for D-KEFS Trail Making Test – Condition 4: Number Letter Switching.

Hypothesis 5.2: Mean scores for D-KEFS Colour – Word Interference Test – Condition 3: Inhibition.

Hypothesis 5.3: Mean scores for D-KEFS Colour – Word Interference Test – Condition 4: Inhibition/Switching.

- Hypothesis 5.4: Mean scores for D-KEFS Tower Test – Total Achievement Score.
- Hypothesis 5.5: Mean scores for D-KEFS Tower Test – Move Accuracy Ratio.
- Hypothesis 5.6: Mean scores for Motivational Drive.
- Hypothesis 5.7: Mean scores for Organisation.
- Hypothesis 5.8: Mean scores for Strategic planning.
- Hypothesis 5.9: Mean scores for Impulse Control.
- Hypothesis 5.10: Mean scores for Empathy.

Literature suggests that a strong relationship exists between an individual's level of education and his/her performance on certain cognitive tests. Van Hooren et al. (2007) provides proof hereof by reporting that older participants with a middle or high level of education obtained better results on most cognitive tests in comparison with participants with a lower level of education. In their population-based study amongst older adults, Van Hooren et al. (2007) specifically found effects of education on a set-shifting task. Other researchers support these findings by reporting educational effects on choice reaction times (Deary, Der, & Ford, 2001), as well as simple speeded tests (Bosma, Van Boxtel, Ponds, Houx, & Jolles, 2003; Fritsch et al., 2007). Tun and Lachman (2008), Plumet, Gil, and Gaonac'h (2005), including Wecker, Kramer, Hallam, and Delis (2005) also found effects of education on other tests of executive function (Wechsler Abbreviated Scale of Intelligence (WASI) and the Delis–Kaplan Executive Function System (D-KEFS)), suggesting that education might be associated with a general benefit in organising and scheduling complex responses. If the level of education obtained affects the way in which people think, apply reasoning strategies, approach problems, address situations independently, and work productively (Foxcroft, 2004), then it is possible to

suspect that differences in level of academic qualifications may affect performance on the Executive Functioning Battery, especially in the South African context where historical disparities exist in the provision of education (Foxcroft, 2004).

#### **4.5 Research Participants**

A non-probability sampling method was used in order to gain participants who were willing to participate in the current study. This method ensured that participants were not randomly selected, but through means of purposive sampling (Maree & Pietersen, 2007). Thus, pre-existing patients and individuals with PTSD, diagnosed by a professional clinician, were referred to participate and after confirming their suitability for the study, were utilized. Trauma exposed and non-trauma exposed individuals were also recruited to serve as matched controls. The researcher is aware that it would have been ideal for this research design to have included a matched control group consisting only of healthy individuals who had never experienced a DSM-5 PTSD Criterion A event.

Such ideal conditions exist in studies done by Flaks et al. (2014) who investigated adults exposed to urban violence in São Paulo, Brazil. Stein et al. (2002), in their research among female victims experiencing interpersonal violence, also utilized a matched control group consisting of only healthy individuals. Similar research designs were also employed by Twamley et al. (2004) (230 college students of which 87 participants (38%) never experienced a DSM-5 PTSD Criterion A event), Weniger, Lange, Sachsse, and Irle (2008) (23 young women who had experienced severe childhood sexual or physical abuse, and 25 healthy control subjects (108%)), and Yehuda, Golier, Halligan, and Harvey (2004). The latter, for example, did their research among 62 Holocaust survivors and 40 healthy individuals (64%) not exposed to the Holocaust living in New York, United States of America.



However, given the high reported lifetime traumatic event prevalence rate for South Africa (73.8%), witnessing trauma is common in South Africa. Therefore, the researcher found it difficult to recruit participants who had no exposure to traumatic events at any level. From a sample of 88 participants, only 14 participants (16%) reported no exposure to a DSM-5 PTSD Criterion A event. The participants in both groups were matched according to age, gender, race/ethnicity, years of education, language, and socio-economic status.

Participants for the healthy comparison control group were recruited among members of the public, including from participants' friends and acquaintances. Members were also informed and recruited through written invitations, ads on various social media platforms, flyers distributed to faith and other community centers, as well as through word of mouth. The inclusion and exclusion criteria for the healthy comparison control group remained the same as for the PTSD+ group, except for the absence of a diagnosis of PTSD. All of the participants signed the informed consent form. These sampling methods were approved by the Research Ethics Committee of the Faculty of Humanities from the University of Pretoria.

#### **4.5.1 Sample.**

In this research study two different levels of trauma exposure were investigated: PTSD positive (PTSD+), and trauma and non-trauma exposed healthy comparison control samples who were PTSD negative (PTSD-). The sample as a whole was composed of 50 female and 38 male South African adult participants ( $n = 88$ ) aged between 26 years and 64 years with a mean age of 44.77 ( $SD = 11.25$ ).

The PTSD+ group was composed of 25 female and 19 male participants ( $n = 44$ ) aged between 26 years and 64 years with a mean age of 45.13 ( $SD = 11.28$ ) who were exposed to a DSM-5 PTSD Criterion A event and went on to develop PTSD. The PTSD- group was matched according to age and gender and was composed of 25 female and 19 male participants ( $n = 44$ )

aged between 26 years and 64 years with a mean age 44.40 ( $SD = 11.33$ ) who were or were not victims of trauma without PTSD.

The total sample of 88 South African adults had a higher proportion of women (57%) and White participants (86%), although other racial groups were represented (9% Coloured and 5% Black). The use of White, Coloured, Black, and Indian/Asian as racial classifications is based upon the fact that the South African scheme of racial classification uses skin colour and ancestry as criteria (Khalfani & Zuberi, 2001). Classifying participants according to their skin colour in this research project is done not with the intention of reifying them, but instead with an awareness of the ongoing disparities that remain across these groups even post-apartheid, and their possible differential impact on executive functioning performance in individuals diagnosed with PTSD. Furthermore, 21% of the sample reported English as their home language, with majority of the sample being Afrikaans-speaking (75%). Sotho (2%), Swati (1%), and Xhosa (1%) speaking individuals were in the minority. Most of the participants reported being married or in a stable long-term relationship (77%) with 92% of the sample being employed, with 32% possessing a postgraduate degree. Only 6% of the sample reported receiving an estimated monthly income of less than R6 486. Concerning trauma exposure, 84% of the sample reported exposure to a potentially traumatic event (PTE) with 60% of the sample report being victims of multiple traumas. Witnessing of trauma was the highest reported traumatic event (51.14%) with accidents (40%) second and physical violence (33%) third. This is somewhat in line with the reported prevalence of PTE exposure in South Africa reported previously by Atwoli et al. (2013) in their analysis of the South African Health and Stress Study. They found unexpected death of a loved one (39.2%), physical violence (37.6%), and accidents (31%), as the most common traumatic events experienced in South Africa with witnessing of trauma at 29.5%.

#### **4.5.2 Inclusion criteria.**

Participants were included in the PTSD+ group if they had a history of traumatic life experiences as defined and diagnosed according to the DSM-5 criteria. The participants categorized in the PTSD– group did not meet the diagnostic criteria for PTSD at the time of evaluation and reported no PTSD formal diagnosis in the past.

Participants of both groups had good general health, had no additional diseases expected to interfere with the study procedures, had the ability to understand and sign the informed consent form, were South African adult citizens aged between 20 years and 65 years, and had at least a basic Grade 12 level of formal education. This information was not only obtained by the various professional clinicians who referred individuals to participate in the study, but were also confirmed by the Socio-biographical Questionnaire completed by each participant.

The reason for the lower cut-off criterion of 20 years is that the prefrontal networks that are involved in frontal lobe cognitive functions, continue to develop into late adolescence and reach mature levels during young adulthood (Fuster, 2002a; Kolb & Whishaw, 2009). This ensures that the cognitive functions that contribute most to intellectual maturation (e.g., attention, language, and creativity) are intact, enabling the individual to organise behaviour and cognition into goal-directed structures of action (Fuster, 2002a).

The reasons for the top cut-off criterion of 65 years are associated with the challenges accompanying evaluation of cognitive performance among older adults with PTSD. Older adults tend to possess higher percentages of medical co-morbidities and cognitive deficits, posing challenges specific to rendering diagnoses, formulating cases, and type of treatment (Schuitevoerder et al., 2013). There also appear to be a noticeable symptom overlap between PTSD and late-life cognitive disorders, such as hypervigilance, anxiety, and concentration

difficulties. Moreover, Christensen (2001) found that a larger variability exists between older adults' performance on cognitive tests compared to younger adults. Without applying age-corrected norms or statistical co-variation for age, it is difficult to distinguish normal cognitive aging from PTSD (Hannay & Lezak, 2004). This is because most of the lower test scores associated with PTSD portray similar impairments, which generally forms part of the process of normal cognitive aging (Lupien, Mahue, Tu, Fiocco, & Schramek, 2007; Owens, Baker, Kasckow, Ciesla, & Mohamed, 2005). Thus, by limiting the participants to this age range, the decline in cognitive performance due to age-related degeneration is partially controlled for and minimized.

The requirement for participants to possess a basic Grade 12 qualification ensured that participants possessed an adequate level of literacy that would enable them to read, understand, and complete the assessment material. Participants who received psychotherapy as a means of therapeutic intervention for PTSD not exceeding a period of two months, were also included in the study. This limitation prevented "sudden gains", "the phenomenon of rapid, large decreases in symptoms from one session to another" (Doane, Feeny, & Zoellner, 2010, p. 555). Individuals who had been exposed to multiple (cumulative) traumas were also included in the study as it is suggested that symptom severity rather than the type and frequency of the trauma exposure may be associated with impaired executive functioning (Polak et al., 2012).

#### **4.5.3 Exclusion criteria.**

Participants were excluded from the study based upon factors known to affect neurocognitive functioning, because these factors would interfere with understanding the relationship between executive functioning and PTSD. The exclusion criteria included any significant disease affecting the central nervous system; a history of head trauma followed by the loss of consciousness greater than 10 minutes or requiring the hospitalization for 24 hours or more; known structural brain abnormalities or history of other neurological events; severe

sensory loss that was not compensated (e.g., legal blindness in one or both eyes, colour-blindness, hearing loss requiring a hearing aid, or partial or complete deafness in one or both ears); use of or dependence on psychotropic medication such as antidepressants, antipsychotics, tranquilizers, or mood stabilizers in the last 6 months; active dependence or abuse of alcohol and other substances (excluding caffeine or nicotine) in the past 6 months; or the inability to read and understand the consent form, or where informed consent could not be obtained. Participants were also excluded if they already had a psychiatric diagnosis other than PTSD, such as borderline personality disorder, bipolar disorder, generalized anxiety disorder, obsessive compulsive disorder, or major depressive disorder prior to the traumatic event. However, given that the lifetime co-occurrence of depression and PTSD is 95% (Bleich, Koslowski, Dolev, & Lerer, 1997), it is likely that some participants included in the PTSD groups might meet the criteria for other affective disorders, as is common in PTSD (Yehuda, 2002a). PTSD shares several common symptom features with depression. In particular, diminished interest is part of major depressive symptomatology. Moreover, arousal and reactivity symptom criteria of PTSD, such as problems relating to sleep and concentration are also similar to the symptom criteria of depression (Channon & Green, 1999; Kanagaratnam & Asbjørnsen, 2007; Polak et al., 2012; Shalev et al., 1998). Investigating the mediating relationship between PTSD and executive functioning is beyond the scope of the present research. In the subsequent paragraphs, an overview of the data collection procedures is provided followed by a discussion concerning the respective measurement instruments applied in the current study.

#### **4.6 Data Collection Procedures**

Eighty-eight participants were individually assessed over a period of eight months. Assessments were conducted throughout the Republic of South Africa at participants' places of work, their homes, assessment facilities at the University of Pretoria, and the researcher's

office at his home. During the assessment no one other than the examiner and the examinee were present in the room while the test was being conducted. The researcher received training in the respective assessment materials prior to conducting the research and constant supervision were also provided to ensure that the administration procedures met the test requirements. At all the venues listed, the assessments took place in a quiet, adequately lit, and well-ventilated room, thereby ensuring that distractions or interferences were kept to a minimal.

During the assessments the data collection procedures were done by the participants completing two questionnaires as well as the Executive Functioning Battery. The first questionnaire, the Posttraumatic Stress Diagnostic Scale (PDS) was administered to all the participants including the control group to confirm or deny the diagnosis of PTSD. After completing the PDS, the researcher continued to collect data by administering the Socio-biographical Questionnaire to ascertain inter alia demographic variables, educational-, medical-, psychological-, financial-, and social history. Thereafter, the researcher completed the assessment by administering the Executive Functioning Battery in the following order: The Executive Functioning Index were administered first, followed by the D-KEFS Trail-Making Test, D-KEFS Colour-Word Interference Test, and D-KEFS Tower Test. After the completion of the Executive Functioning Battery each participant were thanked and additional questions were attended to on request. However, the researcher is aware that by administering the cognitive tests in the same order to all participants the possibility may exist that potential confounding effects of order or fatigue on within-subjects performance across tests may have been introduced (Buodo et al., 2011).

By conducting the research in this manner the researcher remained sensitive to the ethical rules of conduct for practitioners registered under the Health Professions Act, 1974. This researcher is therefore of the opinion that the processes and procedures that guided data

collection and analysis pertaining to this research study, fully complied with all the guidelines from the Health Professions Council of South Africa (HPCSA) (Government Gazette, 2006).

#### **4.7 Measurement Instruments**

The measurement instruments used in the current study have already been mentioned earlier in this chapter and will be discussed in detail in the following paragraphs. For the purpose of this discussion, the measurement instruments, namely the Executive Functioning Battery and the Posttraumatic Stress Diagnostic Scale (PDS), are presented according to their respective assessment type: Neuropsychological Performance and Clinical Features.

##### **4.7.1 Assessment of Neuropsychological Performance: Executive Functioning Battery.**

The Executive Functioning Battery consists of two specific measuring instruments namely the Executive Functioning Index (EFI) and certain subtests of the Delis Kaplan Executive Function System (D-KEFS). These two tests contain several subscales that measure different dimensions of frontal lobe functions.

###### **4.7.1.1 *The Executive Function Index (EFI) measuring instrument.***

The Executive Functioning Index (EFI) is a pen and paper test containing a 27 item self-reporting scale, developed to serve as a brief, self-rated measure of executive functioning within a normal adult population (Garner, 2009; Miley & Spinella, 2006; Spinella, 2005). Each of the 27 items is rated on a 5-point Likert-type scale ranging from 1 (not at all) to 5 (very much) during which participants rate how well each of the statements describes them. An example of such an item is “I try to plan for the future”. The scale was designed to measure the five aspects of frontal lobe executive functioning according to Miley and Spinella (2006). The EFI comprises the following five subscales that were determined by exploratory factor analysis:

- Impulse control (IC),
- Strategic planning (SP),
- Organisation (ORG),
- Motivational drive (MD), and
- Empathy (EM).

The subscales are elaborated by Garner (2009) as follows:

- The impulse control subscale refers to items assessing the extent to which an individual reports engaging in inappropriate behaviour (e.g., risk taking, substance abuse, excessive spending) and having difficulty controlling his or her temper.
- The strategic planning subscale emphasises planning, organisation, and response to error-driven feedback.
- The organisation subscale refers to behavioural sequencing, integration of information, and freedom from distractibility.
- The motivational drive subscale assess general levels of energy and enthusiasm for novel tasks (e.g., activity level, drive).
- The empathy subscale focuses on an individual's concern for the well-being of others (e.g., pro-social behaviour, aggressive social stance).

#### *4.7.1.1.1 Scoring procedures.*

The sub-scales are scored as follow: For Motivational drive (MD), each participant's responses are counted on items 1, 4 (reverse order), 7 and 14; for Organisation (ORG) each participant's response on items 2 (reverse order), 6 (reverse order), 17 (reverse order), 22 (reverse order) and 23 (reverse order) are calculated; for Strategic planning (SP) each



participant's response on items 3, 9, 10, 13, 19, 26 and 27 are calculated; for Impulse control (IC) each participant's response on items 5 (reverse order), 11 (reverse order), 15 (reverse order), 20 (reverse order), and 24 (reverse order) are calculated and for Empathy (EM) each participant's response on items 8, 12 (reverse order), 16, 18, 21 and 25 are calculated. An aggregate count for the EFI is also calculated by adding the total of each subscale. The scores obtained from the negative valenced items are inverted so that higher scores on the subscales, as well as the overall score, may reflect improving executive functioning and help control for response pattern bias or response set.

#### *4.7.1.1.2 Psychometric properties.*

The EFI showed acceptable internal consistency reliability, with Cronbach's alpha ( $\alpha$ ) for the scales ranging from 0.70 to 0.82 (Garner, 2009; Miley & Spinella, 2006). The reliability of the EFI was confirmed by (Basson, 2014) in his recent empirical study of frontal lobe functioning and positive affect, during which he determined the Cronbach alpha as 0.748. Furthermore, Miley and Spinella (2006) reported a second-order factor analysis which indicated three higher order factors that is consistent with the executive functions associated with dorsolateral (Strategic Planning, Organisation), orbitofrontal (Impulse Control, Empathy), and medial prefrontal (Motivational Drive) regions. Although the EFI continues to undergo psychometric evaluation and is preliminary in nature, the validity was demonstrated by strong correlations with subscales of the Frontal System Behaviour Scale, Barrat Impulsiveness Scale, and Interpersonal Reactivity Index (Miley & Spinella, 2006). Spinella (2005) reviewed related measures that have been validated by using clinical populations, objective measures of behaviour, and neuroimaging studies.

#### ***4.7.1.2 The Delis Kaplan Executive Function System (D-KEFS) measuring instrument.***

The Delis Kaplan Executive Function System (D-KEFS) (Delis et al., 2001) is a comprehensive battery composed of nine tests which provide a standardized assessment of higher levels of cognitive functions, called executive functions (Shunk, Davis, & Dean, 2006). Most of the D-KEFS tests follow a game-like format without making use of right or wrong feedback procedures in order to reduce unproductive discouragements and frustrations caused by repeated negative feedback during testing. The D-KEFS tests were designed to be administered individually or in complement with the other tests with the administration time varying from 15 minutes for one test to 90 minutes for the entire battery. The results obtained from the D-KEFS can be used to assess the integrity of the frontal systems of the brain, together with determining how the deficits in higher order thinking may impact an individual's functioning (Shunk et al., 2006).

##### ***4.7.1.2.1 Content and administration.***

Despite the D-KEFS consisting out of nine subtests that measure different aspects of frontal lobe executive functioning, the Tower Test, Colour-Word Interference Test, and the Trail Making Test were selected as neuropsychological tests for the purpose of evaluating executive functioning abilities. These tests were chosen not only because they are commonly used clinical measures that clinicians and researchers generally classify as “executive”, but they are also tests developed from theoretical frameworks of frontal lobe functioning proposed by Luria in his classical view of frontal lobe functioning, as well as Norman and Shallice's supervisory attentional system (SAS) (Chan et al., 2008) (Table 0.1). The reason for including these tests are not only based on the fact that they are developed from good theoretical frameworks and align with the underlying theoretical framework of the current study, but they also contain good reliability coefficients (Homack, Lee, & Riccio, 2005).

Furthermore, these tests were also chosen based on their inclusion in previous ecological validity research (Chaytor, Schmitter-Edgecombe, & Burr, 2006; Sullivan, Riccio, & Castillo, 2009). This researcher also acknowledges that the chosen tests can also be classified as tests of attention (Tower Test; Colour-Word Interference Test; Trail Making Test), scanning and visuo-motor tracking (Trail Making Test), and cognitive flexibility (Colour-Word Interference Test; Trail Making Test) (Chaytor et al., 2006; Lezak et al., 2012; Sullivan et al., 2009). Acknowledging that the selected tests are multifaceted and likely measure capacities beyond only executive functioning, this researcher do not consider the tests to be pure measures of executive functioning, but instead have a relatively large executive component. The content and administration of each of the selected tests will be discussed next.

#### *4.7.1.2.2 D-KEFS Tower Test.*

This test is a measure of planning and problem-solving in addition to rule learning, inhibition of impulsive responses, and maintenance of instructional sets (Delis et al., 2001; Lezak et al., 2012). The planning component is best conceptualised as involving an organised sequence of steps, which may be behavioural or cognitive and is goal-directed (Cohen, Bronson, & Casey, 1995). It is assumed that solving the task is best accomplished through strategy use and by planning a sequence of moves (Morris, Miotto, Feigenbaum, Bullock, & Polkey, 1997). This test includes five disks that vary in size from small to large, and a board with three vertical pegs. Each task begins with the examiner placing from two to five disks on the pegs in a predetermined starting position and displaying an ending picture of the tower to be built. The examinee is asked to move the disks across the three pegs to build the target tower in the fewest number of moves possible. In constructing the target towers, the examinee is not allowed to move more than one disk at a time and cannot place a larger disk on top of a smaller disk. To obtain the correct solution, an individual should be able to visualise the solution several steps in advance, placing demands on the prefrontal cortex (Harvey S. Levin et al.,

1994). Despite the role of planning, working memory, and inhibition being emphasised in effective performance on tower tasks (Cohen et al., 1995; Levin et al., 1994; Luciana & Nelson, 1998), traditionally tower tasks have been considered as planning tasks, because an individual is expected to make fewer moves if they plan their course of action before starting to move the disks (Sullivan et al., 2009). A working memory component is also involved because the number of moves that need to be stored in memory to complete each tower task increases as the item difficulty increases.

#### *4.7.1.2.3 D-KEFS Colour-Word Interference Test.*

An individual's ability to switch his/her attention from one stimulus to another can best be observed when two stimuli are in conflict (Funahashi, 2001). During such conditions, a person needs to exert active inhibition, or as Perner and Lang (1999) put it—executive inhibition—of inappropriate responses. The Colour-Word Interference Test can be used to examine this capacity. This test is based on the Stroop (1935) procedure (Shunk et al., 2006). Stroop (1935) developed this classic procedure for studying verbal interference effects, cognitive flexibility, and inhibition. The primary executive function measured by the Stroop (1935) procedure is the inhibition of a more automatic verbal response (reading) in order to generate a conflicting response of naming the dissonant ink colours. This test presents interference in the form of competing responses in which an examinee must attend to the task. The D-KEFS version contains four conditions which grow in complexity.

The Colour-Word Interference test includes two baseline conditions for evaluating key component skills of the higher-level tasks: Basic naming of colour patches (Condition 1) and basic reading of words that denote colours printed in black ink (Condition 2). On the traditional interference task (Condition 3), the examinee must inhibit reading the words denoting colours in order to name the dissonant ink colours in which those words are printed. The fourth

condition requires the examinee to switch back and forth between naming the dissonant ink colours and reading the conflicting words. This condition is a measure of both inhibition and cognitive flexibility.

To perform this test successfully, an individual must pay attention to the relevant stimulus (the colour of the printing) while at the same time inhibiting the irrelevant but prepotent process of stating the printed word. During this test, individuals take longer to answer 'red' when the word 'green' is printed in red ink than when the word red is printed in red ink. Lesion studies have demonstrated that individuals who have sustained lesions to their right dorsolateral prefrontal region appear to perform more response errors (Vendrell et al., 1995). However, fMRI studies have also indicated that subcortical regions have a functional role in the resolution of Stroop interference. Saban, Gabay, and Kalanthroff (2018) suggests that the anterior cingulate cortex monitors conflict situations and directs this information to different structures in the frontal cortex that are involved in conflict resolution.

#### *4.7.1.2.4 D-KEFS Trail Making Test.*

The justification for inclusion of this test is its ability to isolate the basic components of performance (e.g., motor, simple sequencing) from the higher order executive components (e.g., task-switching, multitasking). It is thus an acceptable measure to assess executive functioning because of its contribution of mental flexibility when altering between number and letter sets (Cangoz, Karakoc, & Selekler, 2009; Demakis, 2004; Lezak et al., 2012). This test consists of five conditions which assess number-letter switching, visual-motor sequencing, and a cognitive set shifting procedure. Baseline measure of visual scanning and motor speed are provided for an examiner to quantify and derive normative data for component processes necessary to perform the switching tasks. Additionally, this test provides a measure of visual scanning problems from the other D-KEFS tests. Like most tests involving motor speed and

attention, the Trail Making Test is also highly vulnerable to the effects of brain injury (Aita, Armitage, Reitan, & Rabinovitz, 1947).

#### *4.7.1.2.5 Scoring procedures.*

There are two options available to the examiner when scoring the protocols of the D-KEFS. Each protocol can be hand scored using the examiner's manual, or scoring software is available to provide quick and convenient scoring of the D-KEFS test. Raw scores are tabulated before they are converted to either scaled scores or cumulative percentile ranks. Additionally, scaled scores are collapsed to yield composite scaled scores for each of the nine stand-alone tests. Most raw scores on the D-KEFS were normally distributed which allowed for conversion into scaled scores. Some variables did not fit this format, therefore conversion to cumulative percentile ranks were utilised and conducted to correct for age group differences. A linear and nonlinear regression model was used to derive the scaled scores which allowed for the linear conversion to convert scaled scores to standard scores. After obtaining the scaled scores and the standard scores using the Delis-Kaplan Executive Function System, Crawford, Garthwaite, Sutherland, and Borland's (2011) D-KEFS Supplementary Analysis programme was used to obtain a composite D-KEFS Executive Index Score for each participant. The composite D-KEFS Executive Index Score, included in the statistical analysis, was calculated for each participant by adding the respective achievement scores obtained on the following conditions for each of the D-KEFS subtests administered. The conditions that formed part of the subscales of the Executive Functioning Battery include the D-KEFS Trail Making Test – Condition 4: Number-Letter Switching, D-KEFS Colour-Word Interference Test – Condition 3: Inhibition D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching, and the D-KEFS Tower Test – Total Achievement Score, and D-KEFS Tower Test – Move Accuracy Ratio. The Executive Index Score for the D-KEFS has a mean of 100 and a standard deviation of 10. Normative data is attainable for a wide spectrum of cognitive variables, including the

following: Component skills, problem-solving strategies, response reaction time, ratio measures, and different error types. These optional measures provide insight for generating possible causes for deficits and guide interpretation (Shunk et al., 2006).

#### *4.7.1.2.6 Psychometric properties.*

The internal consistency coefficients for all three these individual D-KEFS tests range from moderate to high, with the Spearman correlation coefficient ( $r$ ) ranging between .43 to .84 (Tower Test), .62 to .86 (Colour-Word Interference Test), and .57 to .81 (Trail Making Test) (Delis et al., 2001). Homack et al. (2005) also indicates that the D-KEFS has satisfactory validity.

### **4.7.2 Assessment of Clinical Features: Posttraumatic Stress Diagnostic Scale (PDS).**

The PDS was developed by Edna Foa to provide a brief but reliable self-report measure of PTSD for use in both clinical and research settings (McCarthy, 2008). The PDS intends to screen for the presence of PTSD in patients who have identified themselves as victims of a traumatic event or to assess symptom severity and functioning in patients already identified as suffering from PTSD. The PDS is a self-administered questionnaire, which can be completed within 10-15 minutes and requires at least an English reading age of 13 years. The questions within the PDS relate to frequency of distressing and intrusive thoughts, post-trauma avoidance, and hyper arousal (Foa, Cashman, Jaycox, & Perry, 1997). The discontinued long version of the PDS had 49 items. The short version (applied in this study) consists of 17 items. This version consists of a short checklist that identifies potentially traumatizing events. The respondents indicate which of these events have troubled them in the last month, after which they relate their response to this event to determine whether the DSM-5 criteria are met. Using a four point scale, the respondents then rate 17-items that represent the cardinal symptoms of PTSD experienced in the past 30 days (Foa et al., 1997; McCarthy, 2008). Additionally, the

PDS includes a symptom severity score that ranges from 0 to 51, obtained by adding up the individual's responses of the selected items.

#### *4.7.2.1.1 Psychometric properties.*

The PDS has high face validity (Foa et al., 1997; McCarthy, 2008), with a coefficient alpha ( $\alpha$ ) of 0.92. The reason for the high validity is that items directly reflect the experience of PTSD (McCarthy, 2008). The test-retest reliability was also highly satisfactory for a diagnosis of PTSD over a two to three week period (kappa ( $\kappa$ ) = 0.74) (Foa et al., 1997; McCarthy, 2008). McCarthy (2008) reports that analysis revealed a valid and reliable 82% agreement between diagnosis using the PDS and the Structured Clinical Interview for DSM. Furthermore, the PDS is validated on samples aged between 18 to 65 years (McCarthy, 2008). All the participants included in the study falls within this parameter. Concluding Chapter 4, a discussion regarding the data analysis and statistical measures applied in the current study will take place. In the subsequent section the chosen methods for data analyses will be discussed.



Table 0.1. Overview of executive function measures and processes

Neuropsychological measure	Executive functioning processes	Definition	Frontal lobe theory
<i>D-KEFS Tower Test</i>			
Move discs from one peg to another is as few moves as possible without placing a larger disc on top of a smaller disc.	Planning Working memory	Higher order problem-solving necessary for the generation of behaviour to achieve a goal.  Executive planning requires the ability to anticipate change, respond objectively, generate and select alternatives, and sustain attention.	Norman and Shallice's Supervisory Attentional System
<i>D-KEFS Colour-Word Interference Test</i>			
Separates blocks of word reading, colour naming, and incongruent trails (e.g., "red" written in blue ink).	Inhibition	Suppressing or resisting a prepotent (automatic) response in order to make a less automatic but task-relevant response.	Luria's classical view of frontal lobe functioning.  Norman and Shallice's Supervisory Attentional System.
<i>D-KEFS Trail Making Test</i>			
Alternately connect letters and number sets in sequence.	Cognitive flexibility Motor speed Visual scanning attention	Switching between task sets or response rules.	Norman and Shallice's Supervisory Attentional System.

*Note.* Adapted from "Advancing understanding of executive function impairments and psychopathology: Bridging the gap between clinical and cognitive approaches" by H.R. Snyder, A. Miyake, and B.L. Hankin, 2015, *Frontiers in Psychology*, 6, pp. 3-6. Copyright 2015 by The Authors.

## **4.8 Data Analyses and Statistical Measures**

The next section focusses on intended statistical measures for data analyses pertaining to the data that was collected using the Executive Functioning Battery.

### **4.8.1 Analysis of Normal Distribution.**

The normality of subscale distribution has implications for the type of statistical analysis methods that can be used to analyse a set of data. Various statistical measures are only applicable for analysing data that is considered to be normally distributed. The skewness and kurtosis of subscale distributions are observed and compared to the theoretical cumulative distribution for normally distributed data. Subscale distributions of the following tests will be analysed:

- The five subscales of the Executive Function Index (EFI) which consist of Motivational Drive, Organisation, Strategic Planning, Impulse Control, and Empathy.
- The five subscales of the Delis Kaplan Executive Function System (D-KEFS), which consist of the Trail Making Test – Condition 4: Number Letter Switching; Colour-Word Interference Test – Condition 3: Inhibition; Colour-Word Interference Test – Condition 4: Inhibition/Switching; Tower Test – Total Achievement Score; and the Tower Test – Move Accuracy Ratio.

### **4.8.2 Wilcoxon Signed-rank Test.**

To test the hypothesis whether a chronic stress condition, such as PTSD, affects frontal lobe executive functioning, Wilcoxon Signed-rank test will be performed between two groups of participants categorised according to their level of trauma exposure (PTSD+ versus PTSD-). The non-parametric Wilcoxon Signed-rank test will also will be applied to establish whether statistically significant differences exist between the two groups in terms of gender. Although it is evident in the present study that a larger proportion of participants were female

(57%), the size of the male group participants (43%) were still however considered sufficient. With regards to race, white participants over represented (86%), leaving coloured (9%) and black (5%) participants to a great extent underrepresented. As a result thereof, statistical analyses pertaining to racial differences will be excluded from the data analysis. The reason therefore is that the two groups of participants cannot be meaningfully compared to each other based on their performances on the respective measurement instruments in terms of race.

#### **4.8.3 Kruskal-Wallis H Test.**

To determine whether statistically significant differences existed between the two groups in terms of education, the non-parametric Kruskal-Wallis H test will also be applied to investigate the association between level of academic qualification and executive functioning performance between the PTSD+ and the PTSD– groups on the Executive Functioning Battery. The Kruskal-Wallis H Test is a rank-based non-parametric test that can be used to determine if statistically significant differences exist between two or more groups of an independent variable on a continuous or ordinal dependent variable.

Where overall differences between the means of the groups existed, in other words when the null-hypothesis can be rejected that the means are the same, a multiple comparison post hoc correction test was applied to identify which particular differences between pairs of means were significant, while controlling for the experiment-wise error rate. In order to establish which groups were significantly different in terms of their group means from the other groups, a multiple pair-wise comparison between groups were done by using the Dunn-Bonferroni Test. Because the Kruskal-Wallis H test is an omnibus test statistic and cannot determine which groups differ significantly from the other, only that at least two groups differ significantly, post hoc tests allow us to distinguish which specific group differed from each other.

Parametric tests (e.g., *t*-Tests for independent samples and One-way Analysis of Variance (ANOVA)) will also be used on the one hand to confirm the results of the non-parametric tests and on the other hand because they are dependent on normal distribution of scores.

For the current study the level of significance or probability that the null hypothesis will be rejected was set at  $\alpha < 0.05$ . Although one-sided hypothesis is usually investigated at a more stringent probability level and reduces the risk of a Type 1 error, it also imposes excessive demands on research results. Too high probability levels create the danger that only enormous effect sizes would at all contribute to the rejection of the null hypothesis. Consequently, there is usually an attempt to create a balance between the risk of Type 1 errors and the requirements of hypothesis testing. Alpha levels of 0.05, 0.01, and 0.001 are considered acceptable as they provide relatively low risk of errors without placing excessive demands on research results (Salkind, 2008). The data were analysed using SAS statistical software, version 9.4 for Windows.

#### **4.9 Chapter Summary**

In this chapter, the relevance of a positivist approach to the research question was investigated. Furthermore, the impact regarding the choice of methodological paradigm on the selection of participants and the collection and processing of data were addressed. The aim, objectives, and justifications were also discussed by addressing the specific and general objectives of the current study. Numerous motivations were also provided, explaining the significant importance for conducting a study of this nature within the South African context. The methods of data collection were described by focusing on the compilation of the sample, the measuring instruments, and the procedures that were followed to obtain the data. The study participants were described according to characteristics such as age, gender, race, and

language, level of academic qualification, employment, marital status, estimated monthly income, and type of trauma exposure. Thereafter, the composition of the measuring instruments, scoring procedures, and the psychometric properties of the measuring instruments applied in the current study were provided. This chapter concluded with a discussion about the data analyses and statistical measures which will be implemented for the processing of the data. The next chapter will focus on reporting results as derived from the statistical processing of the data.

## CHAPTER 5

### RESULTS

*“One man’s constant is another man’s variable.” – Alan Perlis (1922 – 1990)*

The purpose of the chapter is to outline the results obtained from the various statistical analyses. Firstly, the focus will be on normality of the various subscale distributions. Thereafter, a discussion will follow regarding the differences between the two groups related to their demographic information concerning gender and education. Lastly, this chapter will conclude with a discussion about the differences in neuropsychological performance between the two respective groups, with specific reference to executive functioning performance on the ten subscales of the Executive Functioning Battery.

#### **5.1 Normality of Subscale Distributions**

In order to test the normality of the various subscale distributions, the One-Sample Kolmogorov-Smirnov test and the Shapiro-Wilk test was applied to the data. The rationale for conducting these tests of normality are to determine whether the intended statistical analysis techniques are appropriate. No hypotheses in terms of the normality of distributions were formulated. Subsequently, the normality of the subscale distributions are presented for the D-KEFS and EFI in Table 0.1 and Table 0.2.

Table 0.1. Distribution normality of the One-Sample Kolmogorov-Smirnov Test and Shapiro-Wilk Test for the subscales of the D-KEFS

Measure	Subscale 1: Number-Letter Switching		Subscale 2: Inhibition		Subscale 3: Inhibition/Switching		Subscale 4: Total Achievement Score		Subscale 5: Move Accuracy Ratio	
	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
	10.40	11.00	10.11	10.00	10.22	11.00	10.84	11.00	8.26	8.00
	Sig.		Sig.		Sig.		Sig.		Sig.	
Kolmogorov-Smirnov	0.01*		0.01*		0.01*		0.01*		0.01*	
Shapiro-Wilk	0.0001**		0.0012**		0.0009**		0.0488*		0.0067**	

*Note.* Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ . Number-Letter Switching = Trail Making Test – Condition 4: Number-Letter Switching; Inhibition = Colour-Word Interference Test – Condition 3: Inhibition; Inhibition/Switching = Colour-Word Interference Test – Condition 4: Inhibition/Switching; Total Achievement Score = Tower Test Total Achievement Score; Move Accuracy Ratio = Tower Test Move Accuracy Ratio.

Table 0.2. Distribution normality of the One-Sample Kolmogorov-Smirnov Test and Shapiro-Wilk Test for the subscales of the EFI

Measure	Subscale 1: Motivational Drive		Subscale 2: Organisation		Subscale 3: Strategic Planning		Subscale 4: Impulse Control		Subscale 5: Empathy	
	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
	11.02	11.00	11.37	12.00	19.18	19.00	11.72	12.00	19.12	20.00
	Sig.		Sig.		Sig.		Sig.		Sig.	
Kolmogorov-Smirnov	0.01*		0.0225*		0.0306*		0.0339*		0.01*	
Shapiro-Wilk	0.0107*		0.153		0.0013**		0.0787		0.0013**	

Note. Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ .



### **5.1.1 Normality of distribution of the Delis Kaplan Executive Function System (D-KEFS).**

According to Table 0.1, there are statistically significant  $p$ -values for both tests of normality for each of the five D-KEFS subscales. This indicates that the subscales of the D-KEFS are not normally distributed ( $p < 0.05$ ).

### **5.1.2 Normality of distribution of the Executive Functioning Index (EFI).**

According to Table 0.2, there are statistically significant  $p$ -values for each of the five EFI subscales according to the One-Sample Kolmogorov-Smirnov test. However, the Shapiro-Wilk test found only statistically significant  $p$ -values for the Motivational Drive ( $p = 0.0107$ ), Strategic Planning ( $p = 0.0013$ ), and Empathy ( $p = 0.0013$ ). This support the findings of the One-Sample Kolmogorov-Smirnov test, indicating that these subscales are not normally distributed. The Shapiro-Wilk test did however demonstrate that the Organisation ( $p = 0.1530$ ) and the Impulse Control ( $p = 0.0787$ ) subscales are normally distributed.

However, despite the fact that the tests for normality revealed that most of the subscales of the Executive Functioning Battery did not follow strict normal distributions, they were still symmetric. Therefore, the non-parametric Wilcoxon Signed-rank test is an appropriate statistical measure to test for equal means between the PTSD+ group and the PTSD- group. Non-parametric tests involve distribution-free analyses, which is useful as many of the subscales did not demonstrate strict normal distributions. For completeness sake due to the sample size ( $n = 88$ ), the  $t$ -test  $p$ -values for equal means for independent samples are also reported, and because the sample size is relatively large the analyses are robust. Where meaningful variables demonstrate statistically significant results, histograms and boxplots are provided.

## 5.2 Demographic Information

For the current study two socio-demographic variables were included in the statistical analyses: Gender and education. Gender is categorised according to male and female, while education is classified into four categories depending on the level of academic qualifications obtained: Grade 12, Diploma, Degree, and Postgraduate Degree. Table 0.3 provides an overview of the demographic characteristics for each of the two respective groups.

Table 0.3. Demographic characteristics of the two groups (PTSD+ and PTSD–)

		PTSD+ (n = 44)		PTSD– (n = 44)	
		Mean	SD	Mean	SD
Age		45.13	11.28	44.40	11.33
		PTSD+		PTSD–	
		n (%)		n (%)	
Education	Grade 12	12 (27.27)		6 (13.64)	
	Diploma	13 (29.55)		8 (18.18)	
	Degree	12 (27.27)		9 (20.45)	
	Post-graduate	7 (15.91)		21 (47.73)	
Gender	Male	19 (43.18)		19 (43.18)	
	Female	25 (56.82)		25 (56.82)	

Note. SD = Standard Deviation. n = number of participants. % = percentage of participants.

### 5.2.1 Comparisons in terms of gender differences.

The non-parametric Wilcoxon Signed-rank test was conducted to compare whether there were gender differences in frontal lobe executive functioning between two groups of participants with different levels of trauma exposure. Concerning the comparisons made for the present study, the parametric *t*-test for independent samples was performed each time to verify the findings of the non-parametric test. Table 0.4 to Table 0.6 report the results of gender differences in frontal lobe executive functioning measured by the D-KEFS and the EFI. The order of the results will be provided by firstly comparing gender differences for the entire

sample. Thereafter, gender differences within the PTSD+ group will be compared, followed by gender differences within the PTSD– group.

### 5.2.1.1 Comparisons for the entire sample in terms of gender.

Null hypothesis: Mean score for each variable between male and female is equal.

Table 0.4. Gender differences in frontal lobe executive functioning (D-KEFS and EFI) in the entire sample ( $n = 88$ ).

Measure	Male ( $n = 38$ )			Female ( $n = 50$ )			Z	Wilcoxon Sig.	t-Test Sig.
	Mdn	M	SD	Mdn	M	SD			
Number-Letter Switching	11	11.13	1.61	10	9.86	2.89	-2.43	0.0870	0.0106
Inhibition	10	10.00	3.09	10	10.20	2.82	0.32	0.8789	0.7528
Inhibition/Switching	10.5	10.21	2.32	11	10.24	2.88	0.05	0.6625	0.9589
Total Achievement Score	11	10.97	2.02	10	10.74	2.52	-0.47	0.6349	0.6408
Move Accuracy Ratio	8	8.29	2.44	8	8.24	2.36	-0.10	0.9425	0.9237
Motivational Drive Organisation	12	11.62	3.10	11	10.56	2.92	-1.66	0.0971	0.1004
Strategic Planning	12	12.34	3.82	11	10.64	4.68	-1.83	0.1826	0.0714
Impulse Control	19.5	19.32	3.47	19	19.08	4.80	-0.26	0.9630	0.7898
Empathy	11.5	11.53	3.49	12	11.88	3.81	0.45	0.7326	0.6558
Empathy	18.5	18.00	3.15	20.5	19.98	3.27	2.86	0.0032**	0.0053**

Note. Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ .  $n$  = number of participants. Mdn = Median; M = Mean; SD = Standard Deviation. *Number-Letter Switching* = Trail Making Test – Condition 4: Number-Letter Switching; *Inhibition* = Colour-Word Interference Test – Condition 3: Inhibition; *Inhibition/Switching* = Colour-Word Interference Test – Condition 4: Inhibition/Switching; *Total Achievement Score* = Tower Test Total Achievement Score; *Move Accuracy Ratio* = Tower Test Move Accuracy Ratio.

The Wilcoxon Signed-rank test was conducted on the entire sample of 88 participants to determine if there were gender differences in executive functioning performance on an Executive Functioning Battery. The male group consisted of 38 participants while the female group consisted of 50 participants. The results of the Wilcoxon Signed-rank test specifically indicated that Empathy was statistically significantly ( $p < 0.01$ ) higher in female participants ( $Mdn = 20.5$ ) than in male participants ( $Mdn = 18.5$ ),  $Z = 2.86$ ,  $p = 0.0032$ ,  $r = 0.30$ . Concerning gender differences in frontal lobe executive functioning, the  $t$ -test for independent samples

confirmed the results of the parametric analysis, namely that gender differences in Empathy are present between male ( $M = 18, SD = 3.15$ ) and female ( $M = 19.98, SD = 3.27$ ) participants,  $p = 0.0053$ . Figure 0.1 below provides a visual representation of the distribution of the performance scores for the entire sample on the Empathy subscale. Therefore, according to these results, hypothesis 2.10 is confirmed, but hypotheses 2.1 – 2.9 remain unconfirmed.

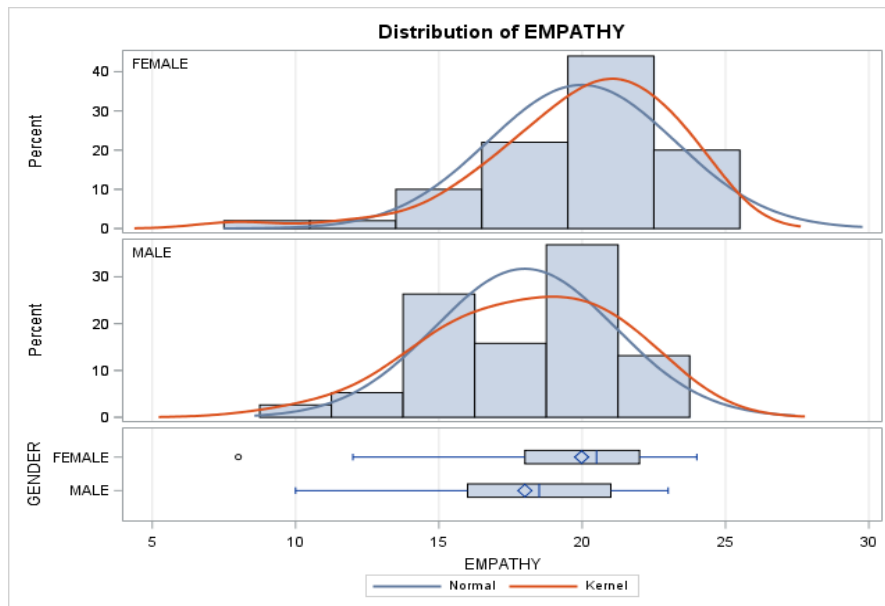


Figure 0.1. Histograms and Boxplots illustrating the distribution of performance scores for the Empathy subscale for the entire sample

### 5.2.1.2 Comparisons for the PTSD+ group in terms of gender.

Null hypothesis: Mean score for each variable below between male and female participants diagnosed with PTSD is equal.

Table 0.5. Gender differences in frontal lobe executive functioning (D-KEFS and EFI) among the PTSD+ sample ( $n = 44$ )

Measure	Male ( $n = 38$ )			Female ( $n = 50$ )			Z	Wilcoxon Sig.	t-Test Sig.
	Mdn	M	SD	Mdn	M	SD			
Number-Letter Switching	11	10.63	1.46	9	8.92	3.51	-1.99	0.1838	0.0527
Inhibition	10	9.47	3.22	9	9.08	2.89	-0.43	0.4877	0.6720
Inhibition/Switching	9	9.58	2.39	9	9.16	3.06	-0.49	0.6934	0.6249
Total Achievement Score	10	10.37	2.17	10	10.20	2.72	-0.22	0.8572	0.8259
Move Accuracy Ratio	7	7.42	2.04	8	7.64	2.14	0.34	0.6120	0.7331
Motivational Drive	11	10.00	3.09	9	9.28	2.88	-0.80	0.4896	0.4306
Organisation	10	10.11	3.33	7	7.76	4.09	-2.04	0.0682	0.0479*
Strategic Planning	18	17.37	3.09	18	17.72	5.77	0.24	0.6685	0.8114
Impulse Control	10	9.84	3.18	11	10.72	3.76	0.82	0.4179	0.4177
Empathy	17	17.21	3.22	21	20.08	3.38	2.85	0.0022**	0.0068**

Note. Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ .  $n$  = number of participants. Mdn = Median; M = Mean; SD = Standard Deviation. *Number-Letter Switching* = Trail Making Test – Condition 4: Number-Letter Switching; *Inhibition* = Colour-Word Interference Test – Condition 3: Inhibition; *Inhibition/Switching* = Colour-Word Interference Test – Condition 4: Inhibition/Switching; *Total Achievement Score* = Tower Test Total Achievement Score; *Move Accuracy Ratio* = Tower Test Move Accuracy Ratio.

Wilcoxon Signed-rank tests were also conducted to determine if gender differences occurred in a sample of 44 participants diagnosed with PTSD in terms of their performance on an Executive Functioning Battery. The PTSD+ sample consisted of 19 male and 25 female participants diagnosed with PTSD. Each participant was purposefully assigned to the PTSD+ group based on their clinical diagnosis. The results of the Wilcoxon Signed-rank test indicated that within the PTSD+ sample, females ( $Mdn = 21$ ) obtained statistically significantly higher scores ( $p < 0.01$ ) for Empathy in comparison to their male counterparts ( $Mdn = 17$ ),  $Z = 2.85$ ,  $p = 0.0022$ ,  $r = 0.43$ . In addition,  $t$ -tests for independent samples confirmed the results of the parametric analysis, by also indicating that gender differences in Empathy occurred between male ( $M = 17.21$ ,  $SD = 3.22$ ) and female ( $M = 20.08$ ,  $SD = 3.38$ ) participants in the PTSD+ sample,  $p = 0.0068$ .

Although the results from the Wilcoxon Signed-rank test did not indicate statistically significant gender differences for the Organisation subscale of the EFI between male and female participants diagnosed with PTSD, the independent sample *t*-test did however indicate statistically significant results. Independent sample *t*-tests showed that males ( $M = 10.11$ ,  $SD = 3.33$ ) reported higher scores for Organisation than females with PTSD ( $M = 7.76$ ,  $SD = 4.09$ ),  $p = 0.0479$ . The Shapiro-Wilk test for normality, found that the Organisation subscale demonstrated normally distributed scores. Thus, the assumption that the data is normally distributed is not violated, making the *t*-test for independent samples an adequate measure for analysing gender differences for the Organisation subscales. The *t*-test for independent samples is a parametric test, which assumes normality of data. Figure 0.2 and Figure 0.3 provide visual illustrations of the distribution of the performance scores for the PTSD+ group on the Empathy subscale and the Organisation subscale.

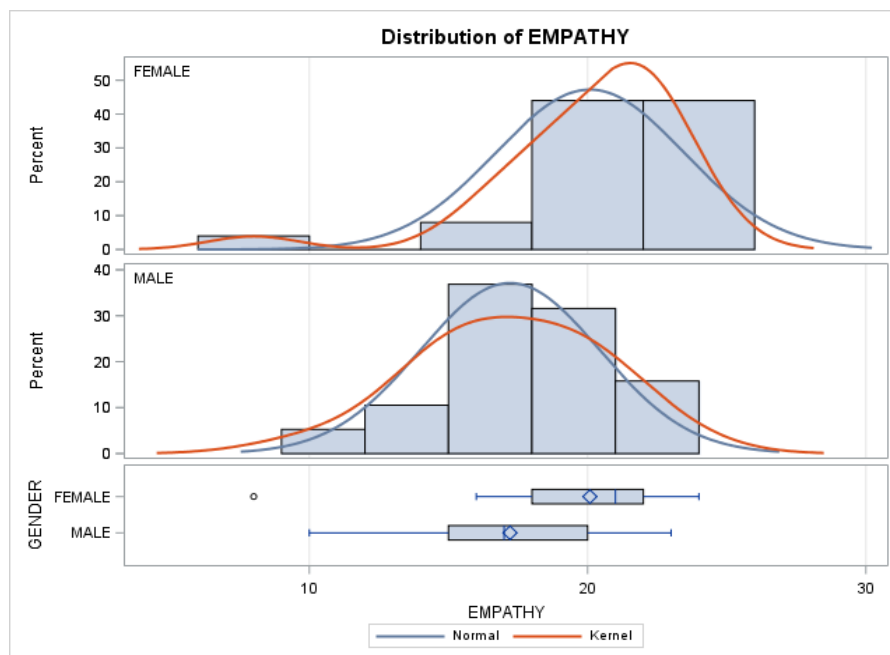
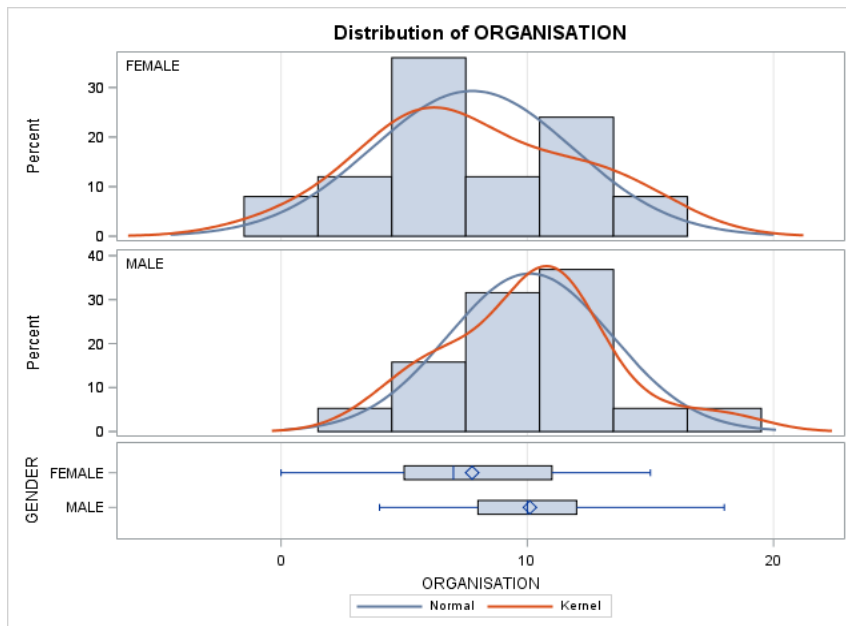


Figure 0.2. Histograms and Boxplots illustrating the distribution of performance scores for the Empathy subscale for the PTSD+ sample.



*Figure 0.3. Histograms and Boxplots illustrating the distribution of performance scores for the Organisation subscale for the PTSD+ sample*

Based on these findings it is evident that hypotheses 3.7 and 3.10 are confirmed, but hypotheses 3.1 – 3.6 and 3.8 – 3.9 are not confirmed.

### **5.2.1.3 Comparisons for the PTSD– group in terms of gender.**

Null hypothesis: Mean score for each variable below between male and female participants without PTSD is equal.

Table 0.6. Gender differences in frontal lobe executive functioning (D-KEFS and EFI) among the PTSD– sample ( $n = 44$ )

Measure	Male ( $n = 19$ )			Female ( $n = 25$ )			Z	Wilcoxon Sig.	t-Test Sig.
	Mdn	M	SD	Mdn	M	SD			
Number-Letter Switching	12	11.63	1.64	11	10.80	1.71	-1.63	0.1719	0.1112
Inhibition	11	10.53	2.95	12	11.32	2.30	1.00	0.3479	0.3218
Inhibition/Switching	11	10.84	2.12	12	11.32	2.25	0.72	0.2890	0.4779
Total Achievement Score	12	11.58	1.71	11	11.28	2.23	-0.49	0.6390	0.6294
Move Accuracy Ratio	9	9.16	2.54	9	8.84	2.46	-0.42	0.6841	0.6779
Motivational Drive	14	13.26	2.13	12	11.84	2.39	-2.05	0.0587	0.0469*
Organisation	14	14.58	2.89	14	13.52	3.28	-1.12	0.5512	0.2711
Strategic Planning	21	21.26	2.68	20	20.44	3.14	-0.92	0.3047	0.3647
Impulse Control	13	13.21	2.97	12	13.04	3.56	-0.17	0.8957	0.3867
Empathy	19	18.79	2.94	20	19.88	3.22	1.16	0.2526	0.2544

Note. Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ .  $n$  = number of participants. Mdn = Median; M = Mean; SD = Standard Deviation. *Number-Letter Switching* = Trail Making Test – Condition 4: Number-Letter Switching; *Inhibition* = Colour-Word Interference Test – Condition 3: Inhibition; *Inhibition/Switching* = Colour-Word Interference Test – Condition 4: Inhibition/Switching; *Total Achievement Score* = Tower Test Total Achievement Score; *Move Accuracy Ratio* = Tower Test Move Accuracy Ratio.

Wilcoxon Signed-rank tests were also conducted to determine if there were gender differences in frontal lobe executive functioning performance on an Executive Functioning Battery between 44 participants without PTSD. The PTSD– sample consisted of 19 male and 25 female trauma and non-trauma exposed participants who were purposefully assigned to the PTSD– group, because these participants did not meet the DSM-5 diagnostic criteria for PTSD at the time of assessment. Although the Wilcoxon Signed-rank tests did not demonstrate meaningful gender differences, the results of the  $t$ -tests for independent samples did however specifically indicate statistically significant gender differences between male ( $M = 13.26$ ,  $SD = 2.13$ ) and female ( $M = 11.84$ ,  $SD = 2.39$ ) participants of the PTSD– group on the Motivational Drive subscale of the Executive Functioning Index,  $p = 0.0469$ . It is however important to mention that, both tests of normality indicated that the Motivational Drive did not demonstrate a normal distribution of scores. As a result, the assumption of normality is violated for the  $t$ -



test and the null hypothesis that the means are the same is not rejected. Thus, there appear to be no statistically significant results with regard to gender differences in respect of frontal lobe executive functioning between male and female participants without PTSD. Given these results, it can be concluded that hypotheses 4.1 – 4.10 are not confirmed. In the following section, education will be addressed by examining whether different levels of academic qualifications affects frontal lobe executive functioning performance differently on an Executive Functioning Battery.

### **5.2.2 Comparisons in terms of education.**

Depending on the distribution of the data, the non-parametric Kruskal-Wallis H test was applied to compare the effects of education on frontal lobe executive functioning across two groups with different levels of trauma exposure. One-way Analysis of Variance (ANOVA) was performed in conjunction with the Kruskal-Wallis H test to verify the findings of the non-parametric test. Table 0.7 reports the results regarding the effect of education on frontal lobe executive functioning measured by the D-KEFS and EFI according to the level of academic qualification (e.g., Grade 12, Diploma, Degree, and Postgraduate).

#### ***5.2.2.1 Comparisons for entire sample in terms of qualification.***

Null hypothesis: Mean scores by qualification for each of the variables below are equal.

Table 0.7. Educational differences in frontal lobe executive functioning (D-KEFS and EFI) by academic qualification (n = 88)

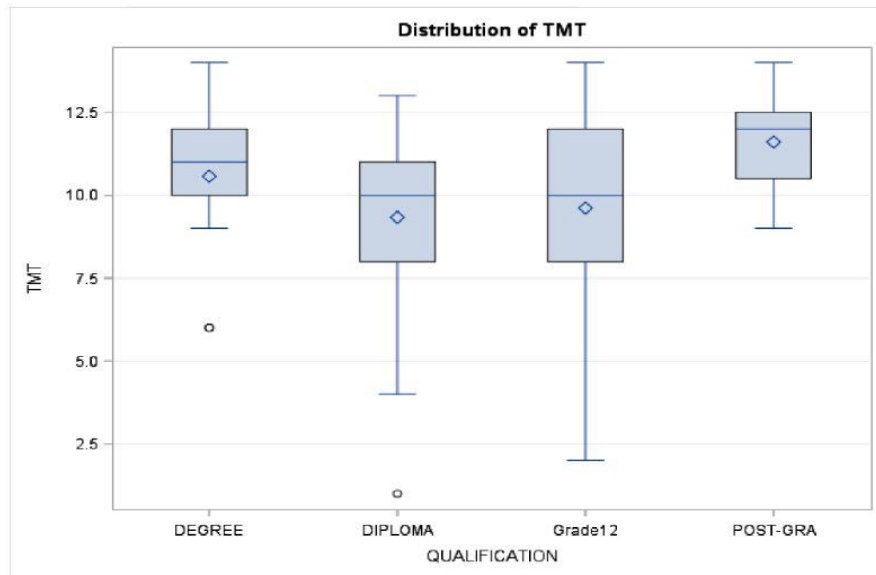
Measure	U	F	Kruskal-Wallis H Sig.	ANOVA Sig.
Number-Letter Switching	11.62	4.61	0.0088**	0.0049**
Inhibition	7.89	2.68	0.0483*	0.0521
Inhibition/Switching	9.24	3.28	0.0262*	0.0250*
Total Achievement Score	3.40	1.06	0.3343	0.3697
Move Accuracy Ratio	3.23	1.14	0.3568	0.3366
Motivational Drive	10.21	4.10	0.0168*	0.0092**
Organisation	13.47	5.14	0.0037**	0.0026**
Strategic Planning	8.54	2.46	0.0360*	0.0681
Impulse Control	4.62	1.31	0.2017	0.2760
Empathy	0.14	0.04	0.9861	0.9903

Note. Sig. = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ . *Number-Letter Switching* = Trail Making Test – Condition 4: Number-Letter Switching; *Inhibition* = Colour-Word Interference Test – Condition 3: Inhibition; *Inhibition/Switching* = Colour-Word Interference Test – Condition 4: Inhibition/Switching; *Total Achievement Score* = Tower Test Total Achievement Score; *Move Accuracy Ratio* = Tower Test Move Accuracy Ratio.

The Kruskal-Wallis H test indicated that statistically significant educational differences were found on the following subscales of the Executive Functioning Battery: Trail Making Tests – Condition 4: Number-Letter Switching ( $X^2(3, n = 88) = 11.62, p = 0.0088$ ); Colour-Word Interference Test – Condition 3: Inhibition ( $X^2(3, n = 88) = 7.89, p = 0.0483$ ); Colour-Word Interference Test – Condition 4: Inhibition/Switching ( $X^2(3, n = 88) = 9.24, p = 0.0262$ ); Motivational Drive ( $X^2(3, n = 88) = 10.21, p = 0.0168$ ); Organisation ( $X^2(3, n = 88) = 13.47, p = 0.0037$ ); and Strategic Planning ( $X^2(3, n = 88) = 8.54, p = 0.0360$ ).

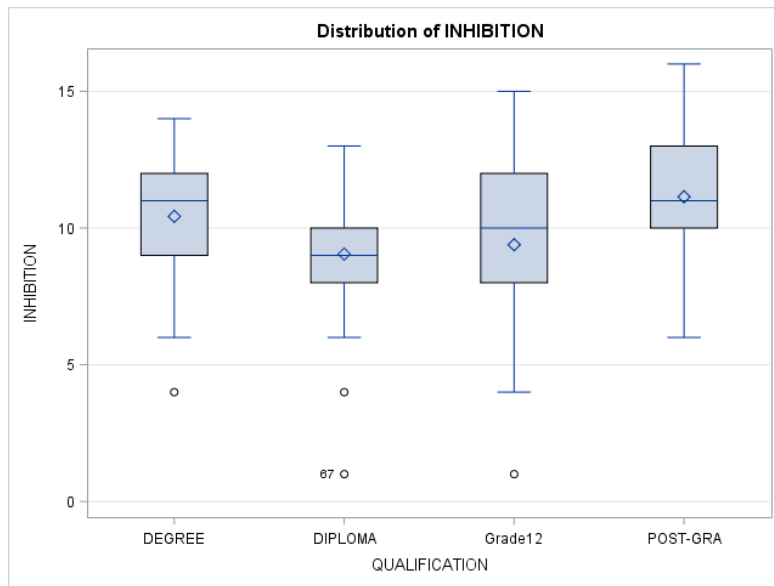
The results of the Dunn-Bonferroni post hoc correction test demonstrated statistically significant differences between participants with a Diploma and participants in possession of a Post-graduate degree regarding their executive functioning performance scores on the Trail Making Test – Condition 4: Number-Letter Switching subscale ( $p = 0.0066$ ). In addition, post hoc test results also revealed a statistically significant difference between participants with a Grade 12 academic qualification versus participants with a Post-graduate degree in terms of their executive functioning performance on the Number-Letter Switching subscale ( $p =$

0.0309). **Error! Reference source not found. Error! Reference source not found.** provides a visual illustration of the distribution of executive functioning performance scores for the Trail Making Test – Condition 4: Number-Letter Switching subscale by level of academic qualification.



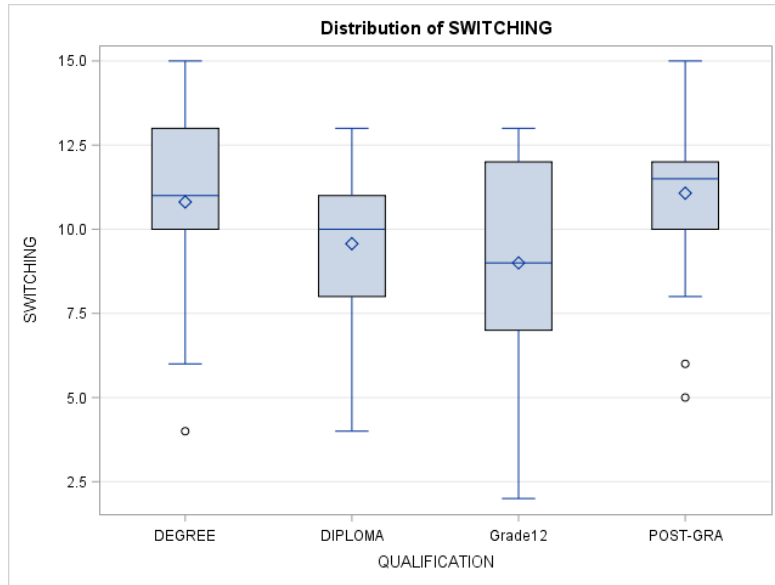
*Figure 0.4. Boxplot representing the executive functioning performance scores for Trail Making Test – Condition 4: Number-Letter Switching by level of academic qualification*

In terms of performance on the Colour-Word Interference Test – Condition 3: Inhibition, post hoc test results found a statistically significant difference between participants with a Diploma and participants with a Post-graduate degree ( $p = 0.0319$ ) (Figure 0.5).



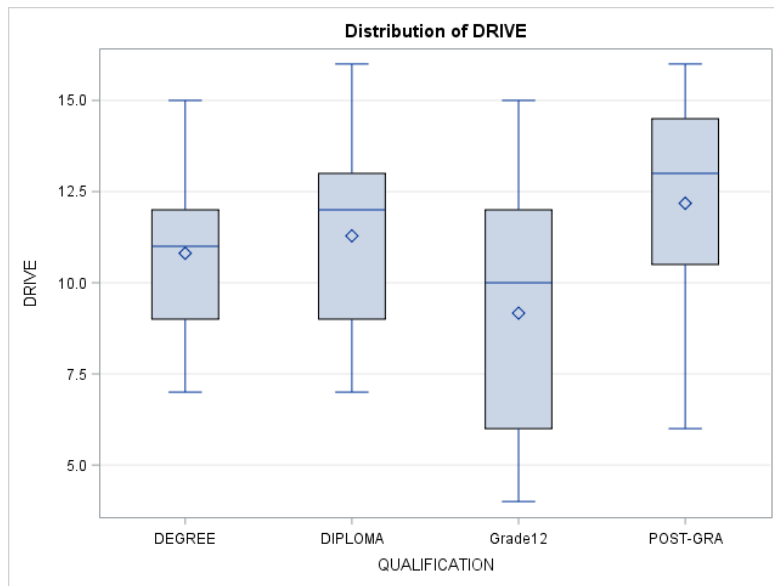
*Figure 0.5. Boxplot representing the executive functioning performance for Colour-Word Interference Test – Condition 3: Inhibition by level of qualification*

Statistically significant differences in performance scores for the Colour-Word Interference Test – Condition 4: Inhibition/Switching were also found between individuals with a Post-graduate degree and a Grade 12 academic qualification ( $p = 0.0406$ ) (Figure 0.6).



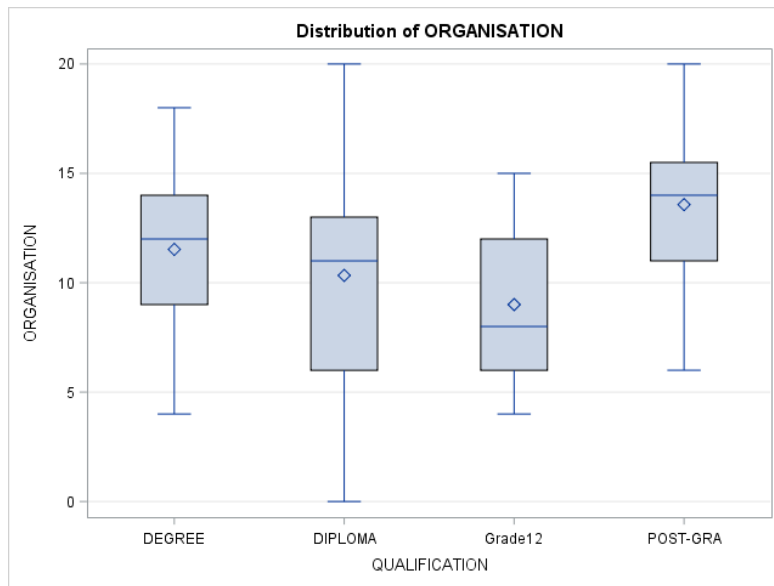
*Figure 0.6. Boxplot representing the executive functioning performance for Colour-Word Interference Test – Condition 4: Inhibition/Switching by level of qualification*

Participants with a Post-graduate degree appeared to report statistically significantly higher scores on the Motivational Drive subscale in comparison to participants with only a Grade 12 academic qualification ( $p = 0.0046$ ) (Figure 0.7).



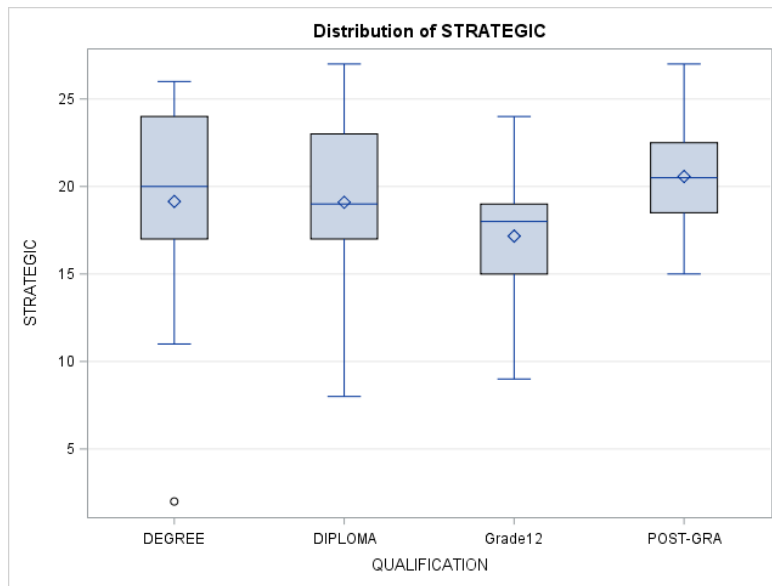
*Figure 0.7. Boxplot representing the executive functioning performance for Motivational Drive by level of qualification*

In terms of the Organisation subscale of the EFI, statistically significant educational differences were found between participants with a Post-graduate degree and Diploma ( $p = 0.0378$ ) including between participants with a Grade 12 academic qualification and Post-graduate degree ( $p = 0.0023$ ) (Figure 0.8).



*Figure 0.8. Boxplot representing the executive functioning performance for Organisation by level of qualification*

Lastly, results revealed that participants with a Post-graduate degree also reported statistically significantly higher scores on the Strategic Planning subscale of the EFI in when compared to participants with a Grade 12 degree ( $p = 0.0395$ ) (Figure 0.9). No significant differences were found between participants with a Post-graduate degree and participants with a Degree academic qualification.



*Figure 0.9. Boxplot representing the executive functioning performance for Strategic Planning by level of qualification*

Given these findings, it is evident that Hypotheses 5.1 – 5.3 and 5.6 – 5.8 are confirmed. However, hypotheses 5.4 – 5.5 and 5.9 – 5.10 remain unconfirmed. In the following section, two groups with and without PTSD were compared to determine whether statistically significant differences in respect of frontal lobe executive functioning on an Executive Functioning Battery exist.

### **5.3 Differences in Neuropsychological Performance**

To compare the effect of PTSD on frontal lobe executive functioning in two groups with different levels of trauma exposure, Wilcoxon Signed-rank tests and Independent sample *t*-tests were applied. Table 0.8 provides a summary of the participant’s performances on the Executive Functioning Battery.



### 5.3.1 Comparisons in terms of level of trauma exposure.

Null Hypothesis: There is no significant difference between the mean scores on each variable between the group of participants with PTSD and the group without PTSD.

Table 0.8. Executive Functioning performance between PTSD+ and PTSD– participants on the D-KEFS and EFI ( $n = 88$ )

Measure	PTSD+ ( $n = 44$ )		PTSD– ( $n = 44$ )		Z	Wilcoxon Sig.	t-Test Sig.
	Mean	SD	Mean	SD			
Number-Letter Switching	9.65	2.91	11.15	1.71	2.94	0.0222*	0.0044**
Inhibition	9.25	3.00	10.97	2.60	2.88	0.0048**	0.0050**
Inhibition/Switching	9.34	2.76	11.11	2.18	3.34	0.0015**	0.0013**
Total Achievement Score	10.27	2.47	11.40	2.00	2.37	0.0117*	0.0201*
Move Accuracy Ratio	7.54	2.07	8.9	2.47	2.94	0.0038**	0.0042**
Motivational Drive Organisation	9.59	2.95	12.45	2.36	5.01	<0.0001**	<0.0001**
Strategic Planning	8.77	3.91	13.97	3.12	6.89	<0.0001**	<0.0001**
Impulse Control	17.56	4.75	20.79	2.94	3.83	0.0005**	0.0003**
Empathy	10.34	3.51	13.11	3.28	3.82	0.0008**	0.0002**
Empathy	18.84	3.57	19.40	3.11	0.79	0.5870	0.4288

Note. Sig = Significance; \* $p < 0.05$ ; \*\* $p < 0.01$ .  $n$  = number of participants. Mdn = Median; M = Mean; SD = Standard Deviation. *Number-Letter Switching* = Trail Making Test – Condition 4: Number-Letter Switching; *Inhibition* = Colour-Word Interference Test – Condition 3: Inhibition; *Inhibition/Switching* = Colour-Word Interference Test – Condition 4: Inhibition/Switching; *Total Achievement Score* = Tower Test Total Achievement Score; *Move Accuracy Ratio* = Tower Test Move Accuracy Ratio.

The Wilcoxon Signed-rank test demonstrated statistically significantly higher performance scores for individuals without PTSD on all of the Executive Functioning Battery subscales, except for the Empathy subscale of the EFI.

Participants in the PTSD– group ( $M = 11.15$ ,  $SD = 1.71$ ) outperformed the PTSD+ group ( $M = 9.65$ ,  $SD = 2.91$ ) on the D-KEFS Trail Making Test – Condition 4: Number-Letter Switching subscale,  $Z = 2.94$ ,  $p = 0.0222$ ,  $r = 0.31$ .

Highly statistically significant results ( $p < 0.01$ ) were also obtained for both subscales of the D-KEFS Colour-Word Interference subtest. Lower scores were associated with the

PTSD+ group ( $M = 9.25$ ,  $SD = 3.00$ ) rather than with the PTSD– group ( $M = 10.97$ ,  $SD = 2.60$ ),  $Z = 2.88$ ,  $p = 0.0048$ ,  $r = 0.31$  on the Condition 3: Inhibition subscale. Statistically significantly poorer scores were also obtained on the Condition 4: Inhibition/Switching subscale by the PTSD+ group ( $M = 9.34$ ,  $SD = 2.76$ ) when compared to the scores obtained by the PTSD– group ( $M = 11.11$ ,  $SD = 2.18$ ),  $Z = 3.34$ ,  $p = 0.0015$ ,  $r = 0.36$ .

The PTSD– group also outperformed the PTSD+ group on both subscales of the D-KEFS Tower Test. The PTSD– group ( $M = 11.40$ ,  $SD = 2.00$ ) performed significantly better on the Total Achievement Score subscale than the PTSD+ group ( $M = 10.27$ ,  $SD = 2.47$ ),  $Z = 2.37$ ,  $p = 0.0117$ ,  $r = 0.25$ . The PTSD+ group ( $M = 7.54$ ,  $SD = 2.07$ ) also scored significantly worse on the Move Accuracy Ratio subscale compared to their PTSD– trauma and non-trauma exposed controls ( $M = 8.9$ ,  $SD = 2.47$ ),  $Z = 2.47$ ,  $p = 0.0038$ ,  $r = 0.26$ .

In addition to the subscales of the D-KEFS, highly statistically significant results ( $p < 0.01$ ) were also indicated for most of the EFI subscales. Lower scores were reported for the Motivational Drive subscale by participants in the PTSD+ group ( $M = 9.59$ ,  $SD = 2.97$ ) when compared to participants in the PTSD– group ( $M = 12.45$ ,  $SD = 2.37$ ),  $Z = 5.01$ ,  $p = 0.0001$ ,  $r = 0.53$ . A significant difference was also detected on the Organisation subscale of the EFI between the PTSD+ group ( $M = 8.77$ ,  $SD = 3.91$ ) and the PTSD– group ( $M = 13.97$ ,  $SD = 3.12$ ),  $Z = 6.89$ ,  $p = 0.0001$ ,  $r = 0.73$ . Similar significant differences were also reported for the Strategic Planning subscale, during which the PTSD– group ( $M = 20.79$ ,  $SD = 2.94$ ) demonstrated higher scores compared to the PTSD+ group ( $M = 17.56$ ,  $SD = 4.75$ ),  $Z = 3.83$ ,  $p = 0.0005$ ,  $r = 0.41$ . Lastly, statistically significant differences were also reported for the Impulse Control subscale, with the PTSD+ group ( $M = 1.34$ ,  $SD = 3.51$ ) reporting lower scores than their PTSD– group counterparts ( $M = 13.11$ ,  $SD = 3.28$ ),  $Z = 3.82$ ,  $p = 0.0008$ ,  $r = 0.41$ .

## 5.4 Chapter Summary

This chapter outlined the results of the various statistical analyses. According to the Kolmogorov-Smirnov test for normality, all the subscales were non-normally distributed. The Shapiro-Wilk test however found similar results for normality, except for two subscales that were not normally distributed (e.g., the Organisation subscale and Impulse Control subscale of the EFI). The implications of this for statistical analyses were that non-parametric tests were relied upon for statistical analysis, which did not require normal distribution of scores as a prerequisite.

Statistically significant differences were found in terms of gender and education regarding executive functioning performances. In addition, statistically significant differences between the two groups (PTSD+ and PTSD-) in terms of frontal lobe executive functioning performance on an Executive Functioning Battery were also found. In the following chapter attempts will be made to explain and discuss the above results.

## CHAPTER 6

### **DISCUSSION OF RESULTS, CONCLUSION, AND RECCOMENDATIONS**

In this chapter the results are discussed on the basis of the research questions and hypotheses formulated in Chapter 4. The conclusions of the study will be discussed based on the results of the theoretical and empirical investigation after which possible limitations of the study will be highlighted. In this discussion, the focus will first and foremost fall on the results pertaining to the neuropsychological performance, specifically relating to executive functioning, thereafter the results pertaining to the demographic information will be addressed. The chapter concludes with recommendations for future research and contribution of the current study.

The present study was aimed at investigating the impact of posttraumatic stress disorder on frontal lobe executive functioning. The study found that adults with PTSD performed significantly worse on executive functioning measures compared to trauma and non-trauma exposed controls without PTSD. Thus, the main hypothesis for the current research study appears to be confirmed in that there seems to be statistically significant differences between two groups of participants with different levels of trauma exposure regarding their mean scores on an Executive Functioning Battery.

This study found a correlation with earlier studies that also examined executive dysfunction among adults with PTSD (Beckham et al., 1998; Buodo et al., 2011; Flaks et al., 2014; Koso & Hansen, 2006; Leskin & White, 2007; Polak et al., 2012; Schuitevoerder et al., 2013; Twamley et al., 2009; Yehuda et al., 2006). The current study highlighted several findings that enhanced the understanding of cognitive and emotional disturbances in adults suffering from PTSD.

This study therefore confirms that adults with PTSD possess deficits in several executive functioning domains. Firstly, the abilities to pay attention and concentrate were found to be impaired, as indicated by lesser performance in Condition 4: Number-Letter Switching of the Trail Making Test. Further, reduced visual scanning and psychomotor speed difficulties emerged as the PTSD+ group performed poorer than the controls in Condition 4: Number-Letter Switching of the Trail Making Test. This is in accordance with similar findings obtained in studies done by Gilbertson et al. (2001), LaGarde et al. (2010), and Shucard et al. (2008) who examined visual sustained attention. The results therefore confirm the DSM-5 diagnostic criteria for PTSD symptoms according to which individuals with PTSD report impaired attention and difficulty to concentrate because of increased arousal (Buodo et al., 2011). In addition, this study found that PTSD+ individuals struggle to switch their attention from one stimulus to focus on a more task-relevant stimulus. This became evident in participants' performance on Condition 4: Inhibition/Switching of the Colour-Word Interference Test. A basic ability of humans is to determine the value of environmental stimuli and rapidly switch attention towards the stimuli as required (e.g., bottom-up influences on attention). On the other hand, it is just as important to establish which stimulus is unimportant or distracting to an individual's immediate goals and to distance oneself from such a stimulus with the intent to focus on the goal that is more relevant (e.g., top-down regulation of attention) (Aupperle, Melrose, et al., 2012). Evidence in literature confirms that PTSD is associated with increased activation in prefrontal networks during tasks that involves non-flexible sustained attention to stimuli, but with hypo-activation of the prefrontal networks during tasks that require inhibition or flexibility in attention (Bryant et al., 2005; Falconer et al., 2008) (e.g., as with Condition 4: Number-Letter Switching of the Trail Making Test and both the Inhibition and Inhibition/Switching conditions of the Colour-Word Interference Test). Thus, this interaction of neural response patterns in PTSD can be associated with individuals' struggle to

switch and re-focus their attention to perform at their best on cognitive tasks. The findings of the current research study therefore support literature which highlight deficits in inhibition and flexibility of attention. These findings explain at least part of the symptom profile in PTSD.

The second executive functioning domain that this study found to be adversely affected by PTSD, is inhibitory control. Impaired inhibitory control was most profound for the PTSD+ group on Condition 3: Inhibition and Condition 4: Inhibition/Switching of the Colour-Word Interference Test when compared to trauma and non-trauma controls (PTSD-). This impairment was noticeable when individuals in the PTSD+ group found it increasingly more difficult to inhibit reading the words denoting colours in order to name the dissonant ink colour in which a word was printed. These findings are supported by previous studies confirming impairment of inhibitory functioning in PTSD samples (Bressan et al., 2009; Casada & Roache, 2005; Cottencin et al., 2006; Falconer et al., 2008; Koso & Hansen, 2006; Leskin & White, 2007; Shucard et al., 2008; Swick et al., 2012; Wu et al., 2010). Therefore, the impaired performance on cognitive tasks given, can be ascribed to impaired inhibitory control, thereby influencing an individual's ability to effectively inhibit emotional memories or physiological arousal in reaction to trauma reminders (Aupperle, Melrose, et al., 2012).

Cognitive flexibility is the third executive functioning domain adversely affected by PTSD. A decline in performance on cognitive flexibility reflected in the results of Condition 4: Number-Letter Switching of the Trail Making Test and Condition 4: Inhibition/Switching of the Colour-Word Interference Test. This study demonstrated that individuals in the PTSD+ group found it increasingly more difficult to effectively shift their attentional responses toward alternative stimuli across various dimensions when compared to their PTSD- counterparts. This research thus replicated similar findings which reflect differences on trail making tasks (Beckham et al., 1998; Jenkins et al., 2000; Stein et al., 2002). These findings obtained from

the Trail Making Test may be ascribed to the large difference in the level of PTSD symptom severity between the PTSD+ and PTSD– groups tested (Leskin & White, 2007).

Performance on the subscales of the D-KEFS Tower Test demonstrated impairment in the following executive functioning domains in the PTSD+ sample, namely: Working memory, spatial planning and problem-solving, inhibition of impulsive responding, inhibition of perseverative responding, and establishing and maintaining the instructional set. The PTSD+ sample obtained significantly lower total achievement scores on this test. In addition, the PTSD+ sample also performed worse in terms of the move accuracy ratio when compared to their PTSD– counterparts. Therefore, these findings suggest that individuals suffering from PTSD are more likely to find it difficult to engage in effective inhibitory control, are more likely to make perseverative errors, struggle to plan effectively and make use of effective strategies (Sullivan et al., 2009). Therefore, impairment in working memory sustained due to the effects of chronic stress on frontal lobe networks may bring about a wide range of problems regarding the control of attention, planning, and switching between tasks (Humphreys & Samson, 2004).

Executive dysfunction was also reported for all of the subscales of the Executive Functioning Index (EFI) except for Empathy. Thus, it can be concluded that PTSD+ individuals are more inclined to display lower levels of motivational drive (energy) and enthusiasm to engage in novel tasks. Lezak et al. (2012) define the intricate process of deciding what one desires or needs together with the conceptualisation of future realisation of that desire or need as volition – a key attribute of executive function. According to Lezak et al. (2012), an important precondition for volitional behaviour is motivation, which includes the ability to initiate activity. This implies that individuals in the PTSD+ group are less likely to take on responsibilities which require the appreciation of future abstract goals and may refrain from engaging in new or unfamiliar activities independently.

Based on the results of the Organisation subscale, PTSD+ individuals are less likely to initiate, maintain, switch and/or stop a series of complex behaviour in an organised and integrate manner. The functions involved in programming are critical for the successful execution of non-routine tasks, but are not required during instances where the action sequence is routine (Shallice, 1982). Therefore, familiar, overlearned, and routine tasks, as well as automatic behaviours, may be significantly less vulnerable to the effects of stress.

The results obtained by the Strategic Planning subscale of the EFI, indicated that a PTSD+ individual's ability to identify and organise steps and elements necessary to carry out an intention or accomplish a goal that requires planning, is compromised. Such an individual will most likely find it difficult to formulate alternatives, weigh and make choices, and consider the subsequent and hierarchical ideas essential for the development of a conceptual framework that will serve as a course of action to carry out a plan. Such an individual, will struggle to set and maintain goals.

The results obtained from the Impulse Control subscale, which measures control over inhibitions, clearly demonstrate that PTSD+ individuals may find it difficult to contain their impulses, habits, or actions, or any other stimuli in their internal or external environment that may distract them. The lack of impulse control may lead to inappropriate behaviour such as risk taking, substance abuse, excessive spending, as well as the difficulty to control one's temper (Garner, 2009).

Concerning gender, Table 0.4 to Table 0.6 indicate that gender differences with respect to dimensions of frontal lobe executive functioning are present. In the present study, gender differences in respect of two aspects of frontal lobe executive functioning were observed, namely Empathy and Organisation.



Females in the entire sample obtained a higher score on Empathy in comparison to their male counterparts. Higher scores for Empathy is indicative of an awareness and experience of pro-social concern for other people. Thus, it can be argued that females included in this study were significantly more concerned about the well-being of others than what their male counterparts appeared to be.

In addition, PTSD+ males scored higher on Organisation when compared to females with PTSD. In light of this, it can be argued that males with PTSD may tend to outperform females with PTSD when it comes to the ability to engage with organised, purposeful behaviour during tasks that requires the execution of multiple tasks (multitasking), performing consecutive tasks in order to achieve a goal, and retaining information in mind in order to make a decision. These males are also likely to be less distractible from task irrelevant information. To conclude gender differences, PTSD+ females obtained higher scores on Empathy than their male counterparts. Therefore, this study suggests that females with PTSD are less likely to take an aggressive social stance than males with PTSD. Existing literature demonstrates that females tend to, from a young age, be superior to males in social skills, demonstrating greater empathy, sensitivity to facial expressions, and a higher developed theory of mind (Baron-Cohen, 2002; Dunn et al., 1991).

Concerning comparisons in terms of education, Table 0.7 indicates that differences related to the level of academic qualification, regarding the dimensions of frontal lobe executive functioning, does exist. In the current study, significant differences were observed for six subscales of the Executive Functioning Battery, namely: (1) Motivational Drive; (2) Organisation; (3) Strategic Planning; (4) D-KEFS Trail Making Test – Condition 4: Number-Letter Switching; (5) D-KEFS Colour-Word Interference Test – Condition 3: Inhibition; and (6) D-KEFS Colour-Word Interference Test – Condition 4: Inhibition/Switching. Higher levels

of education were associated with better performance on measures of attention, cognitive flexibility, inhibitory control, organisation, planning, visual scanning, and motor speed.

Van Hooren et al. (2007) provides similar findings by reporting that participants with a middle or high level of education obtained better results on most cognitive tests in comparison with participants with a lower level of education. Tun and Lachman (2008) also agree with Van Hooren et al. (2007) who conducted a population-based study of older adults that found effects of education on a set-shifting task. However, Reimers and Maylor (2005) who investigated a younger self-selected sample did not show effects of education on task switching, while Ylikoski et al., 1998) could not find any educational effects on a Stroop switching task. However, Tun and Lachman (2008) and Wecker et al. (2005), as well as Plumet et al. (2005) found effects of education on other tests of executive function (Wechsler Abbreviated Scale of Intelligence (WASI) and the Delis–Kaplan Executive Function System (D-KEFS)), suggesting that education might be associated with a general benefit in organising and scheduling complex responses.

In spite of the results confirming the link between PTSD and executive dysfunction, several limitations to the current study need to be pointed out. Firstly, differences between the groups in terms of type of trauma exposure and severity of symptoms were not investigated and may have had an impact on executive functioning performance. Studies are available, linking for example, attentional deficits to PTSD symptom severity (Burriss et al., 2008). Polak et al. (2012) also found that executive functioning performance was differently affected by the type of trauma exposure. However, Koso, Sarač-Hadzihalilovic, and Hansen (2012) found that severity of PTSD symptoms is the main factor instead of type of trauma. It is suggested that future research attempts to clarify the link between symptom severity and executive function impairment in PTSD samples.

Repeated trauma exposure that could contribute to impaired executive functioning is another contributing factor that was not investigated (Polak et al., 2012). This study was not designed to investigate complex symptom presentations such as dissociative symptoms, aggressive or social avoidant behaviours. This study assessed individuals who differed in the duration since trauma exposure occurred and a subsequent diagnosis with PTSD by a professional clinician was made, this study lacked investigation into the effects of temporal proximity of trauma exposure on executive functioning performance (Schoeman et al., 2009).

On a methodological ground, it must be mentioned that the measuring instruments employed were designed and developed outside of South Africa. Although all the participants included in this study were able to comprehend and respond to the test instructions, it must be noted that the instruments were not developed with keeping in mind the diverse South African context, especially pertaining to language and cultural differences.

The final limitation that could impact the results of this study is the lack of information about participants' pre-traumatic cognitive functioning. It was therefore not possible to correlate neuropsychological impairments with events that occurred prior to the trauma. It is possible that these impairments may be partially responsible for the inability to cope with traumatic stress (e.g., response inhibition and attention regulation). According to Aupperle, Melrose, et al. (2012), impaired pre-trauma cognitive functioning, especially in domains of executive function, attention, and memory could serve as a potential risk factor for the development of PTSD. Therefore, it is imperative that any conclusions based on impairments associated with PTSD should carefully consider alternative explanations such as the potential effect of confounding factors.

With the lifetime traumatic event prevalence rate for South Africa at 73.8% and the conditional prevalence of PTSD after trauma exposure at 3.5% (Atwoli, 2013) and the significant individual distress, including the societal burden associated with PTSD, this study provides important findings especially because executive functioning plays a critical role in all areas of daily functioning stretching beyond occupational functioning. It is therefore imperative that cognitive rehabilitation programs should be developed to assist individuals to maximise independence in daily life. Executive functioning impacts a number of varying cognitive processes and individuals who receive neuropsychological services may be more likely to exhibit executive dysfunction than any other cognitive impairment. Deficits in executive functioning interrupt a person's ability to adequately employ intact areas of functioning, and impair the efficient self-management of other areas of dysfunction, preventing the use of compensatory strategies. Therefore, it is critical that assessment of an individual's executive functioning form part of a comprehensive neurorehabilitation plan. Insight gained from cognitive and neuroscientific research may play an integral role for guiding the development of innovative, more effective, future treatments for PTSD.

## **6.1 The Contribution of the Current Study**

As far as this researcher is aware, no South African study has thus far investigated the unique relationship between frontal lobe executive functions and posttraumatic stress disorder in an adult population. In this regard, the study makes an important contribution to the field of cognitive neuropsychology in South Africa.

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