Working Memory, Inhibition, and Impulsivity: The Promise of Working Memory Training for the Burden of Depression.

Matthew J. T. L. Snelling

ACSENT Laboratory

Department of Psychology

University of Cape Town

Supervisor: Dr Susan Malcolm-Smith

Co-Supervisor: Dr Samantha Brooks

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Abstract

Depression is regarded as the single most burdensome disease in terms of years lived with disability. Currently, psychotherapy is only effective in milder cases of depression, due to cognitive impairments present in moderate and severe depression. Working memory training has been found to reduce cognitive impairments. The purpose of the present research was to unify literature related to working memory processes, inhibition, impulsivity and depression in order to determine whether the relationship between these would inform a working memory training intervention in depression. Forty-two undergraduate psychology students were recruited from the University of Cape Town. Participants completed self-report measures related to inhibition, impulsivity and depression. Participants then completed performance measures related to working memory. Working memory was measured by two types of error: misses and false positives. The results of hierarchical multiple regression analyses suggested that false positives negatively predicted inhibition. Further, these analyses suggested that inhibition negatively predicted depression, and impulsivity positively predicted depression. A mediation analysis was conducted. The result of the mediation analysis suggested that impulsivity mediated the relationship between inhibition and depression. The limitations and directions for future research are discussed. These were primarily related to time and resource limitations. The practical implications are discussed. It is suggested that there is sufficient evidence to recommend the use of working memory training in cases of moderate and severe depression.

Keywords: *working memory, impulsivity, inhibition, cognitive training, neuroplasticity*

Table of Contents

Depression is one of the most commonly diagnosed mental disorders. Half of all lifetime cases begin by the age of 14 – three quarters begin by the age of 24. Fifty percent of those who recover from a depressive episode relapse within two years, and subsequent relapses increase in frequency with every subsequent episode. The World Health Organization has ranked depression as the single most burdensome disease in terms of years lived with disability. The financial costs of depression are immense, amounting to hundreds of billions of dollars in medical costs, loss of life, absenteeism and disability (Richards, 2011). Currently, psychotherapy is only effective in milder cases of depression due to cognitive impairments present in moderate and severe depression (Mc Dermott & Ebmeier, 2009). It is suggested that if cognitive impairment could be reduced, psychotherapies like Cognitive Behavioural Therapy (CBT), could be made effective for moderate and severe cases of depression. This would likely reduce the vast burden of the disease presented by the disorder worldwide (Gohier et al., 2009; Gruber, Zilles, Kennel, Gruber, & Falkai, 2011). This research hoped to explore how this could be possible. In order to do this, it was first vital to frame depression in relation to working memory, inhibition, impulsivity, neuroplasticity and cognitive training.

Perspectives on depression

There are various theoretical perspectives on depression. Some of the major perspectives form part of psychodynamic theories, humanistic theories, learning theories and cognitive theories. Broadly speaking, all of these perspectives add some insight into the internal world of depression. Psychodynamic perspectives draw attention to the role of loss and self-focus (Kendler, Hettema, Butera, Gardner, & Prescott 2003; Muraven, 2005). Humanistic theories illuminate the role of self-worth and personal identity (Sheldon & Kasser, 2001). Learning theories introduce the importance of reward and reinforcement (McHugh, Smits, & Otto, 2009). Cognitive theories draw attention to the way that people think about themselves and the world around them (Beck & Alford, 2009). A particularly popular and foundational form of cognitive theory is pertinent to the current research, as it was developed specifically to treat depression. This form of cognitive theory proposes that a cognitive triad of negative beliefs concerning the self, the environment and the future, increase the risk of becoming depressed. Changing this triad of beliefs is a large part of cognitive therapies (Beck, 2005). Evidence suggests that this approach is effective (Butler, Chapman, Forman, & Beck, 2006). Further, many national treatment guidelines support its use (National Collaborating Centre for Mental Health UK, 2010). However, psychotherapies

like CBT are not always effective. Neuroscientific views commonly seek to expand the efficacy of psychotherapies, like CBT, by determining the neural correlates found in people with depression. This is done in order to ground theoretical inferences in neuroscientific observation (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). The dual process view of depression is one of these observations.

The dual process view of depression. The dual process view of depression suggests that an evolutionarily old, highly reactive, associative and impulsive, 'reflexive system' competes with an evolutionarily recent deliberative and goal directed, 'executive system' (Carver, Johnson, & Joormann, 2013). The reflexive system is suggested to develop early in life and is suggested to be ventral, subcortical, limbic and paralimbic. The executive system, which develops later in life, is suggested to be dorsal, striatal, cortical and frontal. The reflexive system is thought to become hyperactive in depression, and the executive system hypoactive (Carver et al., 2013). Substantial evidence supports this (Bickel, Yi, Landes, Hill, & Baxter, 2011; Romer et al., 2011; Rose & Ebmeier, 2006).

The dual process view suggests that a working memory deficit contributes to an impulsive and disinhibited reaction to emotions, resulting in a chronic negative affective state (Carver et al., 2013; Gohier et al., 2009; Hinson, Jameson, & Whitney, 2003). Further evidence supports this, suggesting that there is indeed a working memory deficit in depression (Mc Dermott & Ebmeier, 2009). In order to understand how this imbalance functions in depression, it is useful to understand the executive system within the context of working memory.

Working Memory

One broadly accepted theory of working memory suggests that working memory is a flexible, limited capacity system, used to process and store information in the service of cognition (Morrison & Chein, 2011). It is suggested that working memory is composed of four subsystems – the phonologic loop, the visuospatial sketchpad, the episodic buffer, and the central executive. The phonologic loop is suggested to consist of the phonologic store and the articulatory rehearsal system. The phonologic store holds a cognitive representation of verbal or auditory information. The articulatory rehearsal system facilitates semantic processing by maintaining information in temporary storage using the repetition of verbal or auditory information. The visuospatial sketchpad is suggested to consist of the visual cache and the inner scribe. The visual cache stores mental imagery and the inner scribe allows its

manipulation and transformation (Baddeley, Cocchini, Della Sala, Logie, & Spinnler, 1999). The episodic buffer is suggested to integrate information from long term memory and both the phonologic loop and visuospatial sketchpad. The episodic buffer is also suggested to be used for the chunking of information to allow more of it to be held on-line. The central executive directs the overall action of the episodic buffer and the other working memory subsystems (Baddeley, 2002; Baddeley et al., 1999).

The Central Executive and Cognitive Inhibition

In a healthy individual, it is suggested that the central executive uses the working memory subsystems to serve established task goals. Task goals are maintained and updated through the monitoring of performance, the correction of errors and the retrieval of information from long-term memory. This is facilitated by the management of retrieval plans and the encoding of new information to long-term memory via the phonologic loop and visuospatial sketchpad (Baddeley, 2002; Baddeley et al., 1999).

The auditory and verbal processing areas of the left hemisphere and visuospatial processing areas of the right hemisphere, are suggested to facilitate the phonologic loop and visuospatial sketchpad respectively. The ventrolateral prefrontal cortex facilitates the selection, retrieval, comparison and judgment of task-relevant information. The mid-dorsolateral prefrontal cortex facilitates maintenance, monitoring and manipulation of task-relevant information (Baddeley, 2002). The orbitofrontal cortex facilitates the integration of emotionally salient cues into this processing system and the anterior cingulate cortex facilitates conflict resolution (Rogers et al., 2004). These areas allow the central executive to plan, control, and manipulate the sequence of actions to be performed by allocating and dividing attention and controlling how information is integrated within the episodic buffer (Hinson et al., 2003). To ensure that this is achieved, many of these areas are also involved in the process of cognitive inhibition. Cognitive inhibition (also referred to in this paper as inhibition) is an active working memory process of the central executive that filters irrelevant information, both internal and external. Inhibition is suggested to contain 3 components – access, restraint and deletion. Inhibition is suggested to restrict the ‘access’ of information that is not relevant to the task at hand. It is also suggested to ‘restrain’ the incorrect retrieval of irrelevant information. Finally, cognitive inhibition is suggested to ‘delete’ information as it becomes irrelevant (Gohier et al., 2009).

Cognitive Inhibition and Cognitive Impulsivity

However, it is purported that an imbalance between ventral and dorsal regions renders the central executive unable to protect the content of the episodic buffer in those with depression (Hinson et al., 2003). Inferior frontal regions, such as the orbitofrontal cortex, appear to be hyperactive in cases of depression (Bickel et al., 2011; Carver et al., 2013). These inferior frontal regions are implicated in the detection of salient internal and external cues relevant to the interruption or alteration of responses (Rogers et al., 2004). Further, these inferior frontal regions are implicated in the semantic coding process of the articulatory rehearsal system (Baddeley, 2002). Hyperactivity of this region is suggested to result in a state of amplified sensitivity to internal and external stimuli, hyper vigilance to error, and repetition of semantic coding (Gohier et al., 2009). The working memory system is suggested to be too overwhelmed by repetitive and irrelevant cues to maintain established task goals. It instead defers to actions that successfully address the cue that happens to be flooding the episodic buffer at that given moment – which is suggested to produce frequent rumination and low cognitive flexibility (Bickel et al., 2011; Marazziti, Consoli, Picchetti, Carlini, & Faravelli, 2010; Mc Dermott & Ebmeier, 2009; Romer et al., 2011). This is suggested to produce cognitive impulsivity (also referred to in this paper as impulsivity).

Research supports this, suggesting that people with depression experience deficits in all 3 aspects of cognitive inhibition, especially the inability to restrict the access of irrelevant information (Gohier et al., 2009). In other words, people with depression are suggested to use a more impulsive and automatic style of reasoning. However, just as a person is not fixed in the way they think, the brain is not fixed. The brain's neuroplastic structure reflects this subjective flexibility in objective form.

Neuroplasticity

Neuroplasticity refers to the brain's ability to adapt its structure and organisation by changing its connections (Harasym, 2008). The main purpose of neuroplastic change is suggested to be to allow an organism to alter its behaviour in keeping with the demands of the environment (Lourenco & Casey, 2013). The supply-demand model suggests that brain structure adapts to experiences by making slow and continuous adaptations. Further, the model suggests that adaptations can either increase or decrease brain volume. Evidence suggests that brain volume increases when environmental demand is greater than neurological supply (Draganski et al., 2004; Lövdén, Wenger, Martensson, Lindenberger, &

Backman, 2013). Conversely, further research suggests that brain volume decreases when environmental demand is less than neurological supply (Langer, Hanggi, Muller, Simmen, & Jancke, 2012). The expansion-partial-renormalisation model, expands upon the supply-demand model by suggesting that increases in brain volume occur nearly immediately after environmental demand exceeds neurological supply, followed by a partial renormalisation of the overall volume. Only useful new neural tissue is spared from this process of renormalisation (Reed et al., 2011).

Evidence supports these two models, suggesting that the increase in overall volume reflects a combination of dendritic growth, synaptic sprouting, the expansion of capillary networks, the presence of new glia, and in select cases, the introduction of new neurons (Lövdén et al., 2013). This research forms an evidentiary foundation for cognitive training.

Cognitive Training and Depression

Cognitive training and psychotherapy attempt to capitalise on neuroplasticity by using specific cognitive exercises, or therapeutic techniques, to change the balance of neurological activity and connections. Two recent studies have successfully demonstrated that cognitive training can result in significant increases in both white matter and grey matter. Additionally, these studies reflect wide age ranges and demonstrate neuroplastic change in a variety of brain areas. Both studies suggest that cognitive training could be used to assist in the treatment of a variety of psychological disorders (Chapman et al., 2013; Kühn, Gleich, Lorenz, Lindenberger, & Gallinat, 2014). Pertinent to the current research, the cognitive training of working memory is suggested to produce increased activation in the executive system and improve control over inhibitory processes (Bickel et al., 2011; Morrison & Chein, 2011; Olesen, Westerberg, & Klingberg, 2004). The current research suggests that a poorly functioning executive system may be partially to blame for the immense global burden of depression (Richards, 2011). Further, this is supported by evidence that suggests that CBT is most effective when it is used to treat relatively mild cases of depression – cases with little working memory deficit. Moderate and severe cases of depression are suggested to be beyond the domain of treatment because working memory deficits largely prevent psychotherapy from being effective (Gruber et al., 2011; Mc Dermott & Ebmeier, 2009).

Working memory training prior to, or in conjunction with, psychotherapies like CBT may present a solution. If working memory training is successful in improving the control of the central executive, interference that is typical of depression may be reduced – as has been

shown in other working memory deficits (Bickel et al., 2011; Morrison & Chein, 2011; Olesen et al., 2004). This may facilitate the delivery of CBT to cases of depression that are currently excluded from treatment due to cognitive deficits (Marazziti et al., 2010; Mc Dermott & Ebmeier, 2009). This would assist in reducing the global burden of depression.

Limitations of Previous Research

The research presented above, although promising, has largely been conducted separately. This means that the argument made for the relationship between these factors is still just an integration of theory. No research has yet attempted to unite this diverse literature by investigating the relationship between working memory performance, inhibition, impulsivity, and depression.

Aims and Hypotheses

To address this separation, this research aimed to clarify the relationship between working memory performance, inhibition, impulsivity and depression. This research argued that if working memory, inhibition and impulsivity were indeed related, performance on a working memory task would be capable of predicting inhibition and impulsivity. Further, if impulsivity and inhibition were related to depression, then theory suggests that impulsivity should be positively related to depression, and inhibition should be negatively related to depression. Finally, these opposing relationships should be capable of predicting the severity of depression. This link is necessary, both to establish the importance of impulsivity and inhibition in depression, and to inform a future implementation of working memory training in a clinical population with Major Depressive Disorder.

To this end, this research proposed a number of hypotheses:

H_1: Working memory performance errors will negatively predict inhibition.

H_2: Working memory performance errors will positively predict impulsivity.

H_3: Depression severity will be negatively related to inhibition.

H_4: Depression severity will be positively related to impulsivity.

H_5: Inhibition and impulsivity will predict depression severity.

Method

Design and Setting

This research used a quantitative correlational design consisting of both self-report and performance measures. Participants were required to fill out a number of self-report measures covering depression, impulsivity, and inhibition deemed vital for later analysis (See *Materials and Apparatus* and *Procedure*). Participants were then required to complete a performance measure containing three levels of the *n*-back task (*n*-back levels 1, 2 and 3).

Participants

Sample Characteristics. Participants were 42 South African undergraduate psychology students from the University of Cape Town. All participants were between the ages of 18 and 23 (M = 20.29, SD = 1.04). Ninety percent of the participants were female and 10% were male. Forty-five percent of the participants were 'white', 31% were 'black', 19% were 'coloured' and 5% were 'indian' (See Table 1).

Sampling Procedure. A screening survey and a demographic questionnaire were made available for a limited period of time, during which, participants were chosen using purposive sampling based on their level of depression. This research required that a range of depression scores be included in order to run the required regression analyses. Forty-seven participants were recruited from undergraduate psychology courses at the University of Cape Town using the Student Research Participation Program (SRPP). Of these, 5 participants were exchange students visiting from other universities, and although they were permitted to take part in the research session, their data were excluded. After these participants' data were excluded, 42 participants remained. The research attempted to create equal numbers of each gender, race, and age, but severity of depression took priority when selecting participants for inclusion. It was not possible to produce equality in these groups, so the research included race, gender and age in analyses to ensure that they were controlled for.

Inclusion and Exclusion criteria. In order to maximise generalisability, this research only used data from participants who were South African. Participants were required to have no comorbid psychological disorders. These would confound any relationship to the disorder under study. Participants could not be taking anti-depressant medication or anti-anxiety medication. These medications produce cognitive changes that would have interfered with the

influence of the working memory tasks (Amado-Boccara, Gougoulis, Poirier Littre, Galinowski, & Loo, 1995; Hindmarch, 1998; Levkovitz, Alpert, Brintz, Mischoulon, & Papakostas, 2012; Silver, Hughes, Bornstein, & Beversdorf, 2004). Participants were required to have no primary sensory deficits. If participants had a primary sensory deficit, they were required to use a corrective aid during the research session.

Table 1
Demographic Characteristics of participants (N=42)

Socio-demographic		Number	Percentage
Race	White	19	45%
	Black	13	31%
	Coloured	8	19%
	Indian	2	5%
Gender	Female	38	90%
	Male	4	10%
Age	18	1	2%
	19	7	17%
	20	20	48%
	21	8	19%
	22	5	12%
	23	1	2%
Year of Study	1st	3	7%
	2nd	16	38%
	3rd	19	45%
	4th	4	10%

Materials and Apparatus

Demographics. The demographic questionnaire contained questions that asked for participants' name, age, gender, ethnicity, year of study, nationality, medical history, psychiatric history, drug history, current medications, and whether the participants used corrective aids such as corrective lenses or hearing aids (See Appendix B).

Depression Screening Measure. The Hospital Anxiety and Depression Scale (HADS) is a widely used 14 item self-report measure designed to identify cases of depression and anxiety via their cognitive features, quickly and efficiently (Kendel et al., 2010; Zigmond & Snaith, 1983). The HADS can be divided into two subscales of equal length (HADS-A and HADS-D), each scored out of 21. The subscales are suggested to be partially correlated due to common environmental factors responsible for both disorders. Despite this, the HADS shows good internal consistency across multiple languages and implementations, and good validity and reliability in a number of countries, including South Africa (Berard, Boermeester, & Viljoen, 1998; Bjelland, Dahl, Haug, & Neckelmann, 2002; Wouters, le Roux Booysen, Ponnet, & Van Loon, 2012). Most importantly for the present study, the HADS is suggested to show good specificity and sensitivity to potential cases of anxiety and depression at a cut off score of 8 for both – detecting between 70% and 90% of diagnosable cases in the general population (Bjelland et al., 2002). However, it is cautioned that this measure requires a high degree of literacy. In the present study this was not a significant problem, as all participants were university students and had undergone university entrance literacy tests (Golden, Conroy, & O'Dwyer, 2007). Further, it is suggested that the HADS is a more efficient measure than longer measures like the BDI at screening for depression. This informed its use during the screening process (de Oliveira et al., 2014) (See Appendix C).

Depression Severity Measure. The Beck Depression Inventory (BDI-II) is a 21 item self-report measure designed to detect depression and its severity (Beck, Steer, Ball, & Ranieri, 1996). The BDI-II is suggested to be a more robust measure than the HADS due to its comprehensive nature and its ability to detect the severity of depression (de Oliveira et al., 2014). The BDI-II provides a score from 0 to 63 and contains cut-off scores that are capable of representing mild (14-19), moderate (20-28), and severe depression (29-63). The BDI-II is suggested to show good reliability and validity and is suggested to show good specificity and selectivity in many countries, including South Africa (Aalto, Elovainio, Kivimäki, Uutela, & Pirkola, 2012; Dolle et al., 2012; Ward, Flisher, Zissis, Muller, & Lombard, 2003). Ability to detect the severity of depression was vital in the present study, so a measure of this nature could not be overlooked (See Appendix D).

Cognitive Impulsivity and Cognitive Inhibition Measures. The Barratt Impulsivity Scale (BIS-11) is a 30-item self-report measure designed to detect the personality construct of impulsiveness and assess its severity (Barratt & Patton, 1983; Stanford et al., 2009). The BIS-11 is suggested to be the most widely used measure for the detection of impulsivity, and has

good validity and reliability across a number of contexts, including South Africa. The BIS-11 is suggested to show little relationship with behavioural indices of impulsivity, but shows a strong relationship with trait impulsivity (Kaliski & Zabow, 1995; Stanford et al., 2009). For this reason, this measure was important for the present study, as it attempted to detect impulsivity that was enduring, rather than transient, similar to the enduring nature of depression and the type of trait-based impulsivity related to it (Carver et al., 2013) (See Appendix E).

The Self-Regulation Questionnaire (SRQ) is a 63-item self-report measure designed to detect an impulse control factor and goal setting factor that serve a global self-regulation or inhibition factor (Brown, Miller, & Lawendowski, 1999). The SRQ is a widely used measure for detecting self-regulatory behaviour and it is suggested to show good validity and reliability (Carey, Neal, & Collins, 2004). Additionally, the SRQ is currently being used in a number of ongoing studies in the Department of Psychiatry and Mental Health at the University of Cape Town. Importantly for the present study, the SRQ, and the factors that underlie it, have been demonstrated to be inversely related to impulsivity and working memory performance (Neal & Carey, 2005; Romer et al., 2011). For these reasons, this measure was used in conjunction with the BIS-11 to provide a comprehensive indication of impulsivity and inhibition (See Appendix F).

Working Memory Task. The *n*-back task is a popular working memory task that requires participants to match the appearance of sequential letters. The task taker is required to match the current letter with a letter a certain '*n*' back from the present one. The *n*-back task is regarded as a relatively pure way of measuring working memory due to its limited complexity and its variable difficulty (Jaeggi, Buschkuehl, Perrig, & Meier, 2010).

Further, the *n*-back task is suggested to be a 'core' training task. This means that the *n*-back task taps into a domain general process that underlies performance on a variety of tasks, rather than just the task at hand. This task has been used successfully in the training of working memory, and has resulted in a general improvement in tasks other than the task that was trained (Morrison & Chein, 2011). The *n*-back task is suggested to be valid at detecting inter-individual differences in higher cognitive functions, like working memory, and is suggested to show moderate reliability across all four levels (Hockey & Geffen, 2004; Jaeggi et al., 2010).

Further, the *n*-back task can be divided into 'correct responses', 'misses', and 'false

positives'. Correct responses indicate that a correct match has been made between two letters; a miss indicates that a correct match between two letters has been overlooked; and a false positive indicates that a correct match between two letters has been misidentified. In the present research, correct responses were excluded, as they could be artificially inflated by random pressing of the spacebar. Misses and false positives, however, were included as they produced two forms of performance-based working memory error potentially related to inhibition and impulsivity. Misses were deemed a performance-based representation of failure to maintain the string of letters in working memory. False positives were deemed to represent a performance-based representation of the interjection of letters into working memory that were not seen (Gohier et al., 2009). For these reasons, the *n*-back task was considered to be ideal for conducting a comprehensive assessment of working memory performance in the present research.

Procedure

This study followed the ethical guidelines for conducting research on human participants, as set out by the University of Cape Town. Data collection only began once ethical approval was granted by the Psychology Department Research Ethics Committee.

Undergraduate psychology students were recruited via the Student Research Participation Programme (SRPP). Students were rewarded with course credits (SRPP Points) that reflected the time taken to participate in the research session. Advertising via the SRPP website consisted of an electronic notice. The electronic notice contained a link to a demographic questionnaire and an electronic version of the HADS. Participants were contacted no later than 48 hours after they had completed the survey in order to arrange a participation date for the research session. A variety of scorers on the HADS for the previous week were chosen for participation during the following week. The online forms took no longer than 30 minutes to complete. Each research session took no longer than 2 hours to complete. Participants received 4 SRPP points for completing the research session.

The research session began with the signing of a consent form (Appendix A). Participants were notified both verbally, and in writing in the consent form, that they could withdraw their participation at any time. Further, participants were reminded that all data collected would be assigned to a participant number and be kept confidential and anonymous.

Participants then completed a measure of depression severity using the BDI-II; a

measure of impulsivity using the BIS-11; and a measure of inhibition using the SRQ. This took approximately 15 minutes to complete.

After a 5 minute break, participants were seated at a laptop and were instructed that they were to begin the first level of a memory game – the *n*-back task, 1-back. Participants were informed that this was a training level designed to allow them to get used to the procedure of the memory game. Participants were instructed that for this memory game, they should press the spacebar when a letter appeared that was the same as the letter before it. In the present research, each letter of the *n*-back task remained for 1.5 seconds. A fixation circle then appeared between each letter for 1.5 seconds. This repeated for 5 minutes, after which time participants took a 1 minute break indicated by a countdown clock. This 6 minute session repeated 5 times in a single *n*-back level. A single level took 30 minutes to complete. To assist participants in learning the *n*-back task, the researcher sat next to the participant for no more than the first 5 minutes of the task in order to confirm that they were responding correctly. During this time, participants were encouraged to ask questions. The researcher then left the participant to complete the remainder of the first level alone. Between *n*-back levels, participants took a 5 minute break.

After the break was complete, participants were instructed that the memory game would change. Participants were informed that this next level of the memory game would teach them the technique needed to complete the final level. Participants then completed the 2-back task. Participants were instructed to press the spacebar every time they saw a letter that was the same as 2 letters before it. To assist the participants in learning this level of the *n*-back task, the researcher sat next to the participant for no more than the first 5 minutes of the task in order to confirm that they were responding correctly. During this time, participants were encouraged to ask questions. The researcher then left the participant to complete the remainder of the second level alone. Participants were then asked to take a 5 minute break.

After the break was complete, participants were then instructed that the task would change one last time in order to test their memory. Participants then completed the 3-back Task. Participants were instructed to press the spacebar every time they saw a letter that was the same as 3 letters before it. Participants were asked to confirm that they understood the concept of the 3-back Task, and were required to explain the task to the researcher before beginning. Participants received no additional assistance with the 3-back Task. This was done so that the results of the 3-back task could be attributed to the performance of the participant,

rather than to external factors related to the interference of the researcher.

After the final task was completed, participants were thanked for their participation, and were debriefed. An important part of the debriefing was the presentation of a pamphlet providing information on depression and providing them with contact details for the UCT Student Wellness Centre, should they find themselves in need of it.

Data Analysis

Data were analysed using the IBM Statistical Package for the Social Sciences (SPSS) version 22. Significance was marked at $p < .05$. Analyses were done in order to link working memory performance to two measures of executive control, in this case represented as impulsivity and inhibition. Impulsivity and inhibition were, in-turn, linked to a single measure of depression. This would support the relationship between working memory training and depression via its relationship with inhibition and impulsivity (Gohier et al., 2009; Gruber et al., 2011; Marazziti et al., 2010).

Assumptions. In order to address the assumptions of the following correlation, multiple regression and mediation analyses, p-p plots, histograms and scatterplots were run to assess the distribution of all continuous variables. These indicated sufficiently normal distributions and no heteroscedasticity (See Appendices G, H and I). No significant outliers were detected in the data for the analyses. Consideration of the Tolerance statistics and Variance Inflation Factors (VIF) suggested that there was no multi-collinearity in any of the models. Race, gender, and age were entered in regressions with all variables of interest, and then removed due to the limitations of the sample size. In all cases, demographic variables were found to be insignificant (See Appendix J). Investigation of the Durbin-Watson statistic suggested autocorrelation may be a concern. However, low scores on this statistic were relatively acceptable due to the small sample size and the number of predictors (Durbin & Watson, 1951).

Hypothesis 1 and 2. A pair of hierarchical multiple regressions were used to investigate the relationships between working memory performance and impulsivity; and working memory performance and inhibition respectively. The first step of the regressions consisted of false positives. False positives were inserted first because they were thought to be most closely related to impulsivity (Gohier et al., 2009). The second step of the regressions consisted of misses. The dependent variable in the first regression was

impulsivity. The dependent variable in the second regression was inhibition. Pearson's correlation coefficients were used to investigate the relationship between misses, false positives, impulsivity and inhibition. Post-hoc power analyses were conducted using G*Power 3.1 and the target power was set at .80.

Hypothesis 3 and 4. Pearson's correlation coefficients were used to examine the relationship between impulsivity, inhibition and depression.

Hypothesis 5. A hierarchical multiple regression was used to investigate the relationship between impulsivity, inhibition and depression. The first step of the regression consisted of impulsivity. The second step of the regression consisted of inhibition. The dependent variable in this regression was depression.

Mediation Analysis. The Preacher and Hayes mediation analysis was conducted, placing impulsivity as a mediator between inhibition and depression. Due to the small sample size, one additional multiple regression was also added to support the mediation analysis. The regression analysis supported this by attempting to better account for the role of inhibition by placing depression and self-regulation as predictors of impulsivity. Bootstrapping was set at 5000 resamples (Preacher & Hayes, 2008). Post-hoc power analyses were conducted using G*Power 3.1 and the target power was set at .80.

Results

Descriptive statistics are presented in Table 2. These include the means (*M*) and standard deviations (*SD*) for the variables presented in the analyses. These include depression, impulsivity, inhibition, misses and false positives.

Table 2
Descriptive Statistics for Variables of Interest

	N	*M*	*SD*
Depression	42	8.74	6.84
Impulsivity	42	60.52	9.48
Inhibition	42	226.05	20.93
Misses	42	24.5	13.73
False Positives	42	24.36	9.58

Working memory errors as predictors of impulsivity and inhibition. During step 1 of both hierarchical multiple regressions presented here, false positives were entered. At

this first step, false positives were not a significant predictor of impulsivity. However, false positives were a significant predictor of inhibition (R^2 = .10, F [1,40] = 4.28, p < .05). Post-hoc power analyses using G*Power 3.1 revealed that the power was only .54 for this effect size. Step 2 for the first and second regression included misses as a predictor. Impulsivity and inhibition were entered as dependent variables. Together, misses and false positives were not significant predictors of impulsivity or inhibition. However, Pearson's correlation coefficients suggested that false positives (M = 24.36; SD = 9.58) showed a weak positive correlation with depression (M = 8.74; SD = 6.84) (r = .31, p < .05), and a weak negative correlation with inhibition (M = 226.05; SD = 20.93) (r = .31, p < .05). Table 3 presents a summary of the results of these 2 hierarchical multiple regression analyses.

Table 3
Hierarchical Multiple Regression Analysis Predicting Impulsivity and Inhibition

	Impulsivity		Inhibition	
Predictors	ΔR^2	β	ΔR^2	β
Step 1	.07		.10*	
False Positives		.26		-.31*
Step 2	.07		.10	
False Positives		.26		-.30
Misses		.06		-.09
	R = .27		R = .32	
	Adj.R^2 = .02		Adj.R^2 = .10	

*p < .05; **p < .01. ***p < .001

The relationship between inhibition, impulsivity and depression. Depression (M = 8.74; SD = 6.84) showed a strong positive correlation with impulsivity (M = 60.52; SD = 9.48) (r = .63, p < .001), and a strong negative correlation with inhibition (M = 226.05; SD = 20.93) (r = .61, p < .001). Additionally, impulsivity showed a strong negative correlation with inhibition (r = .78, p < .001). Table 4 presents a summary of the results of the correlation analyses.

Table 4
Correlation between Variables of Interest

	Variables	1	2	3	4	5
1	Depression	-				
2	Impulsivity	.63***	-			
3	Inhibition	-.61***	-.78***	-		
4	Misses	.09	.08	-.11	-	
5	False Positives	.31*	.26	-.31*	.08	-

*p < .05; **p < .01. ***p < .001

Impulsivity and inhibition as predictors of depression. Step 1 of this regression included impulsivity as a predictor of depression, this model significantly predicted depression (R^2 = .40, F [1,40] =26.69, p < .001). Post-hoc power analyses revealed the power to be sufficient at .99. The second step of this model included inhibition. The introduction of inhibition increased the size of the prediction (R^2 = .43, F [2,39] = 14.92, p < .001). Post-hoc power analyses revealed the total power of the model to be sufficient at .99. However, further investigation indicated that impulsivity was the only predictor that was significant on its own. The general superiority of the combined model, however, suggested that one variable may be mediating the other. Table 5 presents a summary of the results of this regression analysis.

Table 5
Hierarchical Multiple Regression Analysis Predicting Depression.

	Depression	
Predictors	ΔR^2	β
Step 1	.40***	
Impulsivity		.63
Step 2	.43***	
Impulsivity		.40
Inhibition		-.29
	R = .66	
	Adj.R^2 = .40	

*p < .05; **p < .01. ***p < .001

Impulsivity as a mediator of the relationship between inhibition and depression. A tentative model was constructed in order to account for the relationship between inhibition, impulsivity and depression. It was suggested that impulsivity may be mediating the relationship between inhibition and depression. The Preacher and Hayes mediation analysis

revealed that along the a-path, inhibition was significantly negatively related to impulsivity ($p < .001$). Along the b-path, impulsivity was significantly positively related to depression ($p < .05$). Along the c-path, inhibition was significantly negatively related to depression ($p < .001$). Finally, along the c'-path, inhibition was no longer significantly related to depression. This final model suggested that impulsivity acted as a good mediator between inhibition and depression ($R^2 = .43$, F [2,39] = 14.92, $p < .001$). Figure 1 presents a summary of this mediation analysis (Preacher & Hayes, 2008). In order to strengthen the evidence for the relationship between inhibition, impulsivity and depression, a final multiple regression analysis was run. During the first step of the multiple regression, depression was entered in order to control for it. Inhibition was entered as the second step, and impulsivity was entered as the dependent variable. In the first step of the model, depression significantly predicted impulsivity ($R^2 = .40$, F [1,40] = 26.69, $p < .001$). However, when inhibition was added to the model, the prediction was improved ($R^2 = .64$, F [2,39] = 35.31, $p < .001$). Further investigation of standardised coefficients indicated that inhibition had reduced the contribution of depression from .63 to .25. Inhibition now uniquely accounted for the majority of the relationship with -.62. Post-hoc power analyses using G*Power 3.1 revealed the total power of the model to be sufficient at .99. Table 6 presents a summary of the results of this regression analysis.

Table 6
Hierarchical Multiple Regression Analysis Predicting Impulsivity.

	Impulsivity	
Predictors	ΔR^2	β
Step 1	.40***	
Depression		.63***
Step 2	.64***	
Depression		.27*
Inhibition		-.62***
	R = .80	
	Adj.R^2 = .63	

*$p < .05$; **$p < .01$. ***$p < .001$

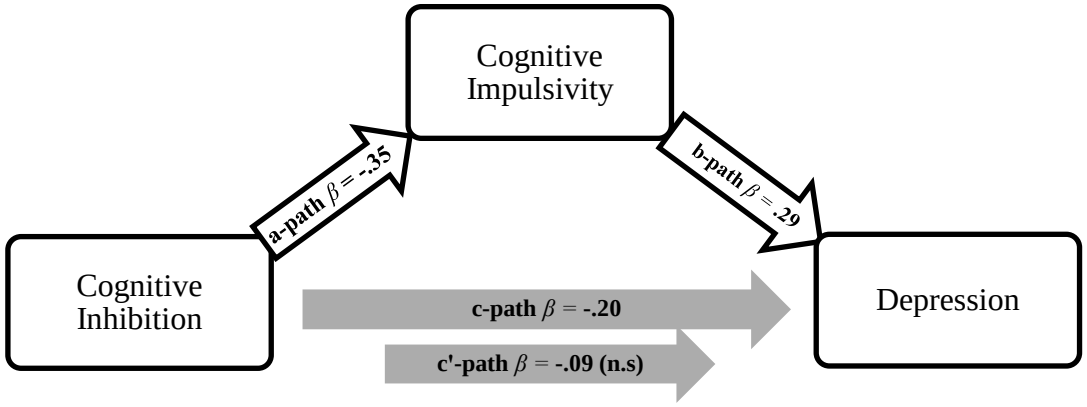

Figure 1. Preacher and Hayes Mediation Analysis: Inhibition and Depression Mediated by Impulsivity.

Discussion

The first hypothesis was supported. Working memory performance was capable of predicting inhibition – but only in part. Working memory performance in this research consisted of both misses and false positives. However, only false positives predicted inhibition significantly – the greater the number of false positives, the lower the amount of inhibition. This finding supported existing literature which suggests that working memory performance is related to cognitive inhibition. Further, the findings of the present research supported that only the inability to inhibit the access of irrelevant information is closely related to cognitive inhibition (Gohier et al., 2009). The second hypothesis was not supported. Working memory performance was not capable of predicting impulsivity. This was not in keeping with the literature, and suggested that there may be a more nuanced relationship between impulsivity and inhibition (Hinson et al., 2003; Romer et al., 2011). The third and fourth hypotheses were both supported. Inhibition showed a strong negative relationship with depression, and impulsivity showed a strong positive relationship with depression. This suggested that the less inhibitory control the participant felt that they had over their thoughts and actions, the greater their level of depression. Further, the more impulsive participants felt they were in their daily thoughts and actions, the greater their level of depression. Prior research supports this, but only indirectly. It is suggested that people with depression have reduced inhibition (Gohier et al., 2009). Further, it is suggested that people

with reduced inhibition show higher impulsivity (Hinson et al., 2003; Romer et al., 2011). The fifth hypothesis was also supported. The results of the present research suggested that inhibition and impulsivity were capable of predicting depression. Inhibition was a negative predictor of depression and impulsivity was a positive predictor of depression. However, impulsivity was a far more significant predictor than inhibition. Additional investigations suggested that impulsivity may mediate the relationship between inhibition and depression. These findings expand literature on inhibition, impulsivity, and depression.

Further, these findings assist in promoting the aims of the present research. The present research aimed to unify the literature by exploring the relationship between working memory processes, inhibition, impulsivity and depression. This was done in order to determine whether the relationship between these variables would inform a working memory training intervention in depression. However, in order to accomplish the second aim, the findings must be explored more thoroughly in relation to the literature.

Impulsivity and working memory – removed but related. The results of the present research suggested that working memory performance was not capable of predicting impulsivity. Neither misses, nor false positives predicted impulsivity. However, this result is valuable, as it alludes to the temporal relationship between working memory, inhibition and impulsivity.

First of all, the type of impulsivity measured in this research was trait-based impulsivity. This was chosen because of its relatively enduring nature and its more cognitive orientation (Stanford, 2009). The results of the present research suggest that the nature of cognitive impulsivity could be too theoretically distant from working memory performance to show a direct relationship. This would suggest that cognitive impulsivity is the result of inhibitory failure, even in its most temporally immediate form, rather than the direct result of the hyperactive reflexive system described by the dual process view of depression (Carver et al., 2013). Further, this would suggest that impulsivity and inhibition are not simple competing processes in depression. Rather, the present research suggests that working memory is more closely related to inhibition, and less closely related to impulsivity - and subsequently, depression. This may be explained by the results of the third and fourth hypotheses, which served to illustrate the relationship between inhibition, impulsivity and depression.

The relationship between inhibition and impulsivity – a false continuum. The third and fourth hypotheses suggested that, impulsivity and inhibition would display a specific and opposite relationship to depression. The vast majority of the research concerned with inhibition, impulsivity and depression implicitly place inhibition and impulsivity on a continuum by describing either inhibition or impulsivity as a state defined by the absence of the other (for an example of this see Tillfors, Mörtberg, Van Zalk, and Kerr, 2013). This may be because of a methodological divide or an assumption regarding these two constructs, common to papers investigating clinical disorders. The dual process view of depression illustrates this simplified form of clinical framing. The dual process view suggests that the reflexive system and the executive system are inversely related in depression. Further, it suggests that when the executive system is hypoactive, the reflexive system is hyperactive (Carver et al., 2013). The present research suggests that this is not an accurate assessment of the relationship between inhibition and impulsivity.

The role of inhibition. Neuroscientific and working memory research supports this. It is suggested that inhibition and impulsivity represent entirely different levels of processing and feedback (Gohier et al., 2009; Rogers et al., 2004). Inhibition is suggested to perform three primary functions. First, inhibition restricts the access of information that is not relevant to the task at hand. Second, inhibition is suggested to restrain the incorrect retrieval of irrelevant information. Third, inhibition is suggested to delete information as it becomes irrelevant. Prior research has suggested that people with depression experience deficits in all 3 aspects of cognitive inhibition, but the inability to restrict the access of irrelevant information is the only aspect that bears a linear relationship with depression (Gohier et al., 2009). The present research suggests that false positives may represent this inability to restrict the access of irrelevant information. Further evidence attributes this to the activity of the orbitofrontal cortex. The orbitofrontal cortex is suggested to inform the content of working memory by attributing emotional salience to neutral stimuli concerned with current task goals (Rogers et al., 2004). The present research suggests that this relationship between false positives and inhibition reflects the misattribution of emotional salience to neutral stimuli. In the present research this would be observed in the matching of letters that were not matched in the working memory task. This evidence supports the proposition that, in the case of depression, the central executive may be incapable of accurately protecting the contents of the episodic buffer. Further, it supports the proposition that the episodic buffer may be flooded with irrelevant information – potentially due to the misattribution of emotional

salience to information that is not salient in the context of established task goals. The results of the fifth hypothesis added to the detail of this finding.

Impulsivity, inhibition and depression. The fifth hypothesis suggested that inhibition and impulsivity would be capable of predicting depression, due to their relationship with it. Together, impulsivity and inhibition accounted for 43% of variability in depression. However, despite the fact that impulsivity and inhibition predicted such a large part of depression, inhibition improved the predictive power of the model by little more than 3%. This suggested that impulsivity had a far stronger relationship with depression. It should be noted that the model was still improved, both in its predictive power and in its ability to generalise to other populations by the introduction of inhibition – but impulsivity was far more important. This is in keeping with neuroscientific and working memory literature, which suggest that people with depression show reduced cognitive inhibition and increased cognitive impulsivity (Gohier et al., 2009; Rogers et al., 2004). However, this does not mean that impulsivity is synonymous with the reflexive system. This literature also suggests that it is the lack of inhibitory control by the central executive that prevents the protection of information in the episodic buffer and the subsequent impulsivity (Baddeley, 2002; Rogers et al., 2004). The present findings suggested that impulsivity may mediate the relationship between inhibition and depression. This mediation model was supported, and suggested that inhibition and depression were mediated by impulsivity. However, due to the small sample size present in this study, and the limited ability of the data to support such a model, it was necessary to posit the possibility that depression and inhibition caused impulsivity, in order to rule it out. It was certainly possible to argue that the inhibitory deficit caused by the misattribution of emotional salience resulted in the negative appraisals observed in depression, and that these, in turn, resulted in subsequent impulsive attributions. However, the results suggested that this was not the case. Depression accounted for a small fraction of impulsivity, and inhibition accounted for more than half of it. This suggested that inhibition was, in fact, largely predicting impulsivity. In turn, this suggested that impulsivity was largely predicting depression.

Impulsivity, inhibition, depression and cognitive theory. This is in keeping with the clinical and associated literature. The cognitive theory of emotional processing suggests that emotions relate the inner concerns of an organism to external events. Within this framework, depression is suggested to be the result of a consistent and impulsive negative evaluation of the environment (Oatley & Johnson-Laird, 2014). More classical cognitive

theory suggests that these impulsive negative evaluations are concerned with the environment, the self, and the future. Further, it is suggested that these impulsive negative evaluations increase the chance of becoming depressed (Beck, 2005). This is in keeping with the observed results. The cognitive theory of depression continues to suggest that these automatic ways of thinking can be divided into a number of cognitive distortions of reality. These cognitive distortions include all-or-nothing thinking, overgeneralisation, over-attending to negative events, ignoring positive events, jumping to unfounded conclusions about causal relationships in the world, and emotional reasoning (Gilbert, 1998). These distortions, especially those concerned with the impact of emotional salience, are expanded by research on executive functioning, which suggest that both the amygdala and the orbitofrontal cortex are responsible for learning and activating these automatic responses (Oatley & Johnson-Laird, 2014; Roberts et al., 2004). However, the present research suggests that impulsivity holds a complex relationship with inhibition. Neuroscientific research related to the organisation of the brain may assist in accounting for this relationship.

A nested neurological hierarchy of impulsivity and inhibition in the context of working memory and depression. The structure of a nested neural hierarchy is suggested to be composed of higher order features at the top of the hierarchy, with lower order features at the bottom of the hierarchy. The nested nature of this hierarchy means that the less complex lower order features are held within the more complex higher order features (Feinberg, 2011). Neural synchrony allows different parts of the hierarchy to synchronise their firing in a flexible and adaptive manner. This process allows lower order features and higher order features to contribute in series and in parallel (Uhlhaas et al., 2009). Finally, highly interconnected association regions, consisting of amygdaloid and cortex regions change how the hierarchy communicates. This allows simultaneous automatic and voluntary control of processing that reflects what is observed in impulsivity and inhibition (Pessoa & Adolphs, 2010).

Further, this allows for unified experiences, such as anxiety or depression, to modulate the behaviour of many brain structures (Pessoa & Adolphs, 2010). In the present research, inhibitory failure is suggested to be caused by the combination of low cognitive control and the misattribution of emotional salience. This is, in a sense, a form of impulsivity, but the evidence suggests that it is more closely related to inhibition. The present research suggests that low inhibitory control may allow impulsive cognitive processing to emerge. Once these impulsive cognitive processes emerge, they may further inform the misattribution

of emotional salience in future processing (Rogers et al., 2004). After this occurs, inhibitory processes may be further suppressed by the disorganised and repetitive flow of information in the episodic buffer. Thus, once a depressed state begins, it may inform further processing that maintains the depressed state, such as impulsive attributions (Pessoa & Adolphs, 2010). This could account for the difficulties encountered in psychotherapy. However, it also suggests that working memory training may be a viable form of cognitive training for moderate and severe depression, as this training increases the activity of cortical association regions that are capable of moderating neighbouring brain activity (Baddeley, 2002; Bickel et al., 2011; Feinberg, 2011; Morrison & Chein, 2011; Olesen et al., 2004; Uhlhaas et al., 2009).

Therefore, both the results of this research and the combined literature, suggest that it is impulsivity that is most closely linked to depression, and in turn, that inhibition is most closely linked with impulsivity. The role of working memory is suggested to provide some foundation for lack of inhibition, but these may also be informed by impulsive cognitive processes. It is as of yet uncertain precisely how this may operate, and it is almost certainly impossible to reduce these processes to a simple collection of boxes and arrows. However, this research also suggests that traditional cognitive models may be useful in exploring them. The evidence presented in this research suggests that the strengthening of inhibitory processes may facilitate a reduction in impulsivity and a subsequent reduction in the cognitive features of depression.

Limitations and Directions for Future Research

This study sought to amalgamate a body of literature spanning working memory, impulsivity, inhibition, neuroplasticity, and cognitive training. Further, it sought to do this using a relatively long research session; at a single point in time; using self-report and performance measures; from a single group of participants; gathered using relatively ad-hoc sampling methods. The primary reason for this was the lack of time and resources. This does not mean that this research was without merit. A large and well-established body of research provides substantial support for the findings of the present research. However, there are a number of considerations that should be mentioned, and a number of recommendations that should be made.

First and foremost, this study was a correlational design. This research used statistical techniques suited to establishing a limited degree of causality. However, an experimental or quasi-experimental design should be conducted in the future. Further, a future research design

should attempt to include brain imaging – specifically structural and functional magnetic resonance imaging (fMRI). This would allow for the researcher to determine whether functional or structural differences exist between depressed and non-depressed groups, and whether change occurred over the course of an experimental intervention. To accommodate this, future research should attempt to collect data along multiple data points, preferably culminating in a longitudinal approach spanning a minimum of 4 weeks. This would account for the time needed for enduring neurological changes to occur (Lövdén et al., 2013). The present research sessions were also relatively long at 2 hours. Although it is possible that cognitive fatigue could have facilitated the emergence of the relationships observed in this research, it is also possible that fatigue caused participants' performance to be lower than it otherwise would have been. This could easily have interfered with the data that was collected.

Second, this study used a single working memory measure – the *n*-back task. The *n*-back task is considered to be a relatively pure and uncomplicated way to test the performance of working memory (Jaeggie et al., 2010). It is also capable of producing a variety of qualitatively different forms of data. These include a variety of levels, capable of detecting small differences between participants, and a variety of different forms of error. For these reasons, the *n*-back working memory task should be included in future research. However, a variety of other working memory tasks exist that are capable of making their own unique contribution. The Tower of London, the Trail-making Task, and the Stroop Task are just a small number of working memory tasks that should be considered for inclusion in future research (Rogers et al., 2004). The present research also used only a small variety of self-report measures for determining levels of impulsivity and inhibition. The measures chosen, although widely used and well supported, should be complimented with more targeted measures, capable of detecting cognitive impulsivity and cognitive inhibition more directly. Further, these measures should seek to avoid some of the common problems present in self-report measures, such as response sets. Performance measures or stimulus-response paradigms may be an ideal replacement.

Third, this study used a subclinical population. This was necessary due to time and resource limitations. Research suggests that the greatest cognitive deficits emerge in moderate and severe depression (Gruber, Zilles, Kennel, Gruber, & Falkai, 2011; Mc Dermott & Ebmeier, 2009). Despite the fact that the present research was able to detect some evidence of cognitive deficits, the present research cannot comment on how this relationship functions in more severe depressed samples. For this reason, it is essential that future research

include a clinical sample.

Fourth, this study used purposive sampling. This was done to ensure that there was an adequate spread of depression scores in the sample, considering its small size. This was necessary due to time and resource constraints. This meant that this research could not adequately control other details of the sample it ended up investigating. However, this research did manage to control a small number of demographic characteristics. All participants were South African, all participants were undergraduate psychology students, and all participants were between the age of 18 and 23. However, beyond these demographic characteristics, the need to account for a spread of depression data meant that the study could not account for other characteristics. The result of this is that this research had a large number of females (n = 38), and few males (n = 4). Further, this study had more white people than any other racial group. This sample is in keeping with the demographic characteristics of undergraduate psychology courses at the University of Cape Town. However, in a country like South Africa, with a variety of cultures and languages, it is important to produce data that is equal in all racial groups. The present research was forced to control for these factors by inserting them into the analyses and assessing them – however, this does not mean that the results can be generalised to populations that were underrepresented. Additionally, despite the fact that age and education levels were not deemed important for the present research, this was only deemed to be the case because the sample was drawn from undergraduate psychology students. All students were roughly of a similar age and a similar education level. This also means that the results of this study cannot be generalised beyond young adults of university-level education. Overall, the implication of this is that the results of this research may be more in keeping with findings in highly developed western contexts. For the present research, this may have been beneficial, due to the aims of the research. However, this also means that the results were not generalisable to the majority of the population of South Africa or other developing nations. Future research in South Africa should ensure that it is representative of all racial groups.

Contributions and Practical Implications

No previous research has attempted to investigate working memory, cognitive impulsivity and cognitive inhibition in a South African context. This research has given evidence to support the universal nature of these constructs. Additionally, no previous research has attempted to understand the underlying mechanisms of depression using the

combined literature from working memory, inhibition, impulsivity, neuroplasticity and cognitive training. This research has succeeded in providing empirical evidence for the integration of this literature. Impulsivity and inhibition have commonly not been included in cognitive theories of depression. When they have been included, any mention of impulsivity and inhibition is either divorced from foundational executive functioning literature and associated neuroscientific evidence, or one of these constructs has been excluded (Carver et al., 2013; Gohier et al., 2009; Gruber et al., 2011; Marazziti et al., 2010; McDermott & Ebmeier, 2009; Oatley & Johnson-Laird, 2014; Richards, 2011). The present research suggests that the integration of working memory, inhibition and impulsivity allows for a richer and more descriptive integration of related neuroscientific evidence. Further, this research suggests that the integration of this literature draws attention to the cognitively impulsive nature of depression. This serves as a stark contrast to the traditional conception of depression as a disorder of simple cognitive incapacitation and psychological slowness. This research suggests that an expanded perspective of depression will facilitate the use of cognitive training interventions, specifically those designed to increase the performance of working memory processes, such as cognitive inhibition.

Beyond these immediate implications, wider implications should be considered that take due consideration of cognitive training, psychopharmacology and neuroplasticity. A significant body of literature supports the highly plastic nature of the brain (Draganski et al., 2004; Lövdén et al., 2013; Raz & Lindenberger, 2013; Reed et al., 2011). Further, associated bodies of literature concerned with general health and wellness suggest that a number of physical and psychological factors impact the level of neuroplasticity in the brain. Factors such as physical exercise are suggested to increase the neuroplasticity of the brain. Conversely, high states of negative arousal such as anxiety or depression are suggested to reduce neuroplasticity (Gapp, Woldemichael, Bohacek, & Mansuy, 2012; Li, 2013). Further evidence suggests that a number of different psychopharmaceuticals may also increase the neuroplastic qualities of the brain (Krystal, 2007).

When depression and cognitive training are considered in the context of the present research and associated literature, there is significant evidence to suggest that working memory training may improve the outcomes of psychotherapy through its effect on executive control processes (Bickel et al., 2011; Morrison & Chein, 2011; Olesen et al., 2004). Additionally, specific physical activities and psychopharmaceuticals, may serve to increase neuroplasticity. These would further assist in reducing the deficits of executive functioning

present in depression by increasing the rate of neurological change (Gapp et al., 2012; Krystal, 2007; Li, 2013). This evidence suggests that working memory training could serve to make psychotherapy more effective for cases of moderate and severe depression. Further, if this novel style of treatment is effective, it may assist in reducing the global burden of depression.

Conclusion

This research aimed to clarify the relationship between working memory performance, inhibition, impulsivity and depression in order to determine whether a working memory training intervention could be effective. This research argued that if working memory, inhibition and impulsivity were indeed related, performance on a working memory task would be capable of predicting inhibition and impulsivity. This research found that false positives successfully predicted inhibition. Further, if impulsivity and inhibition were related to depression, then theory suggested that impulsivity would be positively related to depression, and inhibition would be negatively related to depression. The present research found this relationship. Finally, it was suggested that these opposing relationships would be capable of predicting the severity of depression. This research found that inhibition and impulsivity were good predictors of depression. Further impulsivity was found to mediate the relationship between inhibition and depression. This link was necessary, both to establish the importance of impulsivity and inhibition in depression, and to inform a future implementation of working memory training in a clinical population with Major Depressive Disorder. Further implications, although highly tentative given the present findings, suggested that working memory training may allow psychotherapy to be effective in cases of moderate and severe depression. Further, associated literature suggested that the most promising results would occur as the result of a combination between therapeutic practices and interventions designed to maximise neuroplasticity. This would include psychotherapy and working memory training designed to address cognitive distortion and cognitive deficits, and psychopharmacological interventions and lifestyle interventions designed to maximise neuroplasticity. Further research is needed to investigate the relationship between these factors and the potential impact of clinical interventions on people with depression.

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Appendix A

Consent Form

You are asked to participate in a study conducted by:

Matthew Snelling (Principal Investigator): mjtlsnelling@gmail.com

Kai Schramm (Principal Investigator): kaitschramm@live.co.za

Dr. Susan Malcolm-Smith (Supervisor): susanmalcolmsmith@gmail.com

Dr. Samantha J. Brooks (Co-Supervisor): drsamanthabrooks@gmail.com

Department of Psychology, University of Cape Town

Your participation in this study is entirely voluntary. Please read the information below and ask questions about anything you do not understand before deciding whether or not to participate.

Purpose of the Study

Memory studies contribute to theoretical knowledge and help us to expand what we know about memory. Understanding how various factors influence performance on memory tasks helps us elucidate precisely how memory functions in daily life, and in clinical populations. The cognitive load required by the *n*-back working memory task can measure discreet variances in working memory capacity. The *n*-back task represents a practical approach to discerning memory functions. Two honours projects may make use of the data collected.

Procedures

If you volunteer to participate in this study, you will be asked to do the following things:

You will be required to complete the following survey.
The survey is designed to help us with screening of appropriate participants. All of the information you provide will be kept confidential, so please answer all questions as accurately as you can.

If you are selected after screening, you will be required to participate in a 2 hour testing session.

The first part of the testing session will consist of 5 measures that need to be completed. These measures, excluding the Trail-making Task (which will be completed on paper), will all be provided as computer based tasks that will allow for you to quickly click your preferred answers. These psychometric tests are all short and will not take up much time.

The remainder of the testing session will consist of a computer-based task that will test your Working Memory. You will first be given an easier level of the task known as the 1-back to get a handle on the task. Following this you will be given the 2-back and 3-back task. Each task will take half an hour of your time. You will receive a short 5 minute break between all Working Memory tasks.

After completion of the 3-back task you will need to fill in your participation form. After this is completed you may ask the experimenter more about the intention of the study you participated in.

Potential Risks and Discomforts

None of the tasks are overly demanding. However, the tasks are computer based, so you might experience some weariness from staring at a computer screen for an extended period of time. You will, however, be provided with breaks to prevent any form of overexertion.

Potential Benefits to Subject or Society

You will participate in a Working Memory task that has the capability of improving Working Memory.

Of societal importance is the possibility that the information we gain could improve our understanding of the relationship between Working Memory and other factors. There are widespread implications for any greater understanding of the relationship between Working Memory and psychopathology.

Compensation for Participation

You will be awarded 4 SRPP points after successfully completing the memory games.
Beyond this, there are no other direct benefits to you for participating.

Confidentiality

Any information that is obtained in connection with this study, and that can be identified with you, will remain confidential and will only be disclosed with your permission or as required by law. Confidentiality will be maintained in the following ways:

Personal identifiers will be removed from research-related information. A code will be attributed to you based on the group you will be representing in the study. Your identity is therefore not connected to the data.

Paper-based / computer–based records will only be available to researchers involved in the study.

Participation and Withdrawal

Participation in this study is voluntary. If you volunteer to participate in this study, you may withdraw from the study at any stage without consequences. You may also refuse to answer questions you do not want to answer. There is no penalty for withdrawing from the study, but completion is required to be awarded SRPP points.

If you are interested in the outcome of our studies, please let us know so that we can inform you of the results once they become available. You are also more than welcome to attend a Colloquium in the Department of Psychology to be held on the 31st of October 2014, where the results will be presented.

I understand the procedures described above. My questions have been answered to my satisfaction, and I agree to participate in this study.

_____

Printed Name of Subject

_____ _____

Signature of Subject Date

_____ _____

Signature of Witness Date

Appendix B

Example of Online Demographic Questionnaire

Name	
Age	
Gender	
Ethnicity	
Year of Study	
Student Number	
Course Code	
Contact E-mail	
Nationality	
Psychiatric Diagnoses	
Drug History	
Current Medications	
Glasses/Hearing Aid	

Appendix C

Hospital Anxiety and Depression Scale

I feel tense or wound up	Most of the time 3	A lot of the time 2	Occasionally 1	Not at all 0
I still enjoy the things I used to enjoy	Definitely as much 3	Not quite as much 2	Only a little 1	Hardly at all 0
I get a sort of frightened feeling as if something awful is about to happen	Quite badly 3	Not too badly 2	A little 1	Not at all 0
I can laugh and see the funny side of things	As much as I always could 3	Not quite so much now 2	Definitely not so much now 1	Not at all 0
Worrying thoughts go through my mind	A great deal of the time 3	A lot of the time 2	From time to time 1	Only occasionally 0
I feel cheerful	Not at all 0	Not often 1	Sometimes 2	A lot 3
I can sit at ease and feel relaxed	Definitely 3	Usually 2	Not often 1	Not at all 0
I feel as if I am slowed down	Nearly all the time 3	Very often 2	Sometimes 1	Not at all 0
I get a sort of frightened feeling like butterflies in my stomach	Not at all 3	Occasionally 2	Quite often 1	Very often 0
I have lost interest in my appearance	Definitely 3	I don't take so much care as I should 2	I may not take quite as much care 1	I take just as much care as ever 0
I feel restless as if I have to be on the move	Very much 3	Quite a lot 2	Not very much 1	Not at all 0
I look forward with enjoyment to things	As much as ever 3	Rather less than I used to 2	Definitely less than before 1	Hardly at all 0
I get sudden feelings of panic	Very often 3	Quite often 2	Not often 1	Not at all 0
I can enjoy a good book or programme	Often 3	Sometimes 2	Not often 1	Very seldom 0

Appendix D

Beck Depression Inventory - II

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Appendix E

Barratt Impulsivity Scale - 11

		Rarely/Never	Occasionally	Often	Almost always
Q.		1	2	3	4
1	I plan tasks carefully				
2	I do things without thinking				
3	I make up my mind quickly				
4	I am happy-go-lucky				
5	I don't "pay attention"				
6	I have "racing thoughts"				
7	I plan trips well ahead of time				
8	I am self-controlled				
9	I concentrate easily				
10	I save regularly				
11	I "squirm" at plays or lectures				
12	I am a careful thinker				
13	I plan for job security				
14	I say things without thinking				
15	I like to think about complex problems				
16	I change jobs				
17	I act on "impulse"				
18	I get easily bored when solving thought problems				
19	I act on the spur of the moment				
20	I am a steady thinker				
21	I change where I live				
22	I buy things on impulse				
23	I can only think about one problem at a time				
24	I change hobbies				
25	I spend or charge more than I own				
26	I have outside thoughts when I am thinking				
27	I am more interested in the present than in the future				
28	I am restless at talks or lectures				
29	I like puzzles				
30	I plan for the future				

Appendix F

Self-Regulation Questionnaire

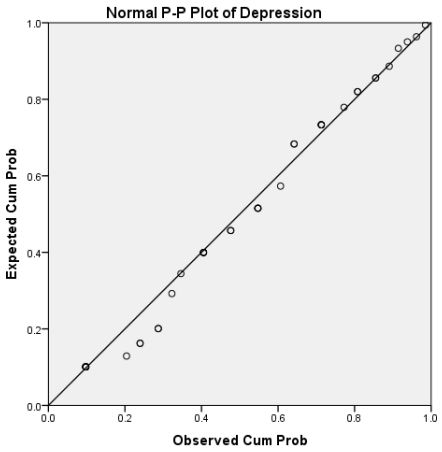
		SD	D	U	A	SA
1	I usually keep track of my progress towards my goals	1	2	3	4	5
2	My behaviour is not that different from other people's	1	2	3	4	5
3	Others tell me that I keep on with things too long	1	2	3	4	5
4	I doubt I could change even if I wanted to	1	2	3	4	5
5	I have trouble making up my mind about things	1	2	3	4	5
6	I get easily distracted from my plans	1	2	3	4	5
7	I reward myself for progress toward my goals	1	2	3	4	5
8	I don't notice the effects of my actions until it's too late	1	2	3	4	5
9	My behaviour is similar to that of my friends	1	2	3	4	5
10	It's hard for me to see anything helpful about changing my ways	1	2	3	4	5
11	I am able to accomplish goals I set for myself	1	2	3	4	5
12	I put off making decisions	1	2	3	4	5
13	I have so many plans that it's hard for me to focus on any one of them	1	2	3	4	5
14	I change the way I do things when I see a problem with how things are going	1	2	3	4	5
15	It's hard for me to notice when I've "had enough" (alcohol, food, sweets)	1	2	3	4	5
16	I think a lot about what other people think of me	1	2	3	4	5
17	I am willing to consider other ways of doing things	1	2	3	4	5
18	If I wanted to change, I am confident that I could do it	1	2	3	4	5
19	When it comes to deciding about change, I feel overwhelmed by the choices	1	2	3	4	5
20	I have trouble following through with things once I've made up my mind to do something	1	2	3	4	5
21	I don't seem to learn from my mistakes	1	2	3	4	5
22	I'm usually careful not to overdo it when working, eating, drinking	1	2	3	4	5

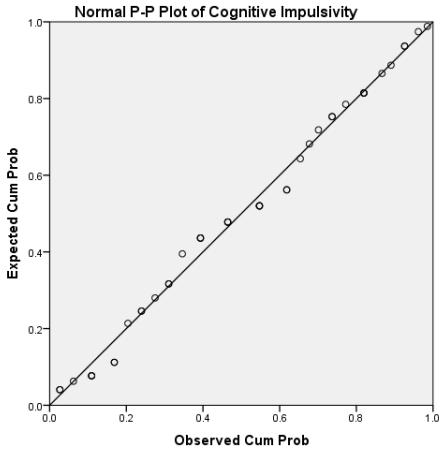
23	I tend to compare myself with other people	1	2	3	4	5
24	I enjoy a routine, and I like things to stay the same	1	2	3	4	5
25	I have sought out advice or information about changing	1	2	3	4	5
26	I can come up with lots of ways to change, but it's hard for me to decide which one to use	1	2	3	4	5
27	I can stick to a plan that's working well	1	2	3	4	5
28	I usually only have to make a mistake one time in order to learn from it	1	2	3	4	5
29	I don't learn well from punishment	1	2	3	4	5
30	I have personal standards, and try to live up to them	1	2	3	4	5
31	I am set in my ways	1	2	3	4	5
32	As soon as I see a problem or challenge, I start looking for possible solution	1	2	3	4	5
33	I have a hard time setting goals for myself	1	2	3	4	5
34	I have a lot of willpower	1	2	3	4	5
35	When I'm trying to change something, I pay a lot of attention to how I'm doing	1	2	3	4	5
36	I usually judge what I'm doing by the consequences of my actions	1	2	3	4	5
37	I don't care if I'm different from most people	1	2	3	4	5
38	As soon as I see things aren't going right I want to do something about it	1	2	3	4	5
39	There is usually more than one way to accomplish something	1	2	3	4	5
40	I have trouble making plans to help me reach my goals	1	2	3	4	5
41	I am able to resist temptation	1	2	3	4	5
42	I set goals for myself and keep track of my progress	1	2	3	4	5
43	Most of the time I don't pay attention to what I'm doing	1	2	3	4	5
44	I try to be like people around me	1	2	3	4	5
45	I tend to keep doing the same thing, even when it doesn't work	1	2	3	4	5
46	I can usually find several different possibilities when I want to change something	1	2	3	4	5
47	Once I have a goal, I can usually plan how to reach it	1	2	3	4	5

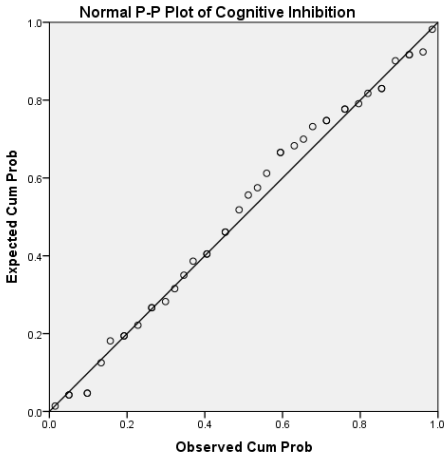
48	I have rules that I stick by no matter what	1	2	3	4	5
49	If I make a resolution to change something, I pay a lot of attention to how I'm doing	1	2	3	4	5
50	Often I don't notice what I'm doing until someone calls it to my attention	1	2	3	4	5
51	I think a lot about how I'm doing	1	2	3	4	5
52	Usually I see the need to change before others do	1	2	3	4	5
53	I'm good at finding different ways to get what I want	1	2	3	4	5
54	I usually think before I act	1	2	3	4	5
55	Little problems or distractions throw me off course	1	2	3	4	5
56	I feel bad when I don't meet my goals	1	2	3	4	5
57	I learn from my mistakes	1	2	3	4	5
58	I know how I want to be	1	2	3	4	5
59	It bothers me when things aren't the way I want them	1	2	3	4	5
60	I call in others for help when I need it	1	2	3	4	5
61	Before making decisions, I consider what is likely to happen if I do one thing or another	1	2	3	4	5
62	I give up quickly	1	2	3	4	5
63	I usually decide to change and hope for the best	1	2	3	4	5

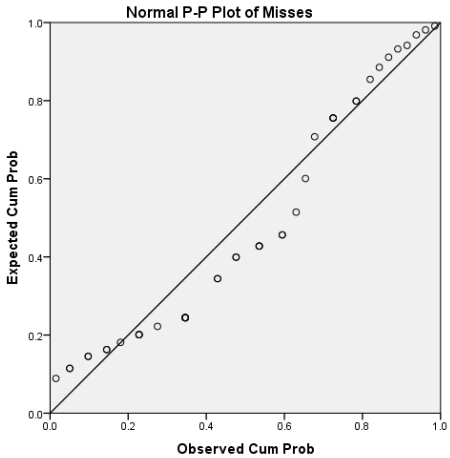
Appendix G

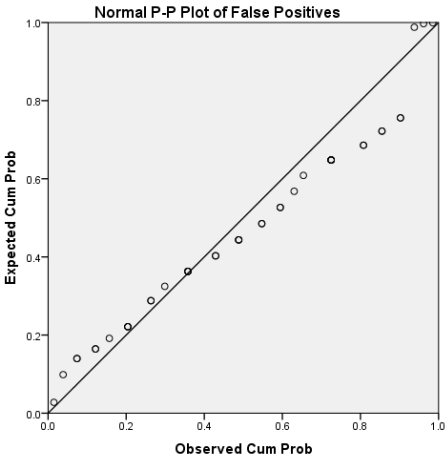
P-p Plots





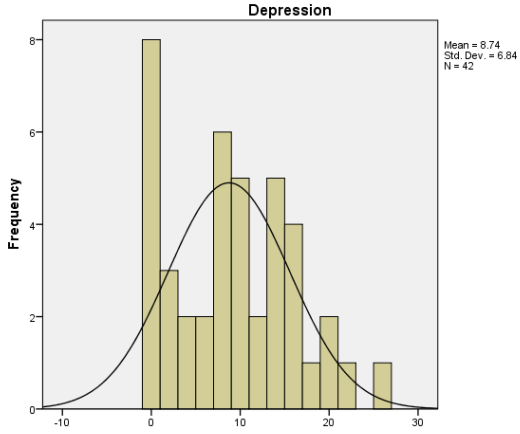


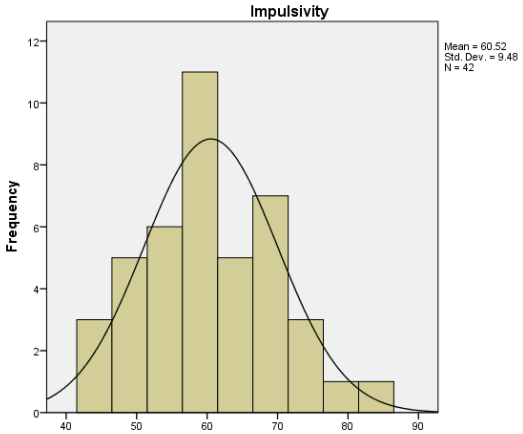


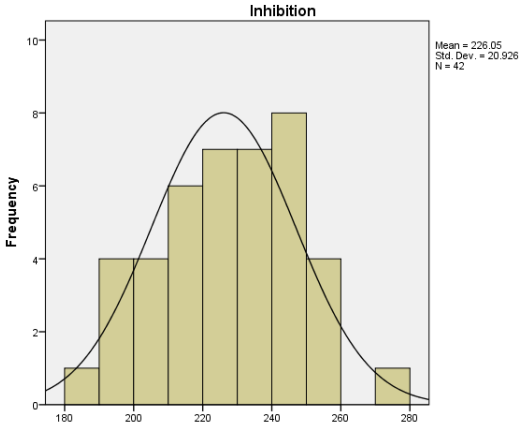


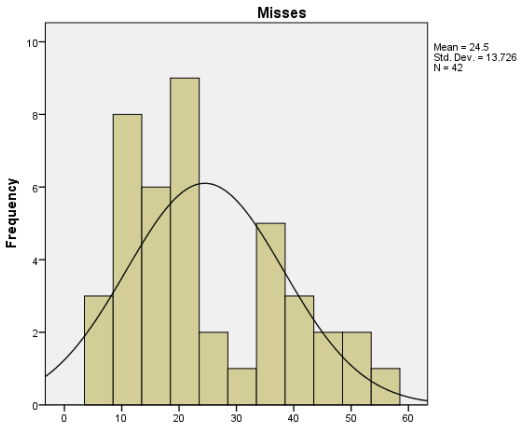
Appendix H

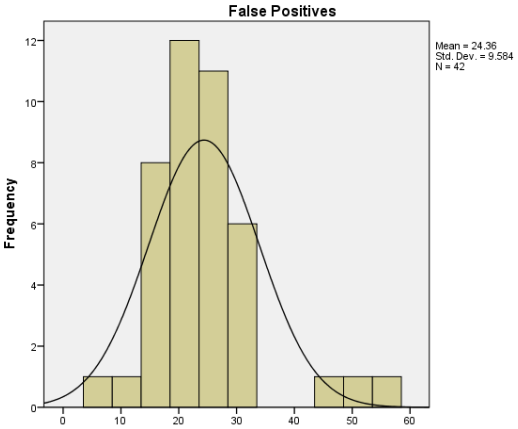
Histograms





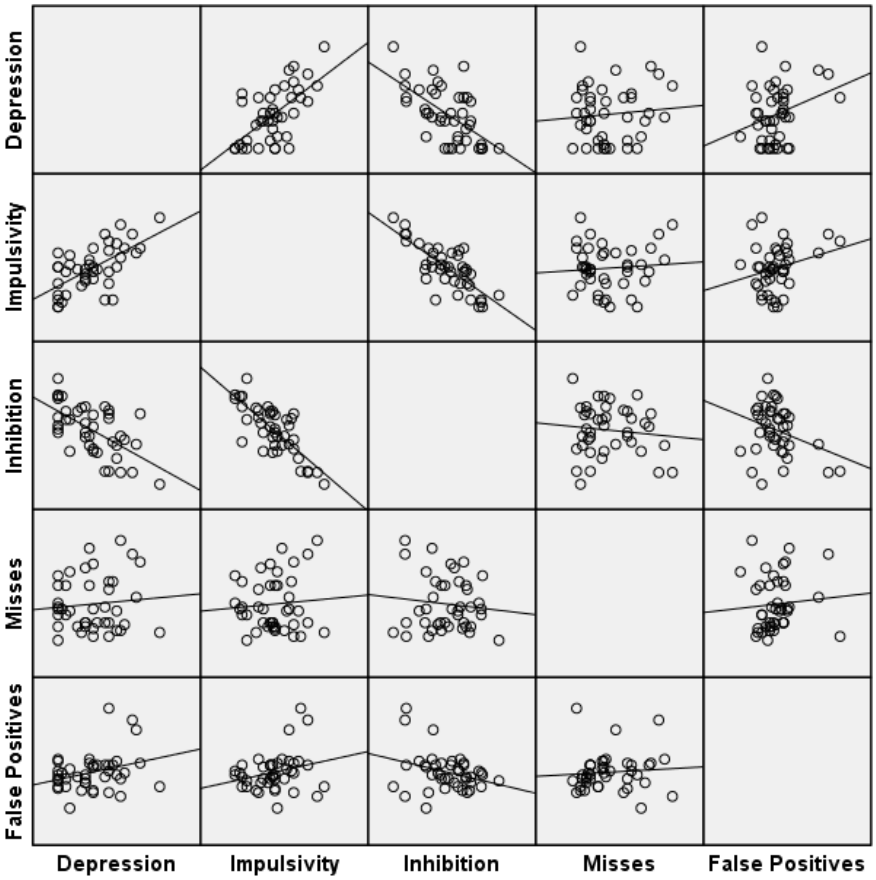






Appendix I

Scatterplots



Appendix J

Regressions with Control Variables Included

Hierarchical Multiple Regression Analysis Predicting Impulsivity and Inhibition with Demographic Variables Controlled for.

	Impulsivity		Inhibition	
Predictors	ΔR^2	β	ΔR^2	β
Step 1	.15		.04	
White vs Indian		-.08		-.01
White vs Coloured		-.07		.08
White vs Black		-.32		.13
Age		.11		.07
Gender		.21		.13
Step 2	.27		.16	
White vs Indian		-.21		14
White vs Coloured		-.10		.12
White vs Black		-.32		.13
Age		.17		.02
Gender		.26		-.18
False Positives		.34*		-.35*
Misses		.12		-.13
	R = .52		R = .40	
	Adj.R^2 = .12		Adj.R^2 = -.01	

*p < .05; **p < .01. ***p < .001

Hierarchical Multiple Regression Analysis Predicting Depression with Demographic Variables Controlled for.

	Depression	
Predictors	ΔR^2	β
Step 1	.05	
White vs Indian		.02
White vs Coloured		-.08
White vs Black		.07
Age		.16
Gender		.19
Step 2	.50***	
White vs Indian		.06
White vs Coloured		-.02
White vs Black		.26
Age		.12
Gender		.05
Impulsivity		.50*
Inhibition		-.24
	R = .71	
	Adj.R^2 = .40	

*p < .05; **p < .01. ***p < .001