

Working Memory Implications of Ruminative Properties in OCD and Nicotine Addiction

Kai T. Schramm (SCHKAI006)

ACSENT Laboratory

Department of Psychology

University of Cape Town

Supervisor: Samantha Brooks

Supervisor: Susan Malcolm-Smith

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### **Abstract**

Various problems, such as impulsiveness and lack of self-control, are known to be influenced by working memory, and many of these problems have been successfully reduced using working memory training. The purpose of this study is to examine the incidence of obsessive compulsive tendencies in nicotine consumers and abstainers. The cognitive load required by the n-back WM task can measure variances in WM deficit according to degrees of smoking behaviour (e.g. never smoked; n=25 & current smokers; n = 22). This quasi-experimental study had a 2x2 ANCOVA design. Measures: Fagerstrom's Test for Nicotine Dependence, Hospital Anxiety and Depression Scale, The Barrat Impulsivity Scale, the Self-Regulation Questionnaire, and a self-report version of the Yale-Brown Obsession and Compulsion Scale. This literature has highlighted the possibility of OCD self-medication with nicotine, which validates investigation as the new proposed method of nicotine treatment could be detrimental due to the WM deficits associated with nicotine consumption. These chronic WM deficits may instead propagate OCD symptomology, even though the acute effects may be beneficial for OCD patients. This study did not find supporting evidence for a working memory deficit in smokers, hence no recommendations could be made in terms of nicotine treatment of persons suffering from OCD.

### Working Memory Implications of Ruminative Properties in OCD and Nicotine Addiction

The irony of mental control and the contradictory nature of mental processing have been put forward by Najmi and Wegner (2009). The fundamental concept portrayed by Wegner is that the established monitoring processes in the brain that enable effective self-regulation have the possibility of malfunctioning under higher mental load, thus producing a counter-intentional error. For instance, a parallel of this ironic processing is exemplified in the rumination processes commonly found in those with nicotine addiction, who are also reactive to negative emotion (Herrera & McChargue, 2011), and Obsessive Compulsive Disorder (OCD), and these ruminations exert a strain on executive functioning under conditions that require a higher mental load (Tumkaya, et al., 2013). However, in those with nicotine addiction there is a lower incidence of OCD symptoms that might suggest that smoking is a self-medication solution for obsessive-compulsive cognitions (Abramovitch, Pizzagalli, Geller, Reuman, & Wilhelm, 2014). The counter-intentional error does raise an interesting idea: do taxing environments link to higher levels of anxiety, and do those who are addicted to nicotine, which may be lowering their OCD symptoms by smoking, cope less well under pressure due to compromised executive functioning?

### **Working Memory: The Mental Control Centre**

Working memory (WM) can be seen as a rumination process, which keeps in mind cognitive strategies particularly for future goals, and does so while in the presence of immediate distraction or attention towards future goals. This system, therefore, allows for the manipulation of variables while keeping them in mind simultaneously. This memory process can influence goal-directed actions by keeping important variables in mind and excluding any distracting variables (Brewin & Smart, 2005; Sweller, 1994). An example of this would be solving a math problem, in which one keeps a number in mind while adding two other numbers and then dividing the number in mind by it (Miller, 1956).

Working memory is comprised of four systems, according to Baddeley and Hitch (1974) that enable the alteration of information in real time. Firstly, the visuospatial sketchpad pertaining to the right prefrontal cortices allows for the storage of mental imagery and the manipulation of information (Baddeley, Cocchini, Della Sala, Logie, & Spinnler,

1999). Secondly, the phonological loop pertaining to the left prefrontal cortices, temporal lobe, and auditory cortices enables an auditory or verbal representation to be kept in mind by means of constant repetition of the relevant information (Logie, Venneri, Sala, Redpath, & Marshall, 2003). Thirdly, the episodic buffer makes long-term storage available to the previous two systems and may also group pieces of information together in order to keep them active for usage, and is associated with hippocampal activation, as well as other cortico-limbic structures (Baddeley, 2000). Lastly, the central executive then coordinates and oversees the action of the other three systems that are often referred to as 'slave systems'. A model of working memory is therefore the operation of the central executive combined with a short-term memory process such as a phonological loop (Baddeley & Hitch, 1974). The central executive controls and integrates information and allocates attention to the salient stimuli and is therefore well placed to be situated in the mid-dorsolateral prefrontal cortex and the ventrolateral prefrontal cortex, based on the associated functions of these brain regions (Collette & Van der Linden, 2002; D'Esposito et al., 1995). Task-relevant information is suggested to be manipulated, monitored, and maintained by the mid-dorsolateral prefrontal, while the selection, retrieval and judgment of task-relevant information is shown to be facilitated by the ventrolateral prefrontal cortex (Badre, Poldrack, Paré-Blagoev, Insler, & Wagner, 2005; Bunge, Ochsner, Desmond, Glover, & Gabrieli, 2001). The final component that facilitates the working memory processes is inhibition, which prevents task-irrelevant information from overloading the capacity of the system and intruding on working memory function. For instance, nicotine craving, derived from activity in mesolimbic arousal and reward regions (e.g. striatum, amygdala, insula), could be seen as a task-irrelevant piece of information that may distract attention.

The dorsolateral prefrontal cortex, the inferior frontal cortex, the anterior cingulate cortex, the supplementary motor area, parietal cortex, and medial prefrontal cortex all show increased activation for tasks that require inhibition (Aron, Robbins, & Poldrack, 2004; Aron & Poldrack, 2006; Bunge et al., 2001; Jonides, Smith, Marshuetz, Koeppe, & Reuter-Lorenz, 1998; Menon, Adelman, White, Glover, & Reiss, 2001). The dorsolateral prefrontal cortex is usually implicated in the inhibition of irrelevant information in situations that require cognitive flexibility, while the other areas seem to be primarily involved in expected inhibition (Aron et al., 2004).

### **WM deficits and Nicotine Consumption**

Consumption of nicotine in the South African population is estimated to be around 21.4% in 2003 (Saloojee & Steyn, 2005) and is linked to core substance-abuse behaviours such as impulsivity and risk-taking. Smokers consume nicotine in order to obtain pleasant feelings and euphoria derived from attachment to the nicotinic acetylcholine receptors that release dopamine in the mesolimbic system (Mameli-Engvall, Evrard, Pons, Maskos, Svensson, Changeux, & Faure, 2006). Constant activation of this system leads to the vulnerability of the attention brain network by degrading the Supervisory Attention System (SAS) proposed by Norman and Shallice (1986), which is a mechanism of habit control. Nicotine use is known to increase proficiency on WM tasks for both smokers and non-smokers, which is determinable by a decrease in omission errors on a task that requires vigilance (Greenstein et al., 2010). Omission errors are mistakes made by not responding to a stimulus that was present, while conversely commission errors are mistakes made by incorrectly responding to a stimulus that is not a target.

Nicotine plays less of a role in executive functions; while of benefit as a means to increase sustained attention, nicotine appears to have no advantageous influence on selective attention, which involves the exclusion of confounding variables in processing (Ernst et al., 2001). Acute administration of nicotine might temporarily boost the activation of cortical and subcortical areas associated with memory due to the presence of nicotinic acetylcholine receptors in these regions (Berke & Hyman, 2000; Volkow, Fowler, Wang, & Goldstein, 2002). On the *n*-back task (Kirchner, 1958), a measure of working memory, smokers and non-smokers performed similarly on lower levels of the task, which requires more sustained attention. Conversely, on higher levels, smokers performed worse than non-smokers, as increased cognitive load requires both selective and sustained attention (L. K. Jacobsen, Mencl, Constable, Westerveld, & Pugh, 2007b; Wilson, Sayette, Fiez, & Brough, 2007). Sutherland et al (Sutherland, Ross, Shakleya, Huestis, & Stein, 2011) demonstrate that acute effects of smoking are linked to improved cognitive performance, and that chronic smoking result in cognitive deficits in the form of executive functioning. They also show that, smokers abstaining for a 12-24 hour period have more difficulty performing at the higher load conditions of the *n*-back task, in comparison to both non-smokers and satiated smokers.

These findings however are inconsistent with others that suggest that smoking abstinence is associated with deficits in accuracy and response time on a WM task (Mendrek

et al., 2006; Xu et al., 2006), while Ernst et al., (2001) reports only a deficit in reaction time and Jacobsen, Mencl, Constable, Westerveld, & Pugh (2007) reports only a deficit in accuracy. Xu et al. (2005) found the left dorsolateral prefrontal cortex and bilateral medial supplementary cortex implicated in the performance differences in nicotine satiated and abstinent conditions. These differences may be attributable to the frontal dopamine system and its effects on WM, as both hyper and hypo activations of frontal circuitry associated with dopamine release can influence WM negatively (Volkow et al., 2002). Kumari & Postma (2005) found that the superior frontal, left superior parietal and the anterior cingulate all showed increased activation due to nicotine administration to non-smokers in an n-back study in which both reaction time and speed were enhanced for these participants. Therefore, the WM deficit portrayed in smokers can be conceptualised as either a deficit in, or an interference to central executive processing due to hyper or hypo dopaminergic stimulation.

### **WM link to OCD**

OCD has two components. The obsessions component is built on intrusive thoughts and urges that reoccur and cause anxiety for the person. The compulsive component is comprised of mental acts and repetitive behaviours that aim to reduce the anxiety created by the obsessions. Compulsions should not be confused with impulsive behaviour that characterises nicotine addiction; an impulsive response to the promise of an immediate gain (Exner, Martin, & Rief, 2009; Greenstein & Kassel, 2009; Harkin & Kessler, 2011; Nakao et al., 2009). The simplest delineation of the two would be in the cognitive element in compulsions, as they tend to have rational explanations for irrational behaviour. Conversely, impulsive behaviour may occur prior to a cognitive evaluation after the event (Claes, Vandereycken, & Vertommen, 2002).

WM cognitive training has been shown to help in impulse control for persons at risk of alcoholism (Weiland et al., 2012). This line of cognitive training research has also revealed that training WM using the n-back task can improve the ability to delay gratification by strengthening cognitive control in methamphetamine patients (Bickel, Yi, Landes, Hill, & Baxter, 2011). Such research has shown that a functioning WM increases the ability to focus on task-relevant information and dismiss distractors. WM can therefore be beneficial in subduing sensory capture (bottom-up processing) from the subcortical structures and therefore enhance the top-down processing directed by the dorsolateral prefrontal cortex

(Zanto, Rubens, Thangavel, & Gazzaley, 2011). Similarly, a better WM might allow for greater top-down processing that may assist in the obsessive thoughts experienced by persons with OCD.

The literature on WM functioning in OCD persons is inconsistent, but the most consistent findings are of hyper-activation in frontal striatal circuits, and this is associated with differences in WM (Ciesielski, Hämäläinen, Lesnik, Geller, & Ahlfors, 2005; Morein-Zamir et al., 2010). These findings, however, seem to omit the subjectively experienced pathology of ruminations that is a constituent of OCD pathology (Exner et al., 2009; Meiran, Diamond, Toder, & Nemets, 2011). On the other hand, some studies show that WM in OCD patients is impaired on spatial WM tasks as well as on verbal WM tasks of increased complexity, implicating a degree of task complexity or required mental load (Harkin & Kessler, 2011; Nakao et al., 2009; Purcell, Maruff, Kyrios, & Pantelis, 1998; Shin et al., 2006; van der Wee, Nic JA et al., 2003). Shin et al. (2006) found that this impairment may be due to a difference in neural circuitry employed during WM task by OCD persons. This study compared OCD patients' performance on the n-back task during a neutral state and symptom provoked state. They found that during the symptom provoked state the right cingulate cortex and the right superior parietal cortex showed increased activations, while in the neutral state the right caudate and the right superior parietal cortex showed increased activation. The activity between the caudate and the orbitofrontal cortex was thus altered in the symptom provoked state. This study is corroborated by the literature in that OCD patients consistently have increased activation in prefrontal-striatal, thalamic and anterior cingulate cortices in response to various cognitive tasks (McGuire et al., 1994; Saxena, Brody, Schwartz, & Baxter, 1998). Collectively, these findings could suggest a deficit in inhibitory mechanisms, which lead to an unselective over-processing of both task-relevant and task-irrelevant information. Alternatively, this activation may represent a compensatory mechanism employed to inhibit task-irrelevant information and therefore enable an over exaggerated control for error (Ciesielski et al., 2005).



### **OCD link to Nicotine Addiction**

The markedly lower 14.5% OCD in smokers may designate this population as more rigid in their thinking than the smoking population without OCD, as they are less likely to succumb to social pressures of smoking. This may be linked to the personality traits that prevail in OCD persons like risk avoidance, vigilant planning, time consuming decision making, and attention to detail (Bejerot, von Knorring, & Ekselius, 2000; Exner et al., 2009). These traits seem to display a stark contrast to the impulsive behaviours associated with nicotine addiction. This may explain the lower prevalence rates of smokers in persons suffering from OCD, but raises the questions about the mechanisms of ruminations and how they result in action. This paper suggests the contrary, as the 14.5% was based on a sample size of 83 OCD people, which may not provide enough power for its external validity. Rather the suggestion is that smokers are more likely to have OCD symptoms. Exploratory research by Lundberg, Carlsson, Norfeldt, and Carlsson (2004) found nicotine use by persons suffering from OCD to assist in the facilitation of decreased anxiety as well as patients' self-reported increased ability to focus. This may suggest that regular smokers perhaps reduce their OCD symptoms via self-medication with nicotine. Nicotine is well established as having calming effects on the user, but it also acts as a stimulant. A parallel would be the substantial literature between the comorbidity of substance abuse and schizophrenia, in which schizophrenics self-medicate their symptoms with nicotine (Khantzian, 1997; Kumari & Postma, 2005). Similarly, OCD persons may thus be self-medicating their symptoms with nicotine.

The first concern is the addictive consequence, in that the continual stimulation of the mesolimbic system will result in an up-regulation of dopaminergic receptor sites, leading to a need for increased stimulation to satiate higher tolerance levels (Balfour, 2002). The second concern is the relation of the inhibitory cognitive control domains (notably dorsolateral prefrontal cortex and ventromedial prefrontal cortex) to nicotine consumption, in that structural changes to these domains noted impairment of cognitive functions that further dependence (Martin-Soelch, 2013). In other words, the addictive qualities of nicotine could lead to a change in the neural structure of the reward system and inhibition system of OCD persons.

### **WM functions as a Buffer of Ironic Processing**

The suggestion here is that a strengthened WM can act as a buffer for both nicotine addiction and OCD. Acute administration of nicotine via smoking enhances WM function by enabling it to be more flexible. In contrast, chronic nicotine exposure may hinder working memory performance, as a weakened WM performance prevents smokers from delaying the gratification of smoking (Bickel et al., 2011; Fisher, Daniels, Jaworska, Knobelsdorf, & Knott, 2012). Nicotine is capable of increasing task-related attention via the stimulation of nicotinic acetylcholine receptors, aiding working memory ability. Nicotine consumption should therefore increase WM ability during lower, but not higher mental load (Ernst et al., 2001). Nicotine usage reveals that abstaining smokers perform worse than smokers and non-smokers on WM tasks, suggesting that although there is an acute enhancement from nicotine use, there is also a weakening of the WM system caused by chronic use (Greenstein & Kassel, 2009). Nicotine addiction and OCD both exhibit an intrusive thought process which suggests that both of these rely on similar neural processes. Although OCD patients make more commission errors (errors based on impulsivity) on the WM tasks similarly to smokers, nicotine only decreases omission errors (Dawkins, Powell, West, Powell, & Pickering, 2007; Shin et al., 2006). Hence, OCD patients may have difficulty with rigid cognitions pertaining to commission errors, whereas those with nicotine addiction have attention deficits pertaining to omission errors.

### **Summary and Conclusions**

In conclusion, the counter-intentional error can be construed as a failure of WM processes. The ruminations exhibited in both nicotine addiction and OCD suggest similar processing for the execution of behaviour that may be counter-intentional. This review has highlighted the possibility of OCD self-medication with the use of nicotine. It is plausible that self-administration of nicotine modifies the prefrontal cortex systems subserving working memory function, such that the function in this brain area becomes temporarily more effective and flexible. Therefore, it is relevant to understand the effects of nicotine on working memory, in order to make suggestions for the medication of OCD persons with nicotine. The concern would be that while acute effects of nicotine might benefit the

individual suffering from OCD, the chronic effects of nicotine may alter brain connectivity in WM such that an increase in OCD symptomology becomes possible.

### **Specific Aims and Hypotheses**

This study aims to examine the incidence of OCD tendencies in nicotine consumers and non-smokers. The cognitive load required by the n-back WM task can measure discreet variances in WM deficit according to varying degrees of smoking behaviour (e.g. never smoked or current smokers). If these discreet variances in WM performance can be associated with the smoking behaviour in OCD persons, then one may therefore posit a connection between the ruminative state experienced in nicotine addiction and OCD. The failure of the monitoring process under high load of information is the key point to be examined and discussed in this thesis. The following hypothesis will be tested:

1. Chronic nicotine consumers will have a higher omission error rate, than non-smokers on the n-back task on lower (1-back) task levels.
2. Chronic nicotine consumers will achieve a higher commission error rate while non-smokers will have a lower commission error rate on higher (2/3-back) n-back task levels.
3. Nicotine consumers will score lower on measures of self-regulation and higher on measures of impulsivity than non-smokers.
4. Higher scores on a measure of nicotine dependence will correlate with higher scores on a measure of OCD severity.

## **Methods**

### **Design and Setting**

I used a quasi-experimental that consists of two groups (non-smokers and abstinent smokers). The within-subject outcome variable under consideration was the level of performance on the n-back task at three different levels (1, 2 and 3-back). The study additionally controlled for various possible covariates, which were measured by self-report questionnaires on OCD, nicotine dependence, self-regulation, and impulsivity. The between-subjects independent variable was group adherence as measured by the nicotine dependence measure. Any score above zero was treated as a smoker. I collected the data at the University

of Cape Town. Two different venues were used due to extended data collection as almost no experimental group participants had participated.

### **Participants**

Primarily, the student body of Upper Campus participated in the study. Participants were recruited via the Student Research Participation Programme that enables Psychology Undergraduate students to participate in research for SRPP points that are required to achieve Duly Performed in courses; this is therefore a self-selected sampling method. Snowball sampling was utilized to speed up the recruitment process for the smoking group: participants recruited through this method were compensated with R150 Pick'n'Pay vouchers.

Forty-one participants were selected via the SRPP program due to its convenient form of sampling and efficiency in advertising to the target group, as well as informing them of exclusion criteria. Six participants were recruited via snowball sampling. The control group (non-smoking) consisted of 25 (Male = 2, Female = 23) participants. The experimental group (smoking abstinent) consisted of 22 (Male = 1, Female = 21) participants. The groups were matched by age, race, and gender. The selection criteria included the Hospital Anxiety and Depression Scale, which aimed at limiting confounds produced by anxiety or depression (Caseras et al., 2006). Therefore, participants above a score of 8 were screened out, however due to high comorbidity of nicotine addiction and depression the cut-off was more lenient for the smoking group and was at 14 to get a large enough sample.

A power calculation for the number of participants could not be made, as a study that combines nicotine addiction and OCD in terms of Working Memory has not been published. Rather the literature surrounding the field was used as a guideline in that these studies averaged on 20 persons per group for WM research (Abramovitch, Abramowitz, & Mittelman, 2013).

**Exclusion criteria.** OCD can be distinguished by early (10-12 years) and late onset groups (18-23); the SRPP program provided a sample of over 18 years of age, ensuring that there is an inclusion of both onset groups. Testing a younger group would be problematic as males exhibit OCD more frequently than females at the early onset stage (Exner et al., 2009). In regards to WM it is also important to exclude students with learning disorders (dyslexia, ADHD) as these are associated with WM deficits (Sutherland et al., 2011). Furthermore, no other comorbid psychological disorders should be visible in the participants as this would

confound the results. Participants with medication that may alter working memory performance due to the cognitive changes that the drug may produce as in antidepressants were excluded (Levkovitz, Alpert, Brintz, Mischoulon, & Papakostas, 2012). Visual impairments, in particular, deuteranomaly and protanomaly due to the primary usage of red and green colour of the main task needed to be excluded. Smokers needed a minimum score of 1 on the nicotine dependence measure and had to abstain for a 24 hour window prior to testing, this was based on self-report from participants. Snowball sampling enabled a more direct way of finding smokers that were willing to abstain for a 24 hour period.

### Measures and Manipulations

There were two screening measures, one experimental task, and four questionnaire measures. Each is described in turn below.

**Screening measures.** The two screening measures were a demographic questionnaire and the Hospital Anxiety and Depression Scale.

*Demographics.* Basic demographic information was acquired with a short online questionnaire which ascertained age, gender, level of education, smoking habits, medication intake, and current and past psychological and medical illness. The questionnaire was available on <https://eu.qualtrics.com>. Smoking habit was used to designate the group the participant belongs to. See Appendix I.

*Hospital Anxiety and Depression Scale (HADS).* This scale determines the level of anxiety or depression felt at the time of the testing. The scale is rated from 0-3 for a total of 21 possible for either the depression or anxiety subscale. An example of an item of anxiety would be “I am restless and can’t keep still.” A depression item example is “I feel miserable and sad.” The usefulness of this measure is that it ascertains some situational capability in performing the WM task. Anxiety and depression levels have shown to influence circumstantial performance on the n-back task can decrease performance (Caseras et al., 2006). As such the HADS was used as a control measure to select participants with extreme scores. Nicotine use has been shown to have a strong correlation with depression scores on the HADS, as well as low anxiety scores correlating with participants that were not addicted to nicotine (Chettoum, Frih, Djenidi, Rachedi, & Tahraoui, 2012). Lastly, HADS has high levels of internal consistency ( $\alpha = .80$ ; Fatt, Atiya, Heng, & Beng, 2006), and good test-retest reliability ( $\alpha = .91$ ; Spinhoven et al., 1997). The cut-off for the control group exclusion was

set at the recommended score of eight out of 21 for either anxiety or depression (Bjelland, Dahl, Haug, & Neckelmann, 2002). The cut-off for the experimental group was at 14 due the high comorbidity of nicotine addiction and depression.

**Experimental measure: N-back task.** The n-back task designed by Kirchner (1958) is a working memory paradigm that can measure discreet variances in performance between participants. The participant completed the task on a provided laptop. A sequence of letters was presented in which the participant must click the spacebar in response to a stimulus that was  $n$  steps earlier in the task. For instance, 1-back indicates that the participant must click the spacebar if the letter currently on the screen matches the letter that came prior to the current letter. The difficulty of the task is determined by the load factor  $n$ . This study made use of 1-back, 2-back and 3-back conditions. The 1-back condition is a determinant of attention and poor performance on this task indicates unsuitable participants for the 2/3-back conditions that require set-shifting. The task shows promise in regards to face validity, but studies comparing the n-back task to other working memory tasks show only modest correlations at best (Kane, Conway, Miura, & Colflesh, 2007). Notable are the findings that the n-back task seems most effective as a training paradigm for fluid intelligence. Increased performance on the n-back task improves the performance on tests of fluid intelligence (Jaeggi, Buschkuhl, Perrig, & Meier, 2010), but more recently Harrison et al. (2013) suggests it only increases working memory capacity. The n-back task could thus posit as an alternative to the nicotine self-medication by cognitively training the working memory deficit in OCD persons.

Each condition is 25 minutes in length with 5 minutes sessions that are separated by 1 minute break intervals. A timer counts down from 60 seconds during the breaks. The sessions are filled with alternating letters that are represented on a background that simulates distracting variables. A circle is flashed on the screen in between the letters and acts as a focal point. The program used to present the  $n$ -back task is Presentation ([www.neurobs.com](http://www.neurobs.com)). The n-back task performance was calculated in two ways: commission percentage, and omission percentage.

**Questionnaire measures.** Four questionnaires measured nicotine dependence, impulsivity, self-regulation and OCD.

*Fagerstrom Test.* The Fagerstrom Test for Cigarette Dependence (FTND; Fagerström & Furberg, 2008) is designed to determine the level of physical dependence of smokers to nicotine. The FTND has a good test-retest reliability ( $\alpha = .64$ ), which is better than Fagerström Tolerance Questionnaire ( $\alpha = .54$ ; C. S. Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994). The score ranges from 0-10 in total, 0 meaning no nicotine dependence. There are a total of six questions with provided answers (see Appendix E). The control group had participants that only scored zero on the FTND.

*Barrat Impulsiveness Scale.* The Barrat Impulsivity Scale (BIS; Barrat, 1985) is a 30 item self-report scale designed to create a profile of a person's impulsivity based on personality and behaviour. The use of this scale for the study is in the determination of a difference in the profile of impulsivity based in nicotine addiction and in OCD. A divergence in the profiles of the two criteria could be used in two ways. Either a different profile between nicotine addiction and OCD could be used to explain why WM deficits may be a cause for concern for either OCD or nicotine addiction. However, a divergent profile, but similar WM deficits would speak to the confirmation of a general ruminative process in these two criteria. The scale was also chosen due to its short-length (as the participants lack sustained attention capacity) and wide-spread acceptance (Stanford et al., 2009). It has good test-retest reliability ( $\alpha = .83$ ; Stanford et al., 2009).

*Self-Regulation Questionnaire.* The Self-Regulation Questionnaire (SRQ; Brown, Miller, & Lawendowski, 1999) is comprised of 63-items. This self-report measure designed to measure the self-regulation factor that is comprised of goal setting and impulsive factors (Brown, Miller, & Lawendowski, 1999). The SRQ implements a Likert Scale from 1-5, in which 5 represents strong agreement with the statement. The scale is widely used and has good test re-test reliability ( $r_s = .80$ ) and very good internal consistency ( $\alpha = .95$ ) (Büssing et al., 2009). The relevance for the current study is that self-regulation has been shown to be inversely related to working memory performance and impulsivity (Stevens, Quittner, Zuckerman, & Moore, 2002). The SRQ was used to show the deficit in self-regulation in both nicotine addiction and OCD persons (Leary, Adams, & Tate, 2006; O. F. Pomerleau, Fertig, & Shanahan, 1983).

*Self-Report Yale-Brown Obsessive Compulsive Scale.* The Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, et al., 1989) is widely used scale to determine OCD symptoms and severity. Due to the limited scope of the study only the severity of symptoms

were measured. Y-BOCS has recently become available as a self-report scale. Federici et al. (2010) showed a moderate convergence between the clinician and self-report Y-BOCS; however a difference was that resistance items had low agreement and the clinician report yielded severer compulsion ratings. The self-report version asks the same questions as the clinician version. The self-report version is broken down into 5 questions on compulsion and 5 questions on obsession, the scale uses a Likert-Scale from 0-4. The maximum score is 40, while only 0-7 is treated as sub-clinical (Federici et al., 2010).

## **Procedure**

**Preliminary procedure.** The participants were contacted by an advertisement via e-mail from the SRPP program or through a friend and colleague that provided the email address of the researcher. This advertisement allowed for direct access to an online screening measure via a link. After I checked suitability of the participants that met the required criteria for the research, I would then send them an e-mail inviting them to participate in the study. Suitability was determined via the Demographic details and HADS scores obtained in an online survey. I assigned participants to groups based on smoking habits. The smoking group was asked to not smoke for 24 hours prior; this was stressed again in an email the day prior to the smoker's session. Participants were contacted by the researcher and assigned a 2 hour timeslot between 8am and 4pm.

**Follow-up session.** The testing period required an approximate 2 hours of the participant's time. The first step for all participants was a brief explanation of the research being done and the rights the participant has in the research. Participants were made aware that participation is voluntary and they can withdraw from the study at any stage, although completion of the study was required to receive the full amount of SRPP points. A code was used to keep track of the scores of the various participants assuring their anonymity. Informed consent was required before participation was allowed. Participants were then asked to complete a series of measures on Microsoft Excel, which allowed them to click their preferred answer. Directions for each measure were on the measure itself, and questions were answered if there was some confusion. Measures included: Fagerstrom Test, BIS, SRQ, and Y-BOCS.

The n-back task was then explained. The 1-back task was used to ascertain if the participant understands the task as well as determine suitability of participants. The



participants were asked to press the space bar in response to two of the same letters appearing on the screen in sequence. Participants were informed that there would be 5 n-back task sessions of 5 minutes in length for each n-back condition. These 5 minute sessions had a 1 minute break between them. The 1-back condition was 30 minutes in length. A short 5 minute break was provided before the next level. The 2-back condition was then explained to the participant. For the 1 & 2-back condition I would assist participants until I was sure that they had grasped the concept of the task, on the 3-back condition participants were not given any additional help. After completion of the 2-back condition a 5min break was provided to the participants. This allowed participants to better sustain attention in the 3-back task. The 3-back task was then given, which took another 30 minutes. Lastly the participants were thanked for their participation, offered a brief explanation of the study, and given a hand-out on the study aims. Participants that had participated for the gift voucher had to sign off that they had received the voucher instead.

### **Data Analysis**

The data analysis was achieved by implementation of the Statistical Package for the Social Sciences (SPSS) software Version 22. Additionally, score calculations for the measures were programmed on Microsoft Excel to output total scores. The study used a 2x2 ANCOVA, with the co-variants in the form of Questionnaire measures. The smoking status represents the categorical independent variable (smoker and non-smoker). The other independent variable was the cognitive load measured by the n-back task under the 2-back and 3-back condition. The base level working memory was controlled with the 1-back condition. The dependent variable is the WM performance as measured by accuracy, hit rate, omission rate and commission rate.

## **Results**

### **Demographics**

There was no significant difference between the smokers who were acutely abstaining from smoking and the non-smoking healthy controls in age, sex, ethnicity and educational level. See Appendix I for frequency data. Table 1 shows that age, sex, and ethnicity were not significantly different between the smoking and control group.

*Study Exclusions.* From the 151 applicants for the study 47 participants remained after attrition. 63 of the potential participants did not follow through after completion of the online questionnaire nor did they make an appearance for the study itself. 2 participants did not complete the online questionnaire. The remaining 39 participants were excluded as they scored above the cut-off of 14 on the HADS ( $n=24$ ) to control for high levels of depression and anxiety that may detrimentally effect working memory performance (Spinhoven, et al. 1997).

Additionally, some were non-smokers that had applied once the control group was closed due to our cohort limit being reached ( $n=7$ ). Furthermore, a small number of participants had contradicted themselves on the SRQ ( $n=3$ ), and it was important to include only those who were consistent in their answers. We also excluded those who had recently consumed a substance (e.g. cannabis) that might affect working memory performance ( $n=2$ ). Finally, we excluded a Zimbabwean and an American volunteer because we wanted to ensure that the total sample consisted of the local population of South Africans ( $n=2$ ). One person who had admitted to having smoked within the 24hour abstaining period was also excluded.

The remaining 47 participants (Experimental, Smokers  $n=22$ ; Control, Healthy non-smokers  $n=25$ ) were examined in the final analysis. I conducted a 2x2 ANCOVA for each dependent variable of the n-back task, namely errors of omission and errors of commission. The Main effects were group (smokers versus non-smokers) and n-back level (2-back and 3-back). All ANCOVA's were corrected for age, as well as one-back baseline scores as a proxy for baseline IQ, and served as a real-time measure for assessing capability to perform on the working memory task. Table 1 presents the significant differences between the experimental and control groups using T-tests corrected (using Bonferroni correlation) for multiple testing.

Table 1

*Sample Demographic Characteristics (N = 47)*

Variables	Group		<i>t</i>	<i>p</i>	ESE
	Smoking ( <i>n</i> = 22)	Control ( <i>n</i> = 25)			
Age	21.23 (2.81)	20.20 (0.91)	-1.64	.11	0.52
Gender <sup>a</sup>	-	-	.234	.63	0.07 <sup>b</sup>
Ethnicity <sup>a</sup>	-	-	4.529	.19	0.31 <sup>b</sup>
<b>HADS</b>					
Anxiety	7.77 (3.73)	4.28 (3.34)	-3.39	.001**	1.01
Depression	4.64 (3.37)	1.40 (1.38)	-4.12	< .001***	1.32
Total	12.41 (6.72)	5.68 (3.86)	-4.14	< .001***	1.28
BIS	62.55 (9.03)	56.88 (7.12)	-2.40	.02	0.72
SRQ	214.05 (20.83)	234.24 (16.43)	3.71	.001**	1.11
Y-BOCS Obs.	4.91 (4.16)	2.48 (2.08)	-2.48	0.02	0.77
Y-BOCS Com.	5.77 (4.88)	2.36 (2.32)	-2.96	0.01	0.93
Y-BOCS Total	10.68 (7.90)	4.80 (3.79)	-3.19	.003**	0.99
FTND	3.05 (1.70)	0.00 (0.00)	-8.38	.001***	2.68

*Note.* Data in the table are means, with standard deviations in brackets. All probability scores are compared to a threshold that is Bonferroni-corrected for multiple comparisons ( $.05/11 = .005$ ). ESE = effect size estimate (in this case, Cohen's *d*). HADS = Hospital Anxiety and Depression Scale, BIS = Barrat Impulsivity Scale, SRQ = Self-Regulation Questionnaire, Y-BOCS = Yale-Brown Obsessive Compulsive Scale, FTND = Fagerstrom Test for Nicotine Dependence.

<sup>a</sup>Fisher's Exact Test.

<sup>b</sup>Cramer's *V*.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

Firstly, I confirmed that Age, Gender and Ethnicity were equivalent across the groups. For instance it could be problematic if the experimental group was older as this age difference may benefit working memory performance. Age was later analysed in the ANCOVA as a covariate, because there is evidence that brain maturation can influence working memory performance (Casey, Tottenham, Liston, & Durston, 2005). However, for gender and ethnicity there is no evidence to demonstrate that these factors influence working memory (Schmidt et al., 2009).

I had two dependent variables that represented accuracy of working memory performance, these were: commission errors and omission errors. The two main effects investigated were group (smokers abstaining for 24 hours and non-smokers) and cognitive load (2-back and 3-back) as measured by the n-back conditions.

Age and 1-back baseline (a proxy measure of intelligence/trait cognitive ability) were used as covariates in order to control for factors other than smoking status that could influence working memory performance. Additionally, I did not correct for anxiety or depression given that I excluded those volunteers who scored above the cut-off for these measures on the HADS. However, as a subsequent step, I did conduct correlation analyses to examine whether anxiety and depression were related to performance on the working memory task.

### **Working Memory Performance**

*Hypothesis 1* predicted that omission error rates would be higher on the 1-back condition for the experimental group in comparison to the control group. The 2X2 ANCOVA showed no significant difference between groups (experimental & Control). However, excluding the covariates (age and baseline n-back) Table 4 shows a significant difference on the omission errors in the One-back condition. This exclusion was done to explore the effects of age and 1-back baseline; in particular the 1-back baseline was of concern as it may not have functioned as an appropriate proxy for IQ.

**Omission Score.** The Main effect for group on the rate of omission errors was insignificant ( $F=1.433$ ,  $p=.233$ ,  $\eta^2<0.01$ ). The Eta squared indicated a negligible effect size.

On further inspection using post-hoc t-tests we found no significant effect of group on rate of omission on the n-back differences between smokers and non-smokers during the 1-back ( $t=-1.74$ ,  $p=.08$ ), 2-back ( $t=-1.10$ ,  $p=0.28$ ) or 3-back ( $t=-.38$ ,  $p=.70$ ). This is usually not done in the face of an insignificant omnibus; however I wanted to have a closer look at the different levels to see if anything could be discerned as Figure 1 seems to show an effect on the 1-back condition. Similarly, we performed such post-hoc tests for commission scores. The Main effect for cognitive load on omission errors was significant ( $F=59.86$ ,  $p<0.00$ ,  $\eta^2=0.20$ ). Eta squared indicates a small effect size. Using post-hoc t-tests, we found that, regardless of group status (e.g. smokers or non-smokers) all participants had significantly lower omission error rate on the 1-back compared to the 2-back ( $t=-6.16$ ,  $p<0.01$ ). All

participants were also significantly more accurate during the 2-back versus the 3-back ( $t=7.19, p<0.01$ ). Finally, all participants were significantly more accurate during the 1-back compared to the 3-back ( $t=-10.12, p<0.01$ ).

The interaction effect between group and cognitive load was insignificant ( $F=.13, p=0.87, \eta^2<0.01$ ). This Eta squared had a negligible effect size.

However, post-hoc t-tests revealed that during the 1-back level there was a significant difference between smokers and non-smokers in terms of number of omissions ( $t=-3.78, p<0.01, d=1.19$ ) if the scores were not adjusted for age or 1-back baseline. This was done to explore the effect of the 1-back baseline.

Figure 1 shows a representation of the effect of cognitive load on the n-back task. This figure shows that omission errors increase for smokers and non-smokers when task difficulty increases. In the one-back condition the smokers show higher omission errors compared to the non-smokers, but the error rate converges and there is no difference between the groups in terms of omission errors as task difficulty increases.

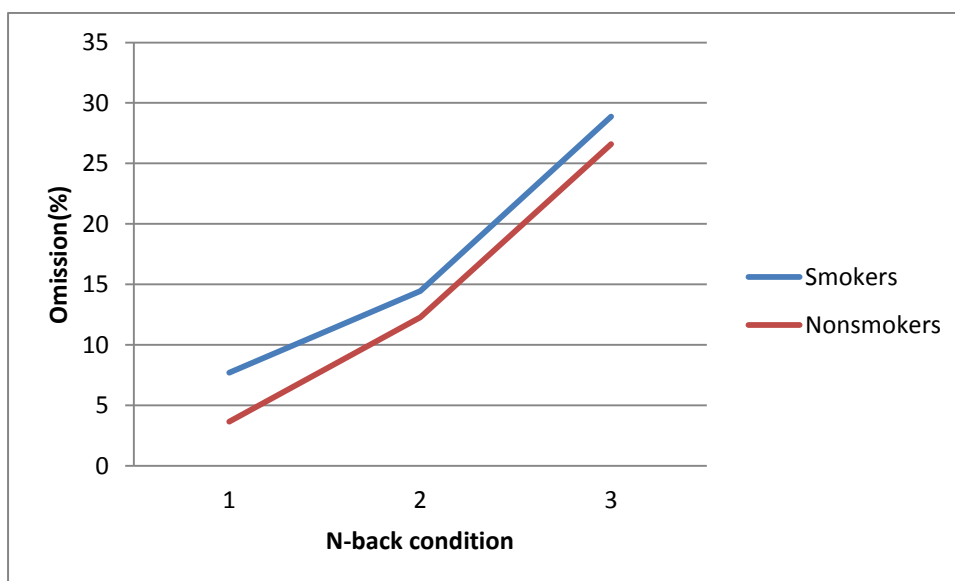


Figure 1. Increase in omission errors by n-back cognitive load condition. X-axis=n-back task difficulty. Y-axis=Omission errors as a percent.

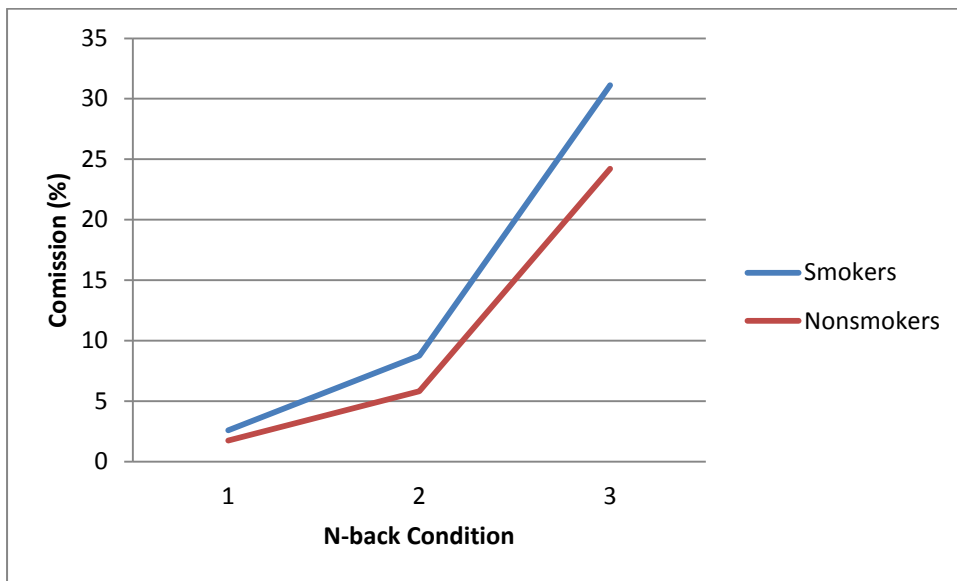
Hypothesis 2 predicted that the experimental group would perform more poorly in terms of commission errors on the Two and Three-back conditions than the control group. Table 4 does show a trend was found if the covariates were not included.

**Commission Score.** The Main effect for group on the rate of commission was insignificant ( $F=0.13$ ,  $p=.72$ ,  $\eta^2<0.01$ ). The Eta squared for the groups had a very small effect size.

On further inspection using post-hoc  $t$ -tests we found no significant differences between smokers and non-smokers during the 1-back ( $t=-1.06$ ,  $p=.29$ ), 2-back ( $t=-1.19$ ,  $p=0.24$ ) and 3-back ( $t=-.41$ ,  $p=.68$ ) tasks. These  $t$ -tests were corrected for age and 1-back baseline and were done as a comparison the  $t$ -tests that excluded age and 1-back baseline.

The Main effect of cognitive load was significant ( $F=163.66$ ,  $p<0.00$ ,  $\eta^2=0.33$ ). Eta squared indicated a small effect size. Post-hoc  $t$ -tests, we found that, regardless of group status (e.g. smokers or non-smokers) all participants had significantly lower omission rate on the 1-back compared to the 2-back ( $t=-6.67$ ,  $p<0.01$ ). All participants were also significantly more accurate during the 2-back versus the 3-back ( $t=-11.69$ ,  $p<0.01$ ). Finally, all participants were significantly more accurate during the 1-back compared to the 3-back ( $t=-14.69$ ,  $p<0.01$ ).

The interaction effect between group and cognitive load was insignificant ( $F=2.15$ ,  $p=0.12$ ,  $\eta^2<0.01$ ). The Eta squared for the interaction indicated a very small effect size. However, there was a trend for higher commission errors ( $t=-1.84$ ,  $p<0.076$ ) during the 1-back level in smokers compared to non-smokers if the statistic was not adjusted for age and 1-back condition. Figure 2 shows a representation of the effect of cognitive load on the n-back task. This figure shows that commission errors increase for smokers and non-smokers when task difficulty increases.



*Figure 2.* Increase in commission errors by  $n$ -back cognitive load condition. X-axis= $n$ -back task difficulty. Y-axis=Commission errors as a percent.

Summarizing the effects on error rate reveals a consistent pattern of a significant main effect for cognitive load; however both group and the interaction effects were insignificant. Although, there is some indication that in the smoking group, omission errors are higher during the easier working memory level, whereas a possible trend for higher commission errors was observed during the more difficult 3-back working memory task compared to the healthy controls.

Table 2

*Post-hoc Independent T-tests of Omission and Commission Rate (N=47)*

Variables	Group				<i>t</i>	<i>p</i>	ESE		
	Smoking n=22		Control n=25						
One-Back									
Omission	7.69	(4.74)	3.64	(1.78)	-3.78	-1.74 <sup>a</sup>	.001***	.09 <sup>a</sup>	1.19
Commission	2.58	(2.84)	1.73	(1.18)	-1.36	-1.06 <sup>a</sup>	.18	.29 <sup>a</sup>	0.41
Two-Back									
Omission	14.44	(12.25)	12.28	(7.90)	-0.73	2.08 <sup>a</sup>	.47	.04 <sup>a</sup>	0.22
Commission	8.76	(6.97)	5.80	(4.57)	-1.69	-1.20 <sup>a</sup>	.09	.24 <sup>a</sup>	0.52
Three-Back									
Omission	28.88	(17.84)	26.60	(14.19)	-0.49	1.79 <sup>a</sup>	.63	.08 <sup>a</sup>	0.15
Commission	31.13	(16.18)	24.20	(7.66)	-1.84	-0.41 <sup>a</sup>	.08	.68 <sup>a</sup>	0.57

*Note.* Data in the table are means, with standard deviations in brackets. All probability scores are compared to a threshold that is Bonferroni-corrected for multiple comparisons ( $.05/6 = .008$ ). ESE = effect size estimate (in this case, Cohen's *d*),

<sup>a</sup>Age and 1-back baseline corrected *t*-statistic and *p*-value .

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

### Relationships between the Measures

To further investigate the relationship of the measures above, bivariate correlations and a multiple regression analysis were done to see the relationship between the variables.

*Hypothesis 3.* Nicotine consumers did display an inverse relationship between self-regulation and impulsivity scores. Non-smokers displayed the inverse of this relationship in that they were less impulsive with higher self-regulation scores.

To investigate the third Hypothesis we thus ran a correlation between the BIS and SRQ scores and found a Pearson correlation of  $-.731(p < 0.01)$  that was significant and displays an inverse relationship between impulsivity and self-regulation.

The Y-BOCS measure had a strong negative correlation with the SRQ and had a medium positive correlation with the BIS (see Table 3). This means that higher scores on impulsivity and lower scores on self-regulation could be seen to establish OCD symptomology.



Table 3.  
*BIS & SRQ Correlations (N = 47)*

	BIS	SRQ
Y-BOCS	$r=0.37$ $p < 0.01$	$r= -0.61$ $p < 0.01$

*Note.* Correlations are significant at the .01 level.

The comorbidity of OCD and depression was confirmed by our analysis. The total scores on the HADS and YBOCS had a positive Pearson correlation of .692( $p < .01$ ). This was a significant correlation showing that higher scores on both measures correlated with each other.

*Hypothesis 4.* This hypothesis was given support by the bivariate correlation and the regression analysis. The correlations were run twice, once within the smoking group only and once with the full sample of participants. Within the smoking group nicotine dependence severity did not show a correlation with OCD severity. Across all participants however the correlation was of medium strength.

For the Fourth Hypothesis I needed to determine if smoking severity had an impact on OCD severity. Firstly, the correlation was conducted with all participants, which showed a medium positive correlation ( $r=.44$ ,  $p < .002$ ) as above in Table 4. This in turn could have been biased to a stronger correlation as half the sample participants had a score of zero (thus overly strengthening the relationship). I thus reran the bivariate only within the experimental group ( $n=22$ ) in order to see if this correlation could be an artefact of the control group. The correlation was no longer significant ( $r = .17$ ,  $p=.45$ ).

Table 4.  
*Correlations with nicotine dependence (N=47)*

	BIS	SRQ	HADS	Y-BOCS
FTND	$r=0.40$ $p < 0.01$	$r= -0.63$ $p < 0.01$	$r=0.49$ $p < 0.01$	$r=0.44$ $p < 0.01$

*Note.* Correlations are significant at the .01 level.

Nicotine dependency for the sample participants ( $n=47$ ) did show medium significant positive correlations with the scores on the BIS, HADS, and Y-BOCS. A strong inverse correlation was shown between nicotine dependency and SRQ. As seen in Table 3.

**OCD as a Predictive Factor for Nicotine Addiction**

In order to show which factors were most predictive of nicotine addiction a linear regression was run with those variables that most significantly correlated in the bivariate correlation analyses. These were SRQ, OCD and HADS, and I examined how these variables predicted performance on nicotine severity only, because no measures correlated with any scores of working memory performance. The best fit model was a stepwise regression with only SRQ predicting FTND.

Stepwise and hierarchical regression models are the most used in statistical analyses; however, hierarchical models assume that certain values hold predominance over others and this could not be assumed in the variables examined here ( Babyak, 2004).

The SRQ accounted for 44% of the variance. Model 1 was significant ( $F=10.59$ ,  $p=.02$ ,  $\beta=0.44$ ) as seen in Table 5. The intention was to show that the Y-BOCS measure is capable of predicting the FTND measure, as this would mean OCD was predictive of nicotine dependence severity. Regardless of the variables that we entered into the regression, the SRQ gave the most predictive value and the other variables were excluded as their coefficients became insignificant when entered with the SRQ. Table 5 is the Model Summary, in which I investigated the relation of the SRQ variable.

Table 5

*Model Summary of Linear Regressions Predicting Smoking Severity (N = 47)*

Model	R Square		Std. Error	R Square	F change		Sig F.	$\beta$
Summary	R	R Square	of the Estimate	Change	F change	Change	Change	
Model 1	.44	.19	1.75	1.91	10.59	.02*	.44	
Model 2	.63	.40	1.50	.40	30.15	.01***	-.63	
Model 3	.64	.41	1.51	.41	14.99	.01***	-.58	

*Note.* Model 1=Y-BOCS→FTND, Model 2=SRQ→FTND, Model 3= Y-BOCS & SRQ→FTND.  $\beta$  =Beta.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

Rather the output generated by SPSS suggested that SRQ was mediating the relationship between OCD tendencies and nicotine dependence (see Table 6 for the Coefficients of each

Model). Sobel's test ( $t=5.11$ ,  $SE=0.20$ ,  $p<0.01$ ) showed that the indirect effect of the independent variable(Y-BOCS) on the dependent variable (FTND) via the mediator (SRQ) is significantly different from zero. Therefore, it can be suggested that the predictive value of Y-BOCS on the FTND score was being mediated by the score on SRQ.

Table 6

*Coefficients of Model 1 through 3 Supporting Mediation*

Coefficients	Unstandardized Coefficients		$\beta$	$t$	$p$
	$B$	Standard Error			
Model 1	0.13	0.04	0.44	3.26	0.01***
Model 2	-1.92	0.37	-0.61	-5.16	0.01***
Model 3					
Y-BOCS	0.02	0.04	0.08	0.55	0.59
SRQ	-0.05	0.01	-0.58	-3.99	0.01***

*Note.* Coefficients of the mediation of SRQ. SRQ= Self-Regulation Questionnaire. Y-BOCS=Yale-Brown Obsessive Compulsive Scale.  $\beta$ = Beta.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

### Discussion

Overall the results I have suggest self-medication with nicotine may help to lower ruminations in those with obsessive compulsive disorder (OCD) traits. This is based on the findings on working memory performance of my experimental group solely. However I'd like to focus more on the non-adjusted scores (age & 1-back baseline) as these are comparative of the literatures associations.

### Working Memory Performance

I would like to group the Hypothesis 1-2 together for the purpose of the discussion. Hypothesis 1 stated that smokers will have more omission errors on the lower task level, while Hypothesis 2 stated that smokers will have more commission errors on the higher task levels in comparison to the healthy controls. The first step therefore is to look at the effect sizes in relation to the n-back conditions, in which we see a reduction in the effect size as the cognitive load increases. This can be suggestive of two things; either a ceiling effect for the

ANCOVA's has occurred in that the cognitive load of the task is too high for both groups and therefore the independent variable (group membership: smokers versus non-smokers) has no effect on the dependent variable. Therefore, the effect of smoking may only be apparent in conditions of low cognitive load, where deficits in the smoking group (e.g. higher omission errors) are easier to detect. Alternatively, it could be that the smoking groups are usually more proficient in working memory, but withdrawal effects from acute nicotine abstinence might impinge on their nicotine-induced proficiency.

**Hypothesis 1.** On the 1-back condition the abstaining smokers performed worse than the control group. The 1-back condition has the lowest cognitive load and so the smokers who are currently abstaining may ruminate more on their craving for nicotine. Their attention may be being drawn away from the task-relevant stimuli (targets during the n-back task), leading to higher rates of mistakes of omission. Greenstein et al. (2010) found that nicotine consumption in both smokers and non-smokers decreases the omission rates for both groups. This supports the view that smokers are able to perform better than the non-smokers under the influence of nicotine; however the lack of nicotine in the case of abstinence may have a detrimental influence on working memory (e.g. increasing the amount of omission errors). The withdrawal-symptoms of nicotine are likely associated with an increase in omission errors, as suggested by (Ernst, et al., 2001), which may suggest that performance on a working memory task can be improved by nicotine satiation in those who smoke.

**Hypothesis 2.** The increased commission errors for smokers on higher cognitive loads was already supported by the literature (Dawkins, Powell, West, Powell, & Pickering, 2007; Shin et al., 2006). This change was only a trend in our data, but increasing the sample size may benefit the rather low effect size for the 3-back commission errors. Smokers may become inflexible in their responses to the targets as they are having difficulty with set-shifting, that is, failing to respond to the non-targets. This suggests that they are incapable of inhibiting response sets, which may be associated with the neural process of rumination. The increase in commission errors held for both groups, which may solely represent that the task-difficulty had increased. The difficulty of the 3-back task may overload the working memory capacity, particularly in those who are also influenced by obsessive-compulsive cognitions.

Additionally, an interesting aspect is that the type of error committed changes with cognitive load. The task difficulty might be responsible for this in that the smokers need to direct their full attention to the higher task condition, the cognitive load of the task thus

decreasing the available time for ruminations about their craving. Instead, however they are making mistakes based on their impulsive nature.

**Problems for Working Memory Conclusions.** Although the data does seem to show support for my hypothesis, they lack one consideration. The smoking group was abstaining for a 24hour period and would have experienced significant physical withdrawal-symptoms (e.g. shaking, increased heart rate, perspiration), that may increase sensory disturbance to lower load levels, and impulsivity in higher load levels. Therefore, one must consider that the findings could be attributable to nicotine withdrawal rather than nicotine consumption. For future research a smoking group should be established to compare satiated and abstaining smoker performance to test this notion. Greenstein and Kassel (2009) found that satiated smokers performed worse on a verbal working memory task than abstaining smokers. This would be supportive of an inverted U-relationship between working memory performance and dopamine levels in the brain (Robbins, 2005; Williams & Castner, 2006). This relationship suggests that excessive dopamine levels in the brain decrease working memory for satiated smokers. The deficit in performance for smokers may thus be conceptualised as a proactive interference with new information by the memory of the previous information. This study would be supportive of such a relationship in that smokers did perform similarly on the n-back task in comparison to non-smokers, meaning that dopamine is not creating a proactive interference.

Hypothesis 3 corroborates the present literature that nicotine addiction correlates with impulsive behaviour (Brody, 2006). found the prefrontal cortex, left anterior cingulate, and right cerebellum to be degraded in smokers in terms of the lower grey matter densities and less volume of their cortical grey matter in comparison to the healthy controls. The impulsive behaviour of smokers may thus be a result of decreased influence of the central executive over the other modalities. The prefrontal and anterior cingulate were both shown to be important for the process of inhibition (A. R. Aron et al., 2004; Hinson, Jameson, & Whitney, 2003; Leung & Cai, 2007). The impulsive nature of smokers may therefore be a product of working memory dysfunction, the lack of inhibitive controls thus allow for impulsive behaviour by not regulating them properly. The central executive dysfunction hence may be a cause for the self-regulation difficulties experienced in nicotine addiction, which further increases the addiction as it may be difficult to control the addiction to nicotine.

Hypothesis 4 was not supported in terms of the severity relationship. Smoking severity was not correlated with OCD tendencies. However there was a higher prevalence of OCD tendencies in nicotine addiction. This would be against the suggestion by Bejerot et al. (2000) that the rigid personality of OCD persons is more likely to protect them from peer pressure that may lead to such an addiction. Furthermore, smoking was shown to correlate with the HADS, a measure of anxiety and depression, which are often comorbid with OCD symptomology (Chettoum et al., 2012; Djordjevic, Fan, Ferguson, & Hoffmann, 1995). This confirms that nicotine addiction is more prevalent in the clinical population. For instance, nicotine consumers with a comorbid psychiatric disorder smoke 34.2% of all cigarettes in the United States in 2001 (Grant, Hasin, Chou, Stinson, & Dawson, 2004). However, OCD tendencies were not predictive of smoking severity, as the predictive value of the OCD measure was found to be mediated by self-regulation.

This paper was not able to directly address the question of whether working memory deficits from nicotine consumption exist. This deficit, however is supported in that both smoking and OCD correlated with SRQ. Hence measures of self-regulation could serve as a determinant for working memory deficit by providing the link between OCD and nicotine addiction. The literature supports a self-regulation difficulty in both disorders (Dawkins, Powell, West, Powell, & Pickering, 2007; Djordjevic, Fan, Ferguson, & Hoffmann, 1995; Exner, Martin, & Rief, 2009; Meiran, Diamond, Toder, & Nemets, 2011). The self-regulation deficit in both disorders entails a deficit in top-down processing by deficits in inhibition modalities. Therefore, any suggestion for self-medication by nicotine for OCD persons should take into consideration their ability to self-regulate prior to adding the negative effects of nicotine consumption that further decrease the capacity to regulate one's own behaviour.

### **Strengths & Limitations**

My study seems to corroborate the literature and its eta findings, although my findings are not as strong as those presented in other studies, which suggests that my study has a power issue. This can be addressed by increasing the number of participants in future studies. However, most of the literature that has looked at working memory performance has had sample sizes smaller than my own study (around 15), but these generally used functional magnetic resonance imaging to bolster their findings. Additionally, I examined smokers who

I asked to abstain from nicotine consumption for 24 hours, to examine working memory independent of acute nicotine effects, but the withdrawal symptoms may weaken the measures of being a smoker on cognitive performance. Furthermore, smokers may have been distracted by their cravings such that they were not able to attend effectively to the targets.

The score of the experimental group was variable from 1-6 on the FTND, and so the range of smoking severity was quite high. It would have been better to have a more consistent level of smoking dependency (mainly higher) as this would be likely to show more of an effect. The venue may also have contributed to a difference in performance. 19 smokers were tested in a room that was much smaller than the original venue. This closer proximity to the researchers may have had a negative effect on task-performance due to stress or a positive effect as they would be more aware of being watched.

A particular concern is the Y-BOCS measure, as the compulsion and obsession questions might have reflected their need for nicotine instead. For instance, the question “How much time do you spend on performing your compulsive behaviours?” can be interpreted by a smoker as how much time they spend smoking. The Y-BOCS in this manner has many questions that could be interpreted within a smoking context, therefore artificially inflating their answers on the Y-BOCS from that of a satiated smoker.

The data may be confounded by comorbid disorders such as anxiety and depression, and so it would be advisable to be more stringent on these adhering to a subclinical population. Recruitment for the smoking group was difficult, and thus I couldn't be as stringent.

Furthermore, the n-back task is fairly difficult to grasp until actually doing the task. The 1 and 2 back condition may thus give a worse representation of actual working memory performance than participant's actual capability as the task was being learnt for the first time. Training can be done, however the practice effects on the n-back task are well known.

### **Implications and Future Research**

The main implication from my research is that self-medication for OCD persons with nicotine may in the long term increase OCD symptomology. Therefore, a careful consideration for the effects of nicotine consumption needs to be made prior to this type of treatment. In particular, the rumination aspect is of concern. If there is a working memory deficit from nicotine consumption then this could further weaken the working memory

capacity to control OCD symptoms. The first step is thus a determination of the basis of the working memory deficit in nicotine addiction which may either be caused by the deterioration in the inhibition centres in the brain (Brody, 2006) or proactive interference caused by excess dopamine to the frontal-circuitry (Greenstein & Kassel, 2009). For future research it would be advisable to have both a satiated and abstaining compared with each other against a non-smoking cohort. Furthermore the nicotine intake needs to be regulated as a significant difference in task-related performance depending on a 2mg or 4mg dose of nicotine. A 4mg dose of nicotine showed significant increased vigilance, while a 2mg dose was non-significant (Parrott & Winder, 1989). This is something that was not regulated in the (Greenstein & Kassel, 2009) paper that may have had an influence on their results if the satiated participants had too much nicotine it may have had adverse effects rather than beneficial effects on working memory performance (Robbins, 2005; Williams & Castner, 2006).

To summarize, this study did not find a working memory deficit in nicotine addiction. However, the study did find some comparative results to the literature in regards to omission and commission errors in smokers on the lower and higher cognitive load level respectively. These comparative results suggest that there is a connection between the working memory performance of nicotine addiction and OCD on the basis of a lack in self-regulation. Hence these disorders both exhibit failures by the central executive to inhibit an action that is driven by impulsivity. If nicotine is the cause of inhibition difficulties in smokers, then nicotine can further transfer these to OCD patients that are being treated with nicotine. Nicotine treatment is therefore capable of increasing OCD symptomology by further damaging defunct inhibition control.



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## Appendix A

## Ethical Approval

 <b>UNIVERSITY OF CAPE TOWN</b> IYUNIVESITHI YASEKAPA - UNIVERSITEIT VAN KAAPSTAD	HUMAN RESEARCH ETHICS COMMITTEE 27 MAY 2014 HEALTH SCIENCES FACULTY UNIVERSITY OF CAPE TOWN	<b>FACULTY OF HEALTH SCIENCES</b> Human Research Ethics Committee
	<b>Form FHS006: Protocol Amendment</b>	

<b>HREC office use only (FWA00001637; IRB00001938)</b>		
<input checked="" type="checkbox"/> Approved	<input checked="" type="checkbox"/> Type of review: Expedited	<input type="checkbox"/> Full committee
This serves as notification that all changes and documentation described below are approved.		
Signature Chairperson of the HREC		Date <u>27/05/14</u>

**Note:** All amendments should include a Synopsis justifying the changes for the amendment ([please see notice dated 23 April 2012](#))

**Principal Investigator to complete the following:****1. Protocol information**

Date form submitted	27/05/2014	
HREC REF Number	113/2014	
Protocol title	Cognitive training in obsessive compulsive disorder (OCD) patients	
Protocol number (if applicable)		
Principal Investigator	Samantha Brooks	
Department / Office Internal Mail Address	Psychiatry and Mental Health/Health Sciences: drsamanthabrooks@gmail.com	
1.1 Is this a major or a minor amendment? (see <a href="#">FHS006hlp</a> )	<input type="checkbox"/> Major	<input checked="" type="checkbox"/> Minor
1.2 Does this protocol receive US Federal funding?	<input type="checkbox"/> Yes	<input checked="" type="checkbox"/> No
1.3 If the amendment is a major amendment and receives US Federal Funding, does the amendment require full committee approval?	<input type="checkbox"/> Yes	<input type="checkbox"/> No

**2. List of Proposed Amendments with Revised Version Numbers and Dates**

<p><b>Please itemise on the page below, all amendments with revised version numbers and dates, which need approval.</b></p> <p>This page will be detached, signed and returned to the PI as notification of approval. Please add extra pages if necessary.</p> <p>Measures to be added:          -Fagerstrom Test for Nicotine Dependence          -Obsessive Compulsive Inventory-Revised          -Self-Report Version of the Y-BOCS</p> <p>Participants:          -Increase of the control group by 60 participants</p> <p>The amendment is done to perform baseline measurements on working memory to covariates.</p>
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Appendix C

Hospital Anxiety and Depression Scale

**Hospital Anxiety and Depression Questionnaire (HADS)**

**Directions:** Please tick the box that best represents you.

I feel tense or wound up

- most of the time
- a lot of the time
- Occasionally
- not at all

I still enjoy the things I used to enjoy

- definitely as much
- not quite as much
- only a little
- hardly at all

I get a sort of frightened feeling as if  
something awful is about to happen

- quite badly
- not too badly
- a little
- not at all

I can laugh and see the funny side of things

- as much as I always
- not quite so much
- definitely not so
- not at all

Worrying thoughts go through my mind

- a great deal of time
- a lot of time
- from time to time
- only occasionally

I feel cheerful	<input type="checkbox"/> not at all <input type="checkbox"/> not often <input type="checkbox"/> sometimes <input type="checkbox"/> a lot
I can sit at ease and feel relaxed	<input type="checkbox"/> definitely <input type="checkbox"/> usually <input type="checkbox"/> not often <input type="checkbox"/> not at all
I feel as if I am slowed down	<input type="checkbox"/> nearly all the time <input type="checkbox"/> very often <input type="checkbox"/> sometimes <input type="checkbox"/> not at all
I get a sort of frightened feeling like butterflies in the stomach	<input type="checkbox"/> not at all <input type="checkbox"/> occasionally <input type="checkbox"/> quite often <input type="checkbox"/> very often
I have lost interest in my appearance	<input type="checkbox"/> definitely <input type="checkbox"/> I don't take so much care as I should <input type="checkbox"/> I may not take quite as much care <input type="checkbox"/> I take just as much care as ever

<p>I feel restless as if I have to be on the move</p>	<input type="checkbox"/> very much <input type="checkbox"/> quite a lot <input type="checkbox"/> not very much <input type="checkbox"/> not at all
<p>I look forward with enjoyment to things</p>	<input type="checkbox"/> as much as ever <input type="checkbox"/> rather less than I used to <input type="checkbox"/> definitely less than before <input type="checkbox"/> hardly at all
<p>I get sudden feelings of panic</p>	<input type="checkbox"/> very often <input type="checkbox"/> quite often <input type="checkbox"/> not often <input type="checkbox"/> not at all
<p>I can enjoy a good book or programme</p>	<input type="checkbox"/> often <input type="checkbox"/> sometimes <input type="checkbox"/> not often <input type="checkbox"/> very seldom

Appendix D

Fagerstrom Test for Nicotine Dependence

<b>Fagerstrom Test</b>			
<b>Please tick (X) one box for each question</b>			
How soon after waking do you smoke your first cigarette?	Within 5 minutes	<input type="checkbox"/>	3
	5-30 minutes	<input type="checkbox"/>	2
	31-60 minutes	<input type="checkbox"/>	1
Do you find it difficult to refrain from smoking in places where it is forbidden? Eg. Church, Library, etc.	Yes	<input type="checkbox"/>	1
	No	<input type="checkbox"/>	0
Which cigarette would you hate to give up?	The first in the morning	<input type="checkbox"/>	1
	Any other	<input type="checkbox"/>	0
How many cigarettes a day do you smoke?	10 or less	<input type="checkbox"/>	0
	11 to 20	<input type="checkbox"/>	1
	21 to 30	<input type="checkbox"/>	2
	31 or more	<input type="checkbox"/>	3
Do you smoke more frequently in the morning?	Yes	<input type="checkbox"/>	1
	No	<input type="checkbox"/>	0
Do you smoke even if you are sick in bed most of the day?	Yes	<input type="checkbox"/>	1
	No	<input type="checkbox"/>	0
<b>Total Score</b>			

## Appendix E

## Self-Report Yale-Brown Obsessive Compulsive Scale

**Y-BOCS**

Answer each question based on the average occurrence of each item over the past week. The first 5 questions relate to obsessive thoughts, the last 5 questions relate to compulsive behaviors.

**Obsessions**

are unwelcome or distressing ideas, thoughts, images or impulses that repeatedly enter your mind. They may seem to occur against your will. They may be repugnant to you, are often senseless, and may not fit your actual personality at all (for example, the recurrent thought or impulse to harm to your children, even though you never would).

**1. How much of your time is occupied by obsessive thoughts?**

- None
- Mild, less than 1hr/day or occasional intrusion
- Moderate, 1 to 3 hrs/day or frequent intrusion
- Severe, greater than 3 and up to 8 hrs/day or very frequent intrusion
- Extreme, greater than 8 hrs/day or near constant intrusion

**2. How much do your obsessive thoughts interfere with functioning in your social, work, or other roles?**

- None
- Mild, slight interference but overall performance not impaired
- Moderate, definite (but manageable) interference



- Severe, causes substantial impairment
- Extreme, incapacitating

**3. How much distress do your obsessive thoughts cause you?**

- None
- Mild
- Moderately disturbing, but still manageable
- Severe, very disturbing
- Extreme, near constant and disabling distress

**4. How much of an effort do you make to resist the obsessive thoughts? How often do you try to disregard or turn your attention away from these thoughts as they enter your mind? (only rate effort made to resist, not success or failure in actually controlling the obsessions)**

- Always make an effort to resist, or don't even need to resist
- Try to resist most of the time
- Make some effort to resist
- Reluctantly yield to all obsessive thoughts
- Completely and willingly yield to all obsessions

**5. How much control do you have over your obsessive thoughts? How successful are you in stopping or diverting your obsessive thinking? Can you dismiss them?**

- Complete control
- Usually able to stop or divert obsessions with some effort and concentration
- Sometimes able
- Little control, rarely able to stop or dismiss, can only divert attention with difficulty
- No control, completely involuntary, rarely able to even momentarily alter obsessive thinking

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### Compulsions

are behaviors or acts that you feel driven to perform, even though you may recognize them as senseless or excessive. At times, you may try to resist doing them, but this may prove difficult. You may experience anxiety that does not diminish until the behavior is completed.

#### **6. How much time do you spend performing compulsive behaviours**

- None
- Less than 1 hour per day or occasional performance of compulsive behaviour
- 1-3 hours per day, or frequent
- 3-8 hours per day, or very often
- More than 8 hours per day or near constant performance (too numerous to count)

#### **7. How much do your compulsive behaviours interfere with your work or social functioning?**

**Is there anything you don't do because of your compulsions?**

- None
- Slight interference, but no impairment
- Definite interference, but manageable
- Substantial interference
- Extreme interference, incapacitating

**8. How anxious would you become if you were prevented from performing your compulsive behaviors?**

- No anxiety
- Only slightly anxious
- Some anxiety, but manageable
- Prominent and disturbing anxiety
- Extreme, incapacitating anxiety

**9. How much of an effort do you make to resist the compulsions thoughts?**

*(only rate effort made to resist, not success or failure in actually controlling the compulsions)*

- Always make an effort to resist, or don't even need to resist
- Try to resist most of the time
- Make some effort to resist
- Reluctantly yield to all compulsions
- Completely and willingly yield to all compulsions

**10. How strong is the drive to perform the compulsive behaviour? How much control do you have over the compulsions?**

- Complete control
- Experiences pressure to perform the behaviour but usually able to exercise voluntary control over it
- Moderate, strong pressure to perform, controls only with difficulty
- Little control, drive very strong, must be carried to completion, can only delay with difficulty
- No control, drive involuntary and overpowering, rarely able to even momentarily delay activity

## 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

## Consent Form

Consent Form to Participate in WM Research**You are asked to participate in a study conducted by:**

Kai Schramm: kaitschramm@live.co.za  
drsamanthabrooks@gmail.com

Dr. Samantha J. Brooks:

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**

The purpose of this study is to examine the incidence of OCD in nicotine consumers and abstainers. The cognitive load required by the n-back Working Memory task can measure discreet variances in Working Memory according to varying degrees of smoking behavior. If these discreet variances between WM can elicit the smoking trends in OCD persons, one may therefore assert to a connection between the ruminative state experienced in nicotine addiction and OCD.

**Procedures**

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

- a) You will be required to participate in a 1.5 hour testing session.
- b) The first part of the testing session will consist of 6 measures that need to be completed. These measures excluding the Trail-making Task (which will be completed on paper), will all be provided as a computer based task that will allow for you to quickly click the preferred answer. These psychometric tests are all short and will not take up much time.
- c) The major constituent will be a computer 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



of the task. Following this will be a 2-back and 3-back task that will both take half an hour of your time. You will receive a short 10 minute break between these two tasks.

- d) After completion of the 3-back task you will need to fill in some basic demographic details. After which 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. The tasks are computer based for the majority, so you might experience some weariness from staring at a computer screen for an extended period of time. You will however be provided with a break to prevent any form of overexertion.

### **Potential Benefits to Subject or Society**

- a) The subject will partake in a WM task that has the capability of improving WM in the form of cognitive training.
- b) Of societal importance is the possibility of a connection between the ruminative processing of OCD and nicotine addiction. This may be an important consideration as nicotine is currently under investigation as a possible treatment for OCD participants. The effects of nicotine on WM circuitry could thus provide input for this matter.

### **Compensation for Participation**

All your required SRPP points for the semester will be taken care of.

### **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:

- a) 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.
- b) Paper-based / computer –based records will only be available to people involved in the study.

**Participation and Withdrawal**

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

If you are interested, please supply us with an email address, so that we can send you information on the outcome of our study once they become available. You are also more than welcome to partake in a Colloquium to be held on the 31<sup>st</sup> of October 2014 that will present the results.

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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 H

## Demographic Questionnaire

Name	
Date of Birth	
Gender	
Ethnicity	
Year of Study	
Medical History	
Drug History	
Current Medications	
Glasses/Hearing aid	
Medical Conditions	
Psychiatric Diagnoses	
Student Number	
Course Code (SRPP)	

## Appendix I

## Frequency Characteristics of Sample

Table I.  
*Frequency Characteristics of Sample (N=47)*

Frequency	Group			
	Smoking (n = 22)		Control (n = 25)	
Sex	Male = 1	Female = 21	Male = 2	Female = 21
Educational Level	3:9:6		0:12:10	
Undergraduate( 1 <sup>st</sup> ,2 <sup>nd</sup> ,3 <sup>rd</sup> )				
Postgraduate(Hons:Ma:Phd)	1:1:2		3:0:0	
Ethnicity	9:2:8:3		12:7:5:11	
White:Black:Coloured:Indian				