Alcohol has a complex effect on the human brain and working memory (Campanella, Maurage, Kornreicha, Verbancka, & Noël 2009). Working memory involves the short-term maintenance, manipulation and storage of information. There are several accounts that explain working memory (Shah & Miyake, 1999). Here, I used a multimodal working memory model as a framework (Logie & Van der Meulen 2009), which dichotomises the visuo-spatial sketchpad into visual and spatial processes. It also has an inner scribe that works on a visual cache and stores dynamic information (processing trajectories of movements and motor actions) and serves spatial rehearsal. Other components include a slave system, otherwise known as the phonological loop and the executive function. The phonological loop operates on inner speech and the executive function controls processes that integrate and regulate the activity of various components through strategy and maintenance processes (Jiang, Makovski, & Mok Shim, 2009; Logie & Van der Meulen, 2009).

The below literature illustrates that alcohol use impairs working memory and shows how executive function, an aspect of working memory, is susceptible to impairment. This is exemplified by a study of 72 moderate alcohol drinkers, who were given alcohol doses, before a visual array and auditory array task, which was adapted from Luck and Vogel’s seminal task (1997). Results revealed alcohol doses had no significant effect on moderate alcohol drinkers’ working memory-holding mechanisms, but impaired their mnemonic and executive strategies, which are needed to retain sequences (Saults, Cowan, Sher, & Moreno, 2007). This suggests that tasks measuring attention and maintenance processes are sensitive to acute alcohol use. Similar findings were found in a study that compared the cognitive profiles of 33 binge drinkers and 49 non-binge drinkers. Binge drinkers were found to have impaired performance in a self-ordered pointing task and Rey verbal auditory learning test. This supports the view that tasks requiring strategy utilisation and the planning of aspects of working memory (Ross, Hanouskova, Giarla, Calhoun, & Tucker, 2007) are susceptible to alcohol induced impairments (Mota et al., 2013). Two larger studies that looked at alcohol users also corroborate these findings. Results revealed that binge drinking impaired performances in self-ordered pointing tasks and backward digit span tasks, while performances in forward digit span tasks remained intact (Parada et al., 2012). When social drinkers were given alcohol doses they were found to have an impaired backward digit span performance (Schweizer et al., 2006). Also, alcohol use was found to predict impaired strategy performance during a task demanding spatial working memory (Piechatzek et al., 2009). These studies indicate that executive function impairments occur as a result of alcohol use, while other aspects of working memory, such as the phonological loop, are not affected.

This notion is supported by another study that looked at a trail making test (TMT) part A&B performance; a set-shifting and visual attention task, in a sample of 92 young adult alcohol users. Their performance in TMT Part B was predicted by alcohol breath, while acute and chronic alcohol use predicted executive function impairments (Day, Celio, Lisman, Johansen, & Spear, 2013). This suggests that set-shifting, a component of executive functions, is
impaired by alcohol use while other aspects of working memory remain intact. These inferences coincide with previous studies that found a similar effect with alcohol use disorder patients (Hanson, Medina, Padula, Tapert, & Brown, 2011) and alcohol dependent patients (Uva et al., 2010) when tested by TMT Part A&B. Also, patients who abstained from alcohol use for 3 weeks were found not to recover their set-shifting abilities (Uva et al., 2010). This suggests that alcohol patients’ set-shifting does not recover through alcohol abstinence.

While questions can be raised about the effects of alcohol on set-shifting, it is clear that once inhibitory control is also introduced in a task, alcohol impairments emerge. This is demonstrated in studies that gave social drinkers alcohol doses, which resulted in impairments in Stroop tasks, go/no go tasks and motor responses (Duka & Townshend, 2004; Gustafson & Kallmen, 1990; Rose & Duka, 2006). However, other findings showed no effect on pro- and anti-saccadic performance, when healthy participants were given alcohol doses (Vorstius, Radach, Lang, & Riccardi, 2008). While questions can be raised on whether social drinking affects inhibitory and set-shifting processes; it is clear that alcohol-related disinhibition does occur with remitted alcoholic patients (Noël et al., 2001) and alcohol use disorder patients (Chao, Meyerhoff, Cardenas, Rothlin, & Weiner, 2003; Tapert et al., 2004). This advocates the view that acute and chronic alcohol use affects different aspects of working memory, in particular executive function.

Another study also found a similar effect in 42 abstinent alcoholic patients, who were tested by a CANTAB battery. Also, despite patients abstaining from alcohol (on average 1.1 years), they continued to have impairments in their attentional control, set-shifting and spatial working memory strategies in contrast to healthy participants (Kopera, Wojnar, Brower, Glass, & Nowosad, 2012). This suggests that alcohol use, in a clinical and non-clinical sample, can result in impaired executive function and spatial working memory. Despite these performance differences, the authors suggested that prolonged alcohol abstinence leads to cognitive recovery (Kopera, et al., 2012).

A 10-year follow-up study (with a sample of remitted users and alcohol use disorder and substance use patients) disagreed with the idea that alcohol abstinence results in cognitive recovery. The study used a battery of cognitive tests that assessed intelligent quotient and working memory globally. When controlling for age and education, alcohol use predicted verbal working memory and executive function impairments, while remitted alcohol users continued to present with working memory impairments (Hanson et al., 2011). This suggests that chronic alcohol use, in a clinical sample, has a deleterious effect on global working memory and cognitive recovery may not occur. This is corroborated with a meta-analysis, which also found similar findings (Stavro, Pelletier, & Potvin, 2012). While it is established that cognitive recovery does not completely occur in a clinical sample, there are no studies, to my knowledge, that investigate this in a healthy population.

While the above studies show that working memory impairments occur as a result of chronic and acute alcohol use, these conclusions are made with samples of heavy alcohol users, participants who were given alcohol doses and clinical populations. Despite this extensive research in the aforementioned populations, there are few studies that consider alcohol use and their effects on working memory in healthy adults (Piechatzek et al., 2009). Further to this, to my knowledge, there are no studies that consider cognitive recovery from alcohol use in healthy adults and look at the discrete cognitive demands that are used during a complex span procedure through statistical analysis. Ergo, it is difficult to surmise what modality of working memory is impaired. In order to bridge this literature gap, I used a sample of healthy adults and observed their alcohol consumption behaviours and working memory. To assess the effects of alcohol use on working memory, age was considered as a predictive variable, which affects working memory (Park, Carp, Hebrank, Park, & Polk, 2010).

Besides considering the effects of age on working memory, I also looked at the specificity of working memory impairments, which are incurred from alcohol use. In order to gauge the specificity of working memory impairments from alcohol use, a battery of cognitive tests was used that is sensitive to working memory globally. This was considered by using a forward digit span task (a phonological measure), backward digit span task (a verbal executive task), visual pattern test (a visual working memory task), Corsi block test, (a spatial task), trail making test Part A&B (a visual attention and set shifting task) and Stroop (an inhibitory task). Similarly to Thompson et al., (2006), who examined the specificity of visuo-spatial and executive impairments, I looked to discern the specificity of working memory impairments from alcohol use through statistical analysis. Therefore, in the present study, a hierarchical regression method was used, which accounts for the moderator effect of age and the use of working memory resources used during a complex span procedure.

I firstly hypothesise that the recent use of alcohol use will predict impaired executive function. Secondly, I hypothesise the years of alcohol use will predict impaired executive function. I also retested the “continuity hypothesis” that proposes a continuum of alcohol consumption effects on the brain and advocates mild to moderate cognitive impairments in moderate to heavy alcohol drinkers (Vollstadt-Klein et al., 2010).

Method
Participants
Two researchers collected a prospective sample of 157 healthy participants. Participants were selected regardless of their alcohol, cigarette and illicit drug consumption. Cigarette smoking, illicit drug consumption and participants who abstained from alcohol for more than 30 days, defined as not being short term abstainers (Stavro et al., 2012), were screened for, resulting in a sample of 70 males and 30 females ($N = 100$, mean age 23.58 and standard deviation 6.67). Participants reported as being non-drinkers, moderate drinkers, intermediate drinkers and heavy drinkers, as presented in Table 1 below.
Table 1: Participants’ Alcohol Drinking Profile.

Note. * Non-drinkers (0 alcohol units per week), moderate drinkers (women: 1–14 alcohol units per week and men: 1–21 alcohol units per week) (International Centre for Alcohol Policies, 2010; National Institute for Health Clinical Excellence, 2010; World Health Organisation, 2011), intermediate drinkers (women: 15–20 units per week and men: 22–27 units per week) and heavy drinkers (women: more than 21 units per week and men: more than 28 units per week) (Baliunas et al., 2009; Batty, Lewars, Emslie, Gale, & Hunt, 2009; Di Castelnuovo et al., 2006; Rehm et al., 2010).

|                | Non-Drinkers | Moderate Drinkers | Intermediate Drinkers | Heavy Drinkers |
|----------------|--------------|-------------------|-----------------------|---------------|
| Female         | 10           | 14                | 3                     | 3             |
| Male           | 26           | 33                | 4                     | 7             |
| Total          | 36           | 47                | 7                     | 10            |

Materials

Participants completed a randomised and computerised battery of cognitive tests, which were sensitive to visual, spatial, phonological and executive function. The battery included; forward digit span (FDS), backward digit span (BDS), visual patterns test (VPT), Corsi block task (CBT), non-verbal trails making test (NvTMT) and Stroop task.

FDS assesses the phonological loop (Baddeley, 1986). The task has 8 levels and each level has 3 trials of number sequences. In each trial, the researcher verbally produces the numbers from the sequence. The first level has two digits. Participants need to recall, correctly, the number sequence in the same order in at least two of the three trials in a level, in order to proceed to the next level, which increases by one more digit. Participants are prompted to recall the sequence by the researcher. The task ceases once all eight levels are completed or when the participant is unable to correctly answer at least two of the three trials in a level.

BDS, a verbal executive task (Baddeley, 2003), has eight levels and each level has three trials of number sequences. In each trial, the numbers from a sequence appear individually (on a time loop of three seconds) on a computer screen. The first level has two digits. When prompted, participants need to recall the number sequence in reverse order by entering the digits into the computer. Participants need to answer at least two of the three trials in a level correctly in order to proceed to the next level, which has an additional digit in each increasing level. The task ceases once all eight levels are completed, or when the participant is unable to correctly answer at least two of the three trials in a level.

VPT measures visual working memory (Della Sala, Gray, Baddeley, Allamano, & Wilson, 1999). On a computer screen, participants are presented with a 2 x 2 checkerboard, which has two filled-in cells. The pattern is shown to them for three seconds. Participants are then asked to recall the patterns, by clicking the cells with a computer mouse, which they recall to be shaded-in cells. To proceed to the next level, participants need to answer two of the three trials correctly, which results in increasing the complexity of the checkerboard. The task ceases once 14 levels are successfully completed, or when the participant is unable to correctly answer at least two of the three trials in a level.

CBT measures spatial executive memory (Della Sala, et al., 1999). On a computer screen, participants are presented with a 2x2 checkerboard, which has two filled-in cells that appear in a sequential order. The spatial sequence is presented at a rate of one stimulus per second with a three second delay prior to the recall phase. Participants are then requested to recall the sequential pattern in the correct order. They need to correctly complete two of three trials in a level in order to proceed to the next level. The task ceases once 14 levels are completed successfully, or when the participant is unable to correctly answer at least two of the three trials in a level.

NvTMT measures set-shifting and visual attention (Sánchez-Cubillo et al., 2009; Day et al., 2013). Participants need to complete three trials as quickly as possible. The first trial asks participants to click on (with a computer mouse) a series of numbers in ascending order, which are scrambled on the screen. In the second trial, participants are asked to click a series of letters in ascending order, which are also scrambled on the screen. In the third trial, participants are then asked to select letters and numbers in ascending alternating order (e.g., A, 1, B, 2, C, 3, D, 4, E, 5, F, 6) as precisely and quickly as possible.

The Stroop task measures inhibition (Stroop, 1935). On a computer screen, participants are randomly shown 43 stimuli, which are congruent (16 stimuli), incongruent (11 stimuli), or neutral (16 stimuli). As quickly as possible, participants are asked to identify (by using a keyboard) whether the stimulus is congruent, incongruent or neutral.

The recreational drug use questionnaire is a paper-based questionnaire (Buchanan et al., 2010; Heffernan, Ling, & Bartholomew, 2004), which examines the respondents’ alcohol use over the past week, when they last consumed alcohol and for how many years they have drank alcohol. The questionnaire also examined the participants’ cigarette and recreational drug use.

Procedure

This study received full ethical approval by Northumbria University’s Psychology department. Before the study began it was ensured that participants were given sufficient information about the study and full consent from participants had been obtained. Participants were also informed about their right to withdraw from the study. Two researchers individually tested participants in either public buildings or within university premises.

Participants were instructed to carry out a self-report alcohol consumption behaviour, cigarette and substance use scale, which lasted five minutes. It required a tick response in accordance to a Likert scale and an ordinal scale. After completing the questionnaire, participants completed a battery of computerised and randomised cognitive tasks (FDS; BDS; VPT; CBT; NvTMT; Stroop) that
lasted 50 minutes. Once the participant completed the cognitive tasks and questionnaire, they were debriefed and were thanked for their participation in the study. Once the data had been collected, the data was collated together in a sample pool. The researchers then extracted participants who met the study’s inclusion criteria from the sample pool.

**Data Analysis**

An omnibus of hierarchical linear regression models (SPSS v20) were used to determine the predictor effect of alcohol units per week, years of alcohol use and days of alcohol abstinence on criterion variables: BDS, FDS, CBT, Stroop and NvTMT part A&B. In order to account for the mediating effect of age on working memory, age was included in the first block. During the entry of age in the first block for CBT, also BDS and VPT were entered into the first block. This allowed the model to account for executive function and visual working memory. For the first block entry for VPT, CBT and BDS were entered. By entering the above-mentioned cognitive tasks in CBT and VPT models, this helps to discern the alcohol effects on aspects of working memory; a similar approach was used by Thompson et al. (2006).

In the second block, alcohol units per week, years of alcohol use and days of alcohol abstinence were entered. The decision to enter these mediators, with this approach, was based on theory (Logie & Van der Meulen, 2009), accounting for known mediators for working memory (Day et al., 2013; Hanson et al., 2011; Kopera et al., 2012) and how tasks performance correlated with each other (see Table 2). When analysing participants’ Stroop and NvTMT reaction time scores, median scores were firstly obtained and then a current ratio was calculated (Incongruent Stroop score divided by Congruent Stroop score). Also, participants’ average span scores for BDS, FDS, CBT and VPT were computed.

**Results**

**Descriptive Statistics**

A total of 100 participants were included in the analysis and the descriptive statistics of their working memory performance can be seen in below in Table 3.

**Table 2:** Pearson’s R Correlations for BDS, FDS, CBT average span and Stroop, NvTMT cost.

|       | BDS  | FDS  | CBT  | VPT  | Stroop |
|-------|------|------|------|------|--------|
| BDS   | .133 | 1    |      |      |        |
| CBT   | .317 | .044 | 1    |      |        |
| VPT   | .448 | .108 | .398 | 1    |        |
| Stroop| −.031| .118 | −.043| −.159| 1      |
| NvTMT | −.263| .080 | −.096| −.259| .270   |

Note: * Correlation is significant at $\alpha = .05$ level (2-tailed).

**Table 3:** Working Memory Task Performance.

|                   | Mean   | Std.   |
|-------------------|--------|--------|
| BDS               | 7.328  | 1.548  |
| FDS               | 5.473  | 1.898  |
| CBT               | 5.356  | 1.270  |
| VPT               | 8.112  | 1.517  |
| NvTMT             | 2.059  | .560   |
| Stroop            | 1.219  | .188   |

**Table 4:** Pearson’s R Correlations for Alcohol consumption behaviours.

|                   | Years of Alcohol Use | Alcohol Abstinence | Age |
|-------------------|----------------------|--------------------|-----|
| Alcohol Units per Week | .375* | .026 | .120 |
| Years of Alcohol Use | 1      | .099 | .629* |
| Alcohol Abstinence  | 1      | −.105|     |

**Correlational Analyses**

Associations between years of alcohol use, alcohol abstinence, alcohol units consumed per week and age are shown in the Table 4 below. The units of alcohol consumed per week and the years of alcohol use were found to have a statistically significant moderate positive correlation. The years of alcohol use and age of participant were found to have a statistically significant strong positive correlation.

The years of alcohol use and alcohol abstinence were found to have a statistically insignificant small positive correlation. The units of alcohol consumed per week had a statistically insignificant small positive correlation with alcohol abstinence and age. The participants’ age and alcohol abstinence was also found to have a statistically insignificant small negative correlation.

**Hierarchical Regression Analyses**

Hierarchical linear regression models were carried out to analyse the association between the various mediators and working memory. An overview of the models and a more detailed summary of the mediators entered in the models are presented below (see Table 5).

Using a hierarchical linear regression method, it was found that the entered variables in the first and second entry explained a significant amount of the variance in various working memory modalities (BDS: first entry, $F(1, 96) = 6.764$, $p = .011$ and second entry $F(4, 93) = 4.184$, $p = .004$; CBT: first entry, $F(3, 94) = 7.256$, $p < .001$ & second entry $F(6, 91) = 4.741$, $p < .001$; VPT: first entry, $F(3, 94) = 12.060$, $p < .001$ & second entry $F(6, 91) = 6.974$, $p < .001$; FDS: second
entry $F(4, 94) = 3.351, p = .013$; NvTMT: second entry $F(4, 94) = 4.275, p = .003$.

However, the entered variables in the first entry for the FDS and NvTMT model did not explain a significant amount of the variance (FDS: first entry, $F(1, 97) = 3.847, p = .053$; NvTMT: first entry, $F(1, 97) = 4.39, p = .050$). Further to this, the entered variables in the first and second entry for Stroop models did not explain a significant amount of the variance (first entry $F(1, 97) = .439, p = .509$ and second entry $F(4, 94) = 1.003, p = .410$).

**Model Summary**

**BDS.** As shown in Table 5, during a first entry, age significantly predicted BDS ($\beta = -.257, p = .018$). During a second entry, age and alcohol abstinence significantly predicted BDS ($\beta = -.294, p = .024$; $\beta = .257, p = .011$). Also during a second entry, alcohol units consumed per week and years of alcohol use did not significantly predict BDS ($\beta = .069, p = .507$; $\beta = .089, p = .517$).

**VPT.** During a first entry, age, BDS and CBT significantly predict VPT ($\beta = -.064, p = .021$; $\beta = .343, p < .001$; $\beta = -.064, p = .021$). During a second entry, alcohol abstinence, BDS and CBT significantly predicted VPT ($\beta = -.205, p = .031$; $\beta = .392, p < .001$; $\beta = .307, p = .002$). While during a second entry, age, alcohol units consumed per week and years of alcohol use did not significantly predict VPT ($\beta = -.096, p = .481$; $\beta = -.046, p = .626$; $\beta = .052, p = .678$).

**CBT.** During a first entry, VPT significantly predicted CBT ($\beta = .314, p < .003$), while age and BDS did not significantly predict CBT ($\beta = -.067, p = .492$; $\beta = .161, p = .133$).

| Tasks   | Model Summary | Standardised Coefficients |
|---------|---------------|---------------------------|
|         | Entry | $R^2$ (adj) | SE  | $\beta$ | $p$  |
| BDS     | 1     | .056        | 1.853 | -.257   | .018*  |
|         | Age   |             |       |         |       |
| BDS     | 2     | .116        | 1.793 | -.294   | .024*  |
|         | Age   |             |       |         |       |
|         | Alcohol Units per week |             |       | .069    | .507   |
|         | Alcohol Abstinence |             |       | .257    | .011*  |
|         | Years of Alcohol Use |             |       | .089    | .517   |
| VPT     | 1     | .255        | 1.407 | -.064   | .021*  |
|         | Age   |             |       |         |       |
|         | BDS   |             |       | .343    | .000*  |
|         | CBT   |             |       | .279    | .003*  |
| VPT     | 2     | .270        | 1.394 | -.096   | .481   |
|         | Age   |             |       |         |       |
|         | BDS   |             |       | .392    | .000*  |
|         | CBT   |             |       | .307    | .002*  |
|         | Alcohol Units per week |             |       | -.046   | .626   |
|         | Alcohol Abstinence |             |       | -.205   | .031*  |
|         | Years of Alcohol Use |             |       | .052    | .678   |
| CBT     | 1     | .162        | 1.170 | -.067   | .492   |
|         | Age   |             |       |         |       |
|         | BDS   |             |       | .161    | .133   |
|         | VPT   |             |       | .314    | .003*  |

(Contd.)
| Tasks       | Entry | Model Summary | Standardised Coefficients |
|-------------|-------|---------------|----------------------------|
|             |       | R² (adj)      | SE            | β     | p   |
| CBT         | 2     | .188          | 1.152         |       |     |
| Age         |       |               |               | .080  | .525 |
| BDS         |       |               |               | .114  | .308 |
| VPT         |       |               |               | .342  | .002*|
| Alcohol Units per week | |               |               | .026  | .795 |
| Alcohol Abstinence | |               |               | .202  | .044*|
| Years of Alcohol Use | |               |               | −.215 | .104 |
| FDS         | 1     | .028          | 1.531         |       |     |
| Age         |       |               |               | −.195 | .053 |
| FDS         | 2     | .088          | 1.483         |       |     |
| Age         |       |               |               | −.130 | .315 |
| Alcohol Units per week | |               |               | −.042 | .693 |
| Alcohol Abstinence | |               |               | −.244 | .016*|
| Years of Alcohol Use | |               |               | −.136 | .326 |
| NvTMT       | 1     | −.006         | .564          |       |     |
| Age         |       |               |               | −.067 | .509 |
| NvTMT       | 2     | .118          | .528          |       |     |
| Age         |       |               |               | .048  | .707 |
| Alcohol Units per week | |               |               | −.181 | .085 |
| Alcohol Abstinence | |               |               | −.240 | .016*|
| Years of Alcohol Use | |               |               | −.188 | .168 |
| Stroop      | 1     | −.010         | .187          |       |     |
| Age         |       |               |               | .001  | .991 |
| Stroop      | 2     | .000          | .187          |       |     |
| Age         |       |               |               | −.034 | .802 |
| Alcohol Units per week | |               |               | −.126 | .257 |
| Alcohol Abstinence | |               |               | −.171 | .104 |
| Years of Alcohol Use | |               |               | .051  | .722 |

**Table 5:** Mediator variables entered in working memory models.  
*Note:* FDS = Forward Digit Span, BDS = Backward Digit Span, NvTMT = Non-Verbal Trail Marking Test. * Variable selected in each step (α = .05). BDS. excluded factor in entry 2: FDS.
During a second entry, alcohol abstinence and VPT significantly predicted CBT ($\beta = .202, p = .044; \beta = .342, p = < .002$). Also during a second entry, age, alcohol units consumed per week, years of alcohol use and BDS did not significantly predict CBT ($\beta = .080, p = .525; \beta = .026, p = .795; \beta = -.215, p = .104; \beta = .114, p = .308$).

**FDS.** During a first entry, age did not significantly predict FDS ($\beta = -.195, p = .053$). In a second entry, alcohol abstinence significantly predicted FDS ($\beta = -.244, p = .016$), while age, alcohol units consumed per week and years of alcohol use did not significantly predict FDS ($\beta = -.130, p = .315; \beta = -.042, p = .693; \beta = -.136, p = .326$).

**NvTMT.** During a first entry, age did not significantly predict NvTMT ($\beta = -.240, p = < .016$), while age, alcohol units consumed per week and years of alcohol use did not significantly predict NvTMT ($\beta = .048, p = .707; \beta = -.181, p = .085; \beta = -.188, p = .168$).

**Stroop.** During a first entry, age did not significantly predict Stroop ($\beta = .001, p = .991$). During a second entry, age, alcohol units consumed per week, alcohol abstinence and years of alcohol use did not significantly predict Stroop ($\beta = -.034, p = .802; \beta = -.126, p = .257; \beta = -.171, p = .104; \beta = -.051, p = .722$).

In summary, the factors entered in a first and second entry for CBT, VPT and BDS resulted in statistically significant models and the entered factors in a second entry for FDS and NvTMT resulted in statistically significant models. Further to this, age significantly predicted BDS in a first entry and continued to have a predictive effect in a second entry. Interestingly, alcohol abstinence significantly predicted BDS, CBT, VPT, FDS and NvTMT.

**Discussion**

The results from this study suggest that the length of alcohol abstinence significantly predicts mild impairments in BDS, CBT, VPT, FDS and NvTMT performance. Age also significantly predicted NvTMT and BDS performance, while none of the entered mediators in the Stroop model were found to predict task performance.

I found that the recent consumption of moderate alcohol use results in mild impairments in various modalities of working memory. Tasks assessing set-shifting, visual attention and verbal executive memory are impaired from recent alcohol use. Set-shifting and visual attention impairments resulting from recent alcohol use has previously been reported in healthy young adults (Day et al., 2013), alcohol use disorder patients (Hanson et al., 2011) and alcohol dependent patients (Uva et al., 2010). Therefore, these findings agree with the view that set-shifting and visual attention is affected by recent and acute alcohol use, which has been reported in healthy adults and also extends to clinical samples, where alcohol use results in further working memory impairments.

Mild verbal executive memory impairments due to recent alcohol use has similarly been reported with alcohol use disorder patients but cognitive recovery was not present in this sample (Hanson et al., 2011). To my knowledge, there are no studies that have reported this effect in a healthy sample. There are studies that establish that binge drinking (Parada et al., 2012) and acute alcohol use (Schweizer et al., 2006) impair verbal executive memory in healthy adults but these studies do not consider the effects of alcohol abstinence on working memory. I also found that a slave system, which operates via inner speech, becomes impaired from recent alcohol use and that recent alcohol consumption impairs an inner scribe that works on a visual cache and stores dynamic information and spatial rehearsal. However, a multimodal working memory account (Logie & Van der Meulen 2009) can explain these mild impairments as components that are intrinsically linked, which are also integrated and regulated by executive function. Therefore, it can be interpreted that the phonological loop may become susceptible to impairments, as it is one of the modalities of working memory. However, the literature contradicts this perspective and suggests that this effect occurs in clinical populations (Desmond et al., 2003; Hanson et al., 2011; Pitel et al., 2008). Therefore the results of the present study are surprising as phonological, spatial and visual impairments are more attributable to excessive alcohol use and in turn I offer the contradictory finding that executive function is an initial process that is impaired by alcohol use (Saults et al., 2007).

With the recent use of alcohol being a predictor for mild impairments in the phonological loop, visual memory, spatial memory, verbal executive memory and set-shifting, it was assumed that through alcohol abstinence these cognitions can also be predicted to recover in moderate alcohol drinkers. To my knowledge, the present finding of cognitive recovery in moderate drinkers is novel. It has been reported that after a 3-week period set-shifting does not recover (Uva et al., 2010), which can be explained by the dysfunction and/or atrophy of the brain, caused by hazardous alcohol use (Campanellaa et al., 2009). Perhaps my findings show that cognitive recovery remains present in moderate alcohol drinkers, while more hazardous alcohol use results in permanent working memory impairments.

I also found that age predicted mild impairments in set-shifting and verbal executive memory. I anticipated that age would have an effect on working memory (Park et al., 2010) as it strongly correlates with years of alcohol use (see Table 1), which also has an effect on working memory (Stavro et al., 2012). Thus, I added age initially in to the statistical models. This enabled me to distinguish between the effects of age and years of alcohol use on working memory. Despite the overlapping nature of these variables, the years of alcohol use did not have a predictive effect on working memory. Perhaps this shows that the years of alcohol use is not a significant predictive factor for young adults’ working memory architecture.

Further to this, none of the models found that alcohol units per week to be a significant predictor. This suggests that the recent use of alcohol is a more pertinent mediator for young adults’ working memory architecture, rather than units of alcohol consumed and years of alcohol.
use. The deleterious impairment that arises from alcohol units per week is perhaps more pertinent to intermediate, heavy and hazardous alcohol drinkers’ cognitions. However, these assumptions were not considered in this study, as the sample consisted mainly of non-alcohol to moderate alcohol drinkers.

Ergo, the current study offers results that support the first hypothesis that executive function is impaired by alcohol use in moderate alcohol drinkers. Therefore, my findings suggest that executive function is a process that is impaired by alcohol use and that other modalities of working memory are predicted by alcohol abstinence. The second and third hypothesis was rejected; alcohol units per week and years of alcohol use did not significantly predict working memory.

The current study further suggests that healthy adults’ phonological loop, visual, spatial, verbal executive and set-shifting components of executive function are predicted to be mildly impaired from alcohol use and can recover through abstinence. Here, the effects of alcohol on working memory can be discerned and the results of this study thus suggest that various modalities of working memory are affected by alcohol consumption. Further to this, when comparing the present findings to clinical studies, it seems that the recent use of alcohol in typical alcohol drinkers is distinct to clinic samples, who have hazardous alcohol consumption behaviours (Campanellaa et al., 2009), and thus their working memory architecture is distinct from typical adults (Stavro et al., 2012). While this distinction is established, the present study is one of a few that show the effects of alcohol on healthy adults’ working memory.

The present study’s main strength lies with the sample size. Another merit is it was possible to distinguish between the use of various working memory resources during a VPT and CBT. In order to make this distinction, battery of cognitive tasks was used that considered a multi-component working memory perspective (Logie & van der Meulen 2009). Based on theoretical consideration, I used a battery of cognitive tasks that accounted for the use of spatial and executive resources being used during a VPT and the use of executive resources utilised during a CBT. However, I did not account for visual resources or the possibility of other executive functions being utilised during a NvTMT. I chose not to consider the use of visual resources or inhibition during a NvTMT model, as both tasks that measure visual working memory and inhibition weakly correlated with NvTMT (see Table 2). Future investigations should consider additional cognitive tasks.

A limitation of this study was that I assessed participants’ alcohol use through self-report. In doing so, the data might be prone to bias as participants often under-report their alcohol use (Stockwell et al., 2004). I accept this limitation and would advocate that future investigations should gain a more comprehensive profile of participants’ alcohol consumption. Previous experimental studies have assessed participants’ blood alcohol concentration (Day et al., 2013; Saults et al., 2007). Here, no such measures were used as I was interested in alcohol consumption behaviours.

In summary, results from this study propose that set-shifting, verbal executive, phonological loop, spatial and visual working memory are predicted by alcohol abstinence. Ergo, the results suggest that the recent use of alcohol and the length of alcohol abstinence play a role in mild working memory impairments and in cognitive recovery in young adults. The years of alcohol use and alcohol units consumed in healthy young adult drinkers were found to not be significant predictive factors. While this study sheds light on the cognitive resources, which are affected by alcohol use, there are few studies that consider this empirically (Piechaczek et al., 2009). More research is needed that enhances our understanding on the effects of alcohol use in healthy young adults’ working memory.

Competing Interests
The author declares that they have no competing interests.

Ethical considerations
Research Ethics number: RE05-03-131756

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References
Baddeley, A. D. (1986). *Working Memory*. Oxford, UK: Oxford University Press. PMcid: PMC2498130.
Baddeley, A. (2003). Working memory: Looking back and looking forward. *Nature Reviews*, 4, 829–839. DOI: http://dx.doi.org/10.1038/nrn1201
Baliunas, D. O., Taylor, B. J., Irving, H., Roerecke, M., Patra, J., Mohapatra, S., & Rehm, J. (2009). Alcohol as a risk factor for type 2 diabetes: A systematic review and meta-analysis. *Diabetes care*, 32(11), 2123–2132. DOI: http://dx.doi.org/10.2337/dc09-0227
Batty, G. D., Lewars, H., Emslie, C., Gale, C. R., & Hunt, K. (2009). Internationally recognized guidelines for ‘sensible’ alcohol consumption: is exceeding them actually detrimental to health and social circumstances? Evidence from a population-based cohort study. *Journal of Public Health*, 31(3), 360–365. DOI: http://dx.doi.org/10.1093/pubmed/fdp063
Buchanan, T., Heffernan, T. M., Parrott, A. C., Ling, J., Rodgers, J., & Scholey, A. (2010). A short self-report measure of problems with executive function suitable for administration via the Internet. *Behaviour and Research Methods*, 42, 709–710. DOI: http://dx.doi.org/10.3758/BRM.42.3.709
Campanellaa, S., Petit, G., Maurage, P., Kornreicha, C., Verbancka, P., & Noël, X. (2009). Chronic alcoholism: Insights from neurophysiology. *Clinical Neurophysiology*, 39, 191–207. DOI: http://dx.doi.org/10.1016/j.neucli.2009.08.002
Chao, L. L., Meyerhoff, D. J., Cardenas, V. A., Rothchild, J. C., & Weiner, M. W. (2003). Abnormal CNV in chronic heavy drinkers. *Clinical Neurophysiology*, 114(11), 2081–2095. DOI: http://dx.doi.org/10.1016/S1388-2457(03)00230-X
Day, A. M., Celio, M. A., Lisman, S. A., Johansen, G. E., & Spear, L. P. (2013). Acute and chronic effects of alcohol on trail making test performance among underage drinkers in a field setting. *Journal of Studies on Alcohol and Drugs*, 74(4), 635. DOI: http://dx.doi.org/10.15288/jsad.2013.74.635. PMid: 23739029; PMCID: PMC3711354.

Della Sala, S., Gray, C., Baddeley, A., Allamano, N., & Wilson, L. (1999). Pattern span: A tool for unwelding visuo-spatial memory. *Neuropsychologia*, 37, 1189–1199. DOI: http://dx.doi.org/10.1016/S0028-7317(99)00159-6.

Desmond, J. E., Chen, A. S. H., DeRosa, E., Pryor, M. R., Pfefferbaum, A., & Sullivan, E. V. (2003). Increased frontocerebellar activation in alcoholics during verbal working memory: an fMRI study. *Neuroimage*, 19, 1510–1520. DOI: http://dx.doi.org/10.1016/S1053-8119(03)00102-2.

Di Castelnuovo, A., Costanzo, S., Bagnardi, V., Donati, M. B., Iacoviello, L., & de Gaetano, G. (2006). Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. *Archives of Internal Medicine*, 166(22), 2437–2445. DOI: http://dx.doi.org/10.1001/archinte.166.22.2437.

Duka, T., & Townshend, J. M. (2004) The priming effect of alcohol pre-load on attentional bias to alcohol-related stimuli. *Psychopharmacology*, 176, 353–361. DOI: http://dx.doi.org/10.1007/s00213-004-1906-7.

Gustafson, R., & Kallmen, H. (1990). Effects of alcohol on prolonged cognitive performance measured with Stroop’s Color Word Test. *Psychological Reports*, 67, 643–650. DOI: http://dx.doi.org/10.2466/pr0.1990.67.2.643; http://dx.doi.org/10.2466/PR0.67.6.643-650. PMid: 2263718.

Hanson, K. L., Medina, K. L., Padula, C. B., Tapert, S. F., & Brown, S. A. (2011). Impact of adolescent alcohol and drug use on neuropsychological functioning in young adulthood: 10-year outcomes. *Journal of Child and Adolescent Substance Abuse*, 20, 135–154. DOI: http://dx.doi.org/10.1080/1067828X.2009.100153.x.

Heffernan, T. M., Ling, T. M., & Bartholomew, J. (2004). Self-rated prospective memory and central executive impairments in excessive alcohol users. *Irish Journal of Psychological Medicine*, 21(4), 122–124. DOI: http://dx.doi.org/10.1093/alc/mcl3.269.

International Centre for Alcohol Policies (ICAP). (2010). *International Drinking Guidelines*. International Centre for Alcohol Policies, 2010.

Jiang, Y. V., Makovski, T., & Mok Shim, W. (2009). Visual memory for features, conjunctions, objects, and locations. In J. R. Brockmole (Ed.), *The Visual World in Memory*. New York, NY: Psychology Press, pp. 33–65.

Kopera, M., Wojnar, M., Brower, K., Glass, J., & Nowosad, I. (2012). Cognitive functions in abstinent alcohol-dependent patients. *Alcohol*, 46, 665–671. DOI: http://dx.doi.org/10.1016/j.alcohol.2012.04.005.

Logie, R. H., & Van der Meulen, M. (2009). Visual memory for features, conjunctions, objects, and locations. In J. R. Brockmole, (Eds.), *The Visual World in Memory: Fragmenting and integrating visuospatial working memory*. New York, NY: Psychological Press, pp. 1–33.

Luck, S. J., & Vogel, E. K. (1997). The capacity of visual working memory for features and conjunctions. *Nature*, 390(6657), 279–281. DOI: http://dx.doi.org/10.1038/38648. PMid: 9384378.

Mota, N., Parada, M., Crego, A., Doallo, S., Caamaño-Isorna, F., Rodríguez Holguín, S., & Corral, M. (2013). Binge drinking trajectory and neuropsychological functioning among university students: A longitudinal study. *Drug and Alcohol Dependence*. DOI: http://dx.doi.org/10.1016/j.drugalcdep.2013.05.024.

National Institute for Health and Clinical Excellence (NICE). (2010). *National Institute for Health and Clinical Excellence: Alcohol use disorders: preventing the development of hazardous and harmful drinking*. London: NICE, 2010.

Noël, X., Paternot, J., Van der Linden, M., Sferrazza, R., Verhas, M., Hanak, C., . . ., Verbanck, P. (2001). Correlation between inhibition, working memory and delimited frontal area blood flow measured by 99mTc–bicisate spect in alcohol–dependent patients. *Alcohol and Alcoholism*, 36(6), 556–563. DOI: http://dx.doi.org/10.1093/alcalc/36.6.556.

Parada, M., Corral, M., Mota, N., Crego, A., Holguin, S. R., & Cadaveira, F. (2012). Executive functioning and alcohol binge drinking in university students. *Addictive Behaviors*, 37, 167–172. DOI: http://dx.doi.org/10.1016/j.addbeh.2011.09.015.

Park, J., Carp, J., Hebrank, A., Park, D. C., & Polk, T. A. (2010). Neural Specificity Predicts Fluid Processing Ability in Older Adults. *The Journal of Neuroscience*, 30(27), 9253–9259. DOI: http://dx.doi.org/10.1523/JNEUROSCI.0853-10.2010.

Piechatzek, M., Indlekofer, F., Daamen, M., Glasmacher, C., Lieb, R., Pfister, H., Tucha, O., Lange, K. W., Wittchen, H. U., & Schutz, C. G. (2009). Is moderate substance use associated with altered executive functioning in a population-based sample of young adults? *Human Psychopharmacology*, 24, 650–665. DOI: http://dx.doi.org/10.1002/hup.1069.

Pitel, A. L., Beunineux, H., Witkowski, T., Vabret, F., Sayette, V., Viader, F., . . ., Eustache, F. (2008). Epidemic and Working Memory Deficits in Alcoholic Korsakoff Patients: The Continuity Theory Revisited. *Alcoholism: Clinical and Experimental Research*, 32(7), 1229–1241. DOI: http://dx.doi.org/10.1111/j.1530-0277.2008.00677.x.

Rehm, J., Taylor, B., Mohapatra, S., Irvine, H., Balianas, D., Patra, J., & Roerecke, M. (2010). Alcohol as a risk factor for liver cirrhosis: A systematic review and meta-analysis. *Drug and Alcohol Review*, 29, 437–445. DOI: http://dx.doi.org/10.1111/j.1465-3362.2009.00153.x.

Rose, A. K., & Duka, T. (2006). Effects of dose and time on the ability of alcohol to prime social drinkers. *Behavioural Pharmacology*, 17, 61–70. DOI: http://dx.doi.org/10.1097/01.fbp.0000189814.61802.92.

Ross, T. P., Hanouskova, E., Giarla, K., Calhoun, E., & Tucker, M. (2007). The reliability and validity of the self-ordered pointing task. *Archives of Clinical
Neuropsychology, 22 449–458. DOI: http://dx.doi.org/10.1016/j.acn.2007.01.023

Sánchez-Cubillo, I., Periáñez, J. A., Adrover-Roig, D., Rodríguez-Sánchez, J. M., Rios-Lago, M., Tirapu, J., & Barceló, F. (2009). Construct validity of the Trail Making Test: role of task-switching, working memory, inhibition/interference control, and visuo-motor abilities. Journal of the International Neuropsychological Society, 15, 438–450. DOI: http://dx.doi.org/10.1017/S1355617709090626

Saults, J. S., Cowan, N., Sher, K. J., & Moreno, M. V. (2007). Differential effects of alcohol on working memory: distinguishing multiple processes. Experimental and Clinical Psychopharmacology, 15(6), 576. DOI: http://dx.doi.org/10.1037/1064-1297.15.6.576

Schweizer, T., Vogel-Sprott, M., Danckert, J., Roy, E., Skakum, A., & Broderick, C. (2006). Neuropsychological profile of acute alcohol intoxication during ascending and descending blood alcohol concentrations. Neuropsychopharmacology, 31, 1301–1309. DOI: http://dx.doi.org/10.1038/sj.npp.1300941

Shah, P., & Miyake, A. (1999). Models of Working Memory. In P. Shah & A. Miyake (Eds.), Models of WM: Mechanisms of active maintenance and executive control. (pp. 1–26). Cambridge, UK: Cambridge University Press. DOI: http://dx.doi.org/10.1017/CBO9781139174909.004

Stavro, K., Pelletier, J., & Potvin, S. (2012). Widespread and sustained cognitive deficits in alcoholism: A meta-analysis. Addiction Biology, 18, 203–213. DOI: http://dx.doi.org/10.1111/j.1369-1600.2011.00418.x

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. Journal of Experimental Psychology, 18, 643–662. DOI: http://dx.doi.org/10.1037/h0054651

Stockwell, T., Donath, S., Cooper-Stanbury, M., Chikritzhs, T., Catalano, P., & Mateo, C. (2004). Under-reporting of alcohol consumption in household surveys: a comparison of quantity–frequency, graduated–frequency and recent recall. Addiction, 99(8), 1024–1033. DOI: http://dx.doi.org/10.1111/j.1360-0443.2004.00815.x

Tapert, S. F., Schweinsburg, A. D., Barlett, V. C., Brown, S. A., Frank, L. R., Brown, G. G., & Meloy, M. J. (2004). Blood Oxygen Level Dependent Response and Spatial Working Memory in Adolescents With Alcohol Use Disorders. Alcoholism: Clinical and Experimental Research, 28, 1577–1586. DOI: http://dx.doi.org/10.1097/01.ALC.0000141812.81234.A6

Thompson, J. M., Hamilton, C. J., Gray, J. M., Quinn, J. G., Mackin, P., Young, A. H., & Ferrier, I. N. (2006). Executive and visuospatial sketchpad resources in euthymic bipolar disorder: Implications for visuospatial working memory architecture. Memory, 14 (4), 437–451. DOI: http://dx.doi.org/10.1080/09602930600870042

Uva, M. C. S., Luminet, O., Cortesi, M., Constant, E., Derely, M., & De Timiry, P. (2010). Cognitive And Behavioural aspects distinct effects of protracted withdrawal on affect, craving, selective attention and executive functions among alcohol-dependent patients. Alcohol & Alcoholism, 45(3), 241–246. DOI: http://dx.doi.org/10.1093/alc/aga012

Vollstadt-Klein, S., Hermann, D., Rabinstein, J., Wichert, S., Klein, O., Ende, G., & Mann, K. (2010). Increased activation of the ACC during a Spatial Working Memory Task in Alcohol-Dependence versus Heavy Social Drinking. Alcoholism: Clinical and Experimental Research, 34(5), 771–776. DOI: http://dx.doi.org/10.1111/j.1530-0277.2010.01149.x

Vorstius, C., Radach, R., Lang, A. R., & Riccardi, C. J. (2008). Specific visuomotor deficits due to alcohol intoxication: Evidence from the pro- and antisaccade paradigms. Psychopharmacology, 196(2), 201–210. DOI: http://dx.doi.org/10.1007/s00213-007-0954-1