

The effects of heavy social drinking on executive function: A systematic review and meta-analytic study of existing literature and new empirical findings

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Abstract

Background: Previous investigations of executive function in alcohol dependent and in social drinkers have not always produced consistent results and have not utilised key indicators of recent theoretical models of EF. The present paper reports the results of two studies which seek to address these limitations: *Method:* Study 1 took the form of a systematic review and meta-analysis of studies examining EF in social drinkers. In Study 2, 41 participants completed an alcohol use inventory and were assigned to either light or heavy alcohol use via median split of average weekly dose. Participants completed measures of the fractionated executive processes of updating, shifting, inhibition and access to semantic memory. *Results:* Study 1 only identified seven studies of EF in social drinkers, and the mean effect size was non-significant. In study 2 the heavy alcohol use group performed worse on all measures of executive functioning except memory updating. *Conclusions:* To our knowledge this is the first systematic investigation executive functioning in social drinkers. Given that the participants were non-treatment seeking social drinking students, the EF deficit in the heavy user group is particularly worrying and could increase the likelihood of developing an alcohol use disorder.

CONFLICT OF INTEREST

None.

Keywords: Alcohol use; executive function; social drinking.

Introduction

Previous research into recreational alcohol use has shown that prospective remembering (remembering to do something at a particular timepoint in the future) is impaired by heavy social drinking (Heffernan et al. 2001). While some studies have investigated executive functioning in social drinkers (Blume et al. 2000; Townshend & Duka, 2005), the results are equivocal and there has not to our knowledge, been a systematic investigation of executive functioning in social drinkers using key indicators which take account of recent theoretical conceptualisations emphasising the fractionated nature of the construct. The present paper reports two studies: Study 1 is a systematic review and meta-analysis of existing research into executive deficits in social drinkers. Study 2 is an investigation of executive function deficits in social drinkers.

Alcohol is the most popular sedative hypnotic in western society, yet it is misused by many. In the UK, individuals frequently report exceeding the government's "safe" guidelines for drinking both in adolescence and adulthood (ONS, 2010). Acute alcohol intoxication results in decreased glutamatergic transmission, particularly in the hippocampus, as alcohol inhibits action at glutamatergic NMDA receptors (Blitzer et al. 1990; Morrisett et al. 1993). Alcohol also potentiates GABA neurotransmission, resulting in increased neuronal inhibition (Kumar et al. 2009). While acute alcohol intoxication in moderate doses will cause reliable changes in GABA and Glutamate neurotransmission, such use is not believed to be neurotoxic, and individuals drinking within safe limits are unlikely to suffer adverse long-term brain effects. However, with long-term heavy use, alcohol can be neurotoxic (Harper 2007). For example in animal models, it has been shown that binge drinking rats will sustain damage to various regions, particularly the hippocampus (Crews & Nixon, 2009; Nixon and Crews, 2002; Obernier et al. 2002). In chronic heavy drinking humans who have developed Wernicke-Korsakoff Syndrome (WKS), degradation can be seen in the diencephalon,

primarily related to Thiamine deficiency (Mann et al. 2001). In chronic heavy drinkers who have not developed WKS, damage can be seen in the hippocampus (Agartz et al. 1999; Cadete-Leite et al. 1988; Obernier et al. 2002).

In addition, chronic heavy alcohol users (i.e. individuals with alcoholism of non-WKS type) show a variety of deficits on tasks designed to tap frontal lobe, medial temporal lobe and executive functioning. For example, word list recall (Bachara et al. 2001), working memory (Ambrose et al. 2001) and switching and inhibitory control (Noel et al. 2001) are all impaired after chronic alcohol use. Furthermore, in a study comparing the performance of chronic alcoholics and controls on tasks that all rely heavily on frontal lobe function, Ratti et al. (2002) found that chronic alcoholics (who had consumed at least 100g of alcohol a day for at least 15 years) performed worse on the Trail Making Test (TMT), the Wisconsin Card Sorting Task (WCST), and had slower reaction times and information processing speed. There were however no significant differences on the Stroop task.

With the ever-increasing trend of binge drinking (e.g. Courtney & Polich, 2009), the implications of heavy social drinking are equally worrying. Non-dependent heavy alcohol users report more prospective memory slips on short-term, long-term and internally cued prospective memory (Heffernan et al. 2001) suggesting that aspects of everyday memory are affected by heavy drinking. More recently Heffernan et al (2010) found that binge drinkers were impaired on the laboratory-based Prospective Remembering Video Task. More specifically binge drinkers failed to execute as many prospective location-action combinations compared to normal drinkers. There appear to be few studies of executive functioning and heavy social drinking, but the available evidence suggests that performance on executive tasks may covary with alcohol consumption. Among college students who met DSM-IV criteria for alcohol abuse or dependence, Blume et al. (2000) found that performance on the Weschler Memory Scale (WMS) was associated with an awareness of

drinking problems but no relationship was observed between awareness and performance on the Wisconsin Card Sorting Task or a figural fluency task. With regard to non-clinical participant samples, Townshend and Duka (2005) found that binge drinking social drinkers actually had faster reaction times on a matching-to-sample task, and while group differences on visuospatial memory were non-significant between binge drinkers and non-binge drinkers, there was a significant binge x gender interaction indicating that female binge drinkers performed worse than female non-binge drinkers. However, Nederkoorn et al. (2009) found that heavy and light social drinkers did not differ in response inhibition (using a stop-signal task). Thus there is a need to clarify this disparity in findings. In addition to heavy drinking causing differences in prefrontal cortical functioning, it is likely that premorbid differences in prefrontal cortical functioning mediate behaviours associated with heavy/risky drinking, or that the two share a reciprocal relationship. Recently, Lyvers et al. (2011) found significant relationships between risky drinking by young adults and self-reports of executive function deficits. In addition, impulsivity, reward sensitivity, and familial alcoholism, were related to more risky drinking in young adults which suggests that in some young adults there may be differences in prefrontal functioning that predate heavy use and predispose to heavier and riskier alcohol use.

Study 1 took the form of a systematic search of academic databases to identify and review studies reporting findings with regard to the presence or absence of alcohol related executive performance impairments in social drinkers, as opposed to clinical populations of drinkers. An additional aim of this first study was to apply meta-analytic techniques to the results of the studies identified, in order to examine the relationship between alcohol consumption and executive performance in social drinkers. The second study sought to examine the relationship between heavy social drinking in non-clinical participant samples,

and performance on different components of executive functioning which have been identified empirically (Miyake et al., 2000).

Study 1

Introduction

The need for a systematic search of relevant databases for studies addressing the relationship between heavy social drinking and executive functioning in non-clinical participant samples was indicated by the small number of studies we were able to identify by less formal search strategies. Participant samples are defined as non-clinical here if they were recruited from non-clinical populations and no formal diagnosis of an alcohol or other substance use disorder was reported for participants. However, it should be noted that assessments indicating heavy social drinking may indicate a level of alcohol consumption for some participants whereby such a diagnosis was appropriate.

Method

Identification of Studies. The databases searched were PsycINFO (1806 – March 2011), Medline (1950 – March 2011), CINALH (1994 – March 2011) and Embase (1980 – March 2011). In Phase 1 of the search the following search terms, singly and in combination with Medical Subjects Headings (MeSH) terms, were used to search the databases: alcohol, executive function, shifting, switching, and updating. The use of the Thesaurus in each database allowed for keywords and terms to be exploded ensuring all relevant terms were included in the search. The search was limited to studies relating to humans and those published in English language peer reviewed journals. Non- English language publications were excluded in order to avoid translation bias. In Phase 2 of the search the additional inclusion criterion of findings based upon a non-clinical sample of social drinkers was added. This criterion had not been employed in Phase 1 in order to avoid any risk of studies not

being identified due to authors describing their samples in different ways. Our strategy, therefore, was to initially identify electronically a relatively large pool of studies which had examined the relationship between alcohol use and executive functioning, and then identify those within that pool which had recruited from the population of interest. One study matching the inclusion criteria, but which had been missed by the database searches, was subsequently added to the review sample.

Data extracted.

For each study included in the review, the location (by country) of data collection was recorded, together with details of sub-groups (e.g. binge drinkers, non-binge drinkers), mean (*SD*) ages for sub-groups, and the population targeted for recruitment (e.g. students). Estimated mean weekly alcohol consumption was recorded in the form reported by the original authors. Details of the executive tasks administered were recorded, together with relevant findings regarding task performance. Given the importance of controlling for potential confounds which might produce misleading results, the details of any statistical procedures to control for such things as age, or other substances consumed were also recorded. Where studies could be included in the meta-analysis the means, standard deviations, and group sizes were recorded for each relevant inter-group comparison.

Analytic strategy.

Summaries of sample details, weekly alcohol consumption, executive tasks used, relevant findings, and statistical controls for confounds were prepared for tabulated presentation. Data for meta-analysis were stored and analysed using Comprehensive Meta-Analysis (CMA 2.0™) software. Data for the meta-analysis took the form of performance means, standard deviations, and group sizes for each inter-group comparison on each dependent variable from

each executive task administered. However, in order for the interpretation of meta-analytic results to be as specific as possible, self-report questionnaire data on perceived executive problems was not included. The meta-analysis, therefore, focussed upon objective measures of performance on executive tasks. Within the meta-analysis each study was represented by the mean effect size for all inter-group comparisons reported. This controlled for the distorting effects which would have arisen if multiple effect sizes, arising from outcomes of comparisons which were not independent, had been used (Borenstein et al., 2009). Hedges' g was chosen as the effect size statistic, as this controls for distortions arising from small samples which can arise with the more commonly used Cohen's d statistic. In the event of a significant meta-analytic result, recourse would be made to Rosenthal's *Fail-safe N*, which would indicate the minimum number of studies required to render the result nonsignificant. This would be necessary as the result would be based upon a search where only studies published in peer reviewed journals had been included. There would, therefore, be a risk that the result may have been influenced by a bias towards the publication of significant findings, with nonsignificant findings being less likely to have been published in such journals (Lipsey & Wilson, 2001).

Study 1 Results

Overview

The process of the literature search is illustrated in Figure 1 where the term 'publications' is used for Phase 1 search outcomes due to variety in the types of outputs identified, and the term 'studies' is used for Phase 2 search outcomes to indicate the greater potential relevance of these outputs to this review. The original database searches failed to identify Heffernan et al. (2004), with this study being added following initial manuscript review. Table 1 summarises details concerning samples, weekly alcohol intake, executive tasks used, relevant

results, and statistical controls for confounds, for each of the seven studies identified. Five of the studies recruited participants from student populations, with the oldest mean age for any of the studies being 25.83 years ($SD = 3.08$ years), reported for one of the non-student samples. The study in question (Piechatzek et al., 2009) differed in its recruitment strategy from the other five studies by drawing upon a larger sample which had been recruited to an epidemiological study of psychopathology development. Nevertheless, none of the participants within this study had any history of diagnosed psychiatric or neurological conditions. The remaining studies had recruited by means of advertisements, or through a general ‘participant pool’ within a university.

Two studies indicated that female participants could be more vulnerable to performance impairments related to heavy drinking than males. Female heavy drinkers showing impaired response inhibition compared to female light drinkers and males in both drinking groups (NederKoorn et al., 2009), and they also made more spatial working memory errors than female non-binge drinkers whilst males showed no performance differences related to drinking status (Townsend & Duka, 2005). Two studies reported examples of the heavy drinkers performing better on a task than the light drinking controls (Piechatzek et al., 2009; Townsend & Duka, 2005). The one study to obtain self-reported measures of perceived executive functioning (Heffernan et al., 2004), using the Dysexecutive Questionnaire (DEX), reported that excessive alcohol users reported a significantly greater number of problems associated with the executive component of memory, than did a control group comprising low dose and nonusers of alcohol.

Insert Figure 1 and Table 1 about here

Meta-analytic findings

Only four of the six studies identified in this review presented detailed findings from inter-group comparisons of the performance of heavy social drinkers and controls on executive tasks (Crego et al., 2009; Hartley et al., 2004; Nederkoorn et al., 2009; Townshend & Duka, 2001). Townshend and Duka (2005) did not report statistical details for inter-group comparisons which failed to be significant. Piechatzek et al. (2009) concentrated on regression analyses of their whole sample but, once again, detailed results for alcohol consumption were only included where a significant result had been obtained. In both cases, the exclusion of nonsignificant results for a meta-analysis would introduce a bias into the result. Heffernan et al. (2004) reported results from a self-report questionnaire of perceived executive functioning, rather than task performance data. Consequently, only the four studies reporting detailed results for inter-group performance comparisons on executive tasks were included in the meta-analysis. The demographic similarity between the samples in these four studies also facilitated the *a priori* choice of a fixed-effect model as appropriate for the meta-analysis, with one true effect size for the population studied being assumed. Recommended practice for meta-analysis is for the choice between a fixed-effect model and a random-effects model to be made on an *a priori* basis, rather than upon findings of heterogeneity in effect sizes (Borenstein et al., 2009). Where a fixed-effect model can be validly chosen as appropriate, this carries the advantage of meta-analysis being possible with as little as two studies included. By contrast, a random-effects model requires a greater number of studies to produce a reliable result as dispersion in true effect sizes has to be addressed. Outcomes from the four studies were coded as negative if the heavy social drinkers performed worse than the controls, and as positive if the outcome was in the opposite direction.

The four studies included in the meta-analysis yielded a mean weighted effect size which was small and nonsignificant (Hedges $g = 0.082$; 95% CI -0.222 (lower) to 0.386 (upper); $z =$

0.529, $p = .597$, two-tailed). The obtained Q statistic, testing the null hypothesis that the studies shared a common effect size, was nonsignificant ($Q [df = 3] = 1.986, p = .575$). Finally, the I^2 statistic measuring the inconsistency of findings across studies yielded a result of 0.000%. Similarly, the T^2 measure of variance in true effect sizes also yielded 0.000 as a result, which is consistent with the assumption of a fixed-effect model. The results of the meta-analysis are illustrated in the forest plot shown in Figure 2.

Insert Figure 2 about here

Study 1 Discussion

The meta-analytic results do not support the hypothesis that executive functioning is impaired in heavy drinking non-clinical participant samples. While the meta analysis shows that attempts have been made to investigate the executive functioning of social drinkers, not all studies have compared heavy social drinkers with normal drinkers (Piechatzek et al., 2009). Furthermore only one or two tasks measuring executive functioning have typically been administered. This is problematic because it is known that individual tasks, while loading on the PFC, also recruit on more posterior AND sub-cortical neural structures in a task-dependent manner (Collette et al., 2005) and so any deficits that are observed might relate to these structures rather than PFC processes. Finally, of the executive tasks listed in Table 1, only the Stroop and stop signal tasks have been empirically related to a specific executive function, in both cases the inhibition of ‘pre-potent responses’ (Fisk & Sharp, 2004, Miyake et al., 2000). Additionally, the tests of visuospatial memory may be assumed to draw upon executive functioning as this area of memory performance has been empirically linked to executive resources (Miyake et al., 2001). However, whilst the remaining tasks listed in Table 1 share some similarity with other tasks for which a link to a specific executive function has been empirically demonstrated, it should not be assumed automatically that the remaining tasks in Table 1 readily map onto contemporary theoretical models of executive

function. A systematic investigation of specific executive functions in heavy social drinkers utilising tasks with a demonstrated link to specific executive functions would be beneficial as such functions may regulate drinking behaviours in social drinkers (e.g. Whitney et al. 2006) and substance-dependent individuals (e.g. Wiers et al. 2007), and awareness of executive deficits could inform treatment programmes.

Moreover, recent theoretical models of executive functioning postulate that the central executive of working memory is fractionated, with its components performing separate tasks with varying degrees of ability. One of the most accepted models of the fractionated central executive was developed by Miyake et al. (2000). They studied the separability of three proposed executive functions: mental set shifting (“shifting”), information updating and monitoring (“updating”), and the inhibition of pre-potent responses (“inhibition”), and how performance on each function contributed to performance on some well-known executive tasks. Structural equation modelling revealed that while the three executive functions were moderately correlated with each other, they were clearly separable and each contributed differentially to performance on the EF tasks. For example, the Wisconsin Card Sorting Task (WCST) was linked to the shifting component, the Tower of Hanoi to the inhibition component, random number generation to both the inhibition and updating components, and operation span to the updating component. In a study of cognitive ageing, Fisk and Sharp (2004) provided further support for Miyake et al’s model. Factor analysis revealed that certain tasks loaded on each of the three components identified by Miyake et al, but there was also a distinct executive function loading on another factor, which Fisk and Sharp termed access to long-term memory (although age was not a significant predictor of performance on “access” tasks). Study 2 utilised this model to assess which components of executive functioning might be susceptible to the effects of heavy alcohol use.

Study 2

Study 2 Introduction

To date there has been no systematic investigation of whether or not heavy *social* drinkers are impaired in the different aspects of executive functioning identified by Miyake et al. (2000). Existing research findings have not always made use of the traditional measures of the different executive subcomponents identified by Miyake et al (2000) and most importantly have not considered the full range of executive tasks within a single study so as to rule out the possibility that any deficits that are observed relate to the non executive task components. Therefore the present study sought to ascertain the nature of executive function deficits in a sample of recreational alcohol drinkers. If a global (multivariate) deficit is observed this would be consistent with a more broadly based alcohol-related PFC deficit. However, if no such multivariate effect is observed, while significant group differences are apparent on one or two individual measures, this might implicate deficits in more posterior or sub-cortical neural areas or alternatively more narrowly defined regions within the PFC.

Impaired inhibitory control has also been cited as a contributor to the development of addictive behaviour (Wiers et al. 2007), and poor inhibitory control may be a cause as well as a consequence of chronic heavy drinking (Nigg et al. 2006). Indeed recent research has shown that Therefore, in the present study it may be that those who are heavier social drinkers may not be able to control their drinking as well as more restrained drinkers and will exhibit specific inhibitory control deficits. Performance on a response inhibition task has been linked to the pre-frontal cortex (Casey et al. 1997; Kiefer et al. 1998), and more specifically has been shown to be sensitive to damage to the inferior frontal gyrus (Aron et al. 2003). Thus if deficits are restricted to inhibitory functions this might implicate the latter structure as being especially sensitive to alcohol-related effects. Less is known about the effects of alcohol on the switching component process. Given the significant group

differences documented above on the TMT and WCST (Ratti et al. 2002), which Miyake et al. (2000) found to load on to the switching component, it is likely that attention switching will be affected by heavy social drinking. Since performance on a switching task has been linked to the anterior cingulate cortex (Posner & Raichle, 1994), the left frontal lobe (Rogers et al. 1998) and the bioccipital and parietal lobes (Moulden et al. 1998), if deficits are restricted to the switching process this might implicate more posterior brain areas as being especially sensitive to alcohol-related effects. If it is the case that impairments emerge solely on the updating function this may be indicative of an alcohol-use related deficit in the dorsolateral prefrontal cortex (Goldman-Rakic, 1996) and more specifically the left fronto-polar cortex (Van-der-Linden et al. 1999). As long-term heavy drinking has been shown to cause degeneration in hippocampal areas, and the Hippocampus may be involved in not only the consolidation to, but the access from long-term memory (Suzuki et al. 2004), it is possible that heavy social drinkers may start to show a similar pattern of degeneration. If it is the case that impairments emerge solely on the access function this might therefore provide further evidence for medial-temporal alcohol related effects rather than PFC problems.

To summarise, study 2 aimed to assess executive function in social drinkers. The executive measures were as follows: Computation Span (memory updating), Random Letter Generation (inhibition), the number-letter task (switching) and the Chicago word fluency task (access to semantic memory). Participants were allocated to either light or heavy alcohol use based on a median split of their average weekly alcohol consumption. It was predicted that heavy social drinkers would perform worse on at least some of the executive function measures.

Study 2 Method

Design

The study utilised a between groups design with level of alcohol use (Light vs. Heavy) as the between groups variable. The dependent variables were the various scores on the executive function tasks. MANOVA was used to analyse the data.

Participants

Forty-one participants took part. Median split was used to divide the participants into two groups based on their average weekly alcohol consumption. The mean age of the light alcohol group was 23.95 years (SD = 3.46; 6 Male; N = 21), and the mean age of the heavy alcohol group was 21.86 years (SD = 5.13; 14 Male). Participants were eligible to take part if they were aged between 18-35, drank alcohol at least occasionally, had *never* used an illicit substance, had never been diagnosed with an alcohol or substance use disorder or been advised to reduce their drinking. The study was approved by Liverpool John Moores University Research Ethics Committee.

Materials.

The UWIST Mood Adjective Checklist (UMACL- Matthews et al. 1994)

As state mood may affect performance on executive function tasks, the UWIST was included to measure state anxiety, arousal and depression. This is an 18 item checklist, and participants have to indicate how they are feeling at the time of testing on a 5–point Likert scale ranging from “not at all” to “extremely”. The test yields scores for State Anxiety (items include: tense, calm), Arousal (items include: fatigued, alert) and Depressed Mood (items include: sad, cheerful). A total score for each scale is calculated by summing the component responses, taking account of reverse scored items, thus a high score (above the midpoint of 18) is indicative of higher levels of anxiety, arousal and depression.

Alcohol Use Disorders Identification Test (AUDIT: Saunders et al. 1993)

The AUDIT consists of 10 Likert scaled items and is used to identify the signs of hazardous drinking, asking questions on the frequency and intensity of recent alcohol use. A score of greater than 8 indicates a strong likelihood of hazardous or harmful alcohol consumption.

Computation Span (Salthouse & Babcock, 1991).

Computation span has been used extensively as an indicator of working memory functioning in the cognitive ageing literature (Fisk & Warr, 1996) and is analogous to the operation span measure used by Miyake et al. (2000). Participants are required to solve a number of arithmetic problems (e.g., $4+7 = ?$) by circling one of three multiple-choice answers as each problem appears on the computer screen. Participants are required to simultaneously remember the second digit of each problem. At the end of each set of problems participants must recall the second digits in the order in which they were presented. The number of arithmetic problems that the participant has to solve, while at the same time remembering each second digit, gradually increases. For each of the first three trials only a single problem is presented. For the next three trials, two problems are presented. Subsequently, the number of problems presented per trial increases by one every third trial. In order to proceed, the participants are required to be correct in at least two of the three trials at each level.

Computation span is defined as the maximum number of end digits recalled in serial order, with the added requirement that the corresponding arithmetic problems are also correct

Chicago Word Fluency Test (CWFT: Cohen & Stanczak 2000)

Firstly participants are given five minutes to write down as many words as they can, beginning with the letter “S”. Secondly, they are given four minutes to write down as many

four-letter words beginning with “C” as they can. In both cases, the score is the number of correct words produced, with higher scores indicative of better access to semantic memory. Participants are instructed not to write any place names, peoples name or plurals in this test.

Number-Letter task (Miyake et al. 2000)

A number letter pair (e.g.D4) is presented in one of four quadrants on a computer screen. If the target is in the top half of the screen, participants must make a letter judgement (indicate if the letter is a vowel or a consonant). If the target is in the bottom half the participants must make a number judgement (is the number is odd or even). The task starts with a practise version of 3 sets. The target is presented in the top half of the screen for 12 trials, then the bottom half for 12 trials, and then in a clockwise rotation around all 4 quadrants for a further 12 trials. The main task follows the same structure, with 64 targets in each block. Thus the third block of the practise and main task requires participants to switch between making number judgements and letter judgements, and this switch is internally driven. The first two blocks require no switching, while the third set does. The switch-cost is the difference between the average reaction times of the third block and the averages of the first two blocks.

Random letter generation (Baddeley 1996)

A computer display and concurrent auditory signal is used to pace responses. Participants are asked to speak aloud a letter every time the signal is presented. They are instructed to avoid creating repeat sequences of letters or producing alphabetical sequences, and to try to speak each letter with the same overall frequency. Participants attempt to produce three sets of 100 letters; one set at a rate of one letter every 4 s, 2s and 1s. The order in which the sets are generated is randomised for each participant. The test yields three scores. First, the number of alphabetically ordered pairs; second, a repeat sequences score corresponding to the number of

times that the same letter pair is repeated; third, a “redundancy” score, which measures the extent to which all 26 letters of the alphabet are produced equally often (0% being truly random). In all cases, higher scores indicate poorer performance. The scores for each separate variable, at each of the three generation rates, were standardised. A single score for each random generation measure was produced by averaging the standardised scores for the three production rates.

Procedure

Participants gave informed consent and completed the AUDIT and the UMACL. Participants then completed Random letter generation, Computation Span, Word Fluency Test and the Number-Letter task. Order of these tasks was counterbalanced to counteract order and fatigue effects. At the end of the session, participants were debriefed and given a £20 store token to show gratitude and cover out of pocket expenses.

Study 2 Results

Scores for the state mood and alcohol use and are displayed in Table 2. The heavy Alcohol group drank significantly more units of alcohol in an average week $t(25) = -6.84$, $p < .001$ (Levene's test was significant so degrees of freedom have been adjusted). The heavy alcohol use group also had significantly higher scores on the AUDIT indicating an increased likelihood of hazardous drinking $t(40) = -4.31$, $p < .001$.

<<Insert Table 2 about here>>

Regarding Mood, there was a main effect of level of alcohol use on mood $F(3,38) = 1.87$, $p < .05$. This was due to significant univariate differences on the arousal subscale with heavy alcohol users reporting being significantly less aroused than the light alcohol users $F(1,40) = 4.49$, $p < .05$. Differences on the anxiety and depression subscales were non-significant ($p > .05$).

Standardised scores for the executive function measures are displayed in Table 3. MANOVA was used to analyse the executive function measures. Computation span level, switch cost, access scores (number of "S" letter words & number of 4-letter "C" words) and the three random letter generation scores (redundancy, number of repeat sequences, number of alphabetical sequences) were entered as dependent variables. The main effect of level of alcohol use on executive function was significant $F(7,33) = 2.08$, $p < .05$ (one-tailed). There were four significant univariate differences. On the Word Fluency-S letter, light alcohol users generated significantly more words than heavy users $F(1,39) = 8.56$, $p < .01$. Again, on the C-letter subscale, light users generated more words $F(1,39) = 3.34$, $p < .05$ (one-tailed). Heavy users also iterated significantly more repeat sequences on RLG, $F(1,39) = 4.23$, $p < .05$; differences on alphabetical sequences also approached significance, $F(1,39) = 2.64$, $p = 0.06$.

(one-tailed). The heavy users also had a significantly greater switch cost on the switching task $F(1,39) = 3.57, p < .05$ (one-tailed). Group differences on computation span and the redundancy measure of RLG were non-significant.

<<Insert Table 3 about here>>

Given the unequal gender distribution between the groups, Gender was included as a covariate in the MANOVA. Inclusion of gender reduced the multivariate main effect to below statistical significance $F(7,32) = 1.71, p > .05$. All of the significant group differences were slightly attenuated after inclusion of gender as a covariate. There were now three significant univariate differences. On the Word Fluency-S letter, light alcohol users generated significantly more words than heavy users $F(1,39) = 7.45, p < .01$. Again, on the C-letter subscale, light users generated more words $F(1,39) = 2.76, p < .05$ (one-tailed). Heavy users also iterated significantly more repeat sequences on RLG, $F(1,39) = 3.74, p < .05$ (one-tailed); differences on alphabetical sequences still approached significance after inclusion of gender as a covariate, $F(1,39) = 2.66, p = 0.06$ (one-tailed). Group differences in switching approached significance $F(1,39) = 2.62, p = 0.06$ (one-tailed). Group differences on computation span and the redundancy measure of RLG remained non-significant.

As there were significant group differences in state arousal, with heavy users reporting lower levels of arousal, it is conceivable that this may have affected their performance. Arousal was incorporated in to the analysis as a covariate. While the main effect of level of alcohol use on executive functions was slightly attenuated, $F(7,32) = 1.93, p < .05$ (one-tailed), in most cases, inclusion of arousal as a covariate increased the univariate F values. Group differences in S and C letter fluency remained significant $F(1,38) = 7.65, p < .01$ and $F(1,38) = 3.74, p < .05$ (one-tailed) respectively. Group differences in repeat sequences and alphabetical sequences were also both significant $F(1,38) = 5.05, p < .05$ and

$F(1,38) = 3.17, p < .05$ (one-tailed) respectively. Heavy alcohol users also had a higher switch cost $F(1,38) = 5.82, p < .05$. Group differences in redundancy and computation span remained non-significant after control for state arousal.

General Discussion

Study 1 found that relatively few studies had examined executive functioning in heavy social drinkers recruited from non-clinical populations. Meta-analysis showed that the studies we identified yielded only a small and non-significant mean weighted effect size. In study 2, we used the conceptual framework of Miyake et al. (2000) and found that heavy social drinkers displayed deficits in executive functioning relative to lighter drinking controls. Specifically, the heavy social drinkers performed worse on word fluency (access to semantic memory), aspects of random letter generation (inhibitory control), and had a greater switch cost on the number-letter task (switching). The results remained significant after controlling for group differences in state arousal. To the authors' knowledge this is the first paper to systematically investigate the effects of heavy social drinking on executive functions.

Firstly, heavy alcohol use adversely affected performance on the switching task. In previous research, heavy social drinkers were not impaired on the Wisconsin Card Sort Task (WCST) (Blume et al. 2000), while alcohol dependent participants were impaired in both the WCST and Trail making task (TMT) (Ratti et al., 2002). The greater switch cost in heavy users relative to light users suggests that the heavy alcohol use group are already exhibiting deficits in task switching, similar to those seen with continued heavy use. According to Miyake et al. (2000) both of these tasks would load on the switching component of the central executive. Given the recruitment of the anterior cingulate (Posner & Raichle, 1994) and bioccipital and parietal regions in task switching (Moulden et al. 1998), it seems reasonable to expect that heavy use of alcohol may be associated with damage to the anterior cingulate, and areas of the bioccipital and parietal areas. The heavy alcohol use group were

also impaired in access to semantic memory, and produced fewer words on a word fluency task; the deficit was most pronounced on the “S” letter category.

The group differences on aspects of Random Letter Generation (inhibitory control) are noteworthy for a number of reasons. First, this is a group of social drinkers, and while they have higher scores on the AUDIT, all were non-treatment seeking and were members of the undergraduate student population. Nonetheless, impaired inhibitory control has been cited as a contributory factor in the development of a substance use disorder (e.g. Wiers et al. 2007). Therefore it remains a possibility that the heavy alcohol use group are at increased risk of the development of an alcohol use disorder despite viewing their drinking habits as the “norm” for a student population. Secondly, the specific pattern of the subcomponents of random letter generation provide further support for the fractionation of the central executive and provide tentative support for the localisation of alcohol-related executive impairments. There were significant univariate effects of level of alcohol use on repeat and alphabet sequences, which Fisk and Sharp (2004) found both loaded on to the inhibition factor of the central executive. However redundancy was not significantly affected by heavy alcohol use. Fisk and Sharp found that redundancy loaded on to a different factor than alphabetic and repeat sequences, so in the present study the effects of heavy alcohol use on the generation of alphabetic and repeat sequences are likely to reflect impaired inhibitory control. Inhibitory control in general is linked to the prefrontal cortex (Casey et al. 1997; Kiefer et al. 1998), and Jahanshahi and Dirnberger (2009) have shown that the left dorsolateral prefrontal cortex is implicated in the generation of random responses. Thus the results of the present study provide support for the effects of heavy alcohol use on inhibitory control, and possible alcohol-related damage to areas of the PFC.

Despite the mean scores for weekly alcohol consumption being within the UK safe limits for the light alcohol group and on the borderline of “safe” for the heavy group, the heavy group exhibited deficits in executive functioning. The heavy alcohol group also had significantly higher scores on the AUDIT. Indeed, the mean AUDIT scores for both the light and heavy groups were over the threshold score of 8 for signs of hazardous drinking, while the heavy alcohol group were over the highest cut off point for alcohol related problems. According to Conigrave et al. (1995), in an inpatient setting such scores on the AUDIT can be indicative of social problems and problems with liver and gastrointestinal function. Given the significant differences in inhibitory control in the present study, which Wiers et al. (2007) suggest may be an important factor in control over drinking behaviours and the development of an AUD, the results are particularly worrying for the heavy alcohol group with high AUDIT scores. However, previous research has also shown that such high scores on the AUDIT are not atypical for student populations where heavy drinking can be the norm (e.g. Kypri et al. 2009), and as no participant reported currently or previously seeking help for problem drinking, it remains a possibility that such drinking habits may be transient and related to student life.

There were a number of limitations to the present study. Firstly we relied on self-report measures of alcohol consumption. It is possible that participants were unable to accurately recall the amount of alcohol that they had consumed recently, or were not truthful about their alcohol use. However, we have no reason to suspect that this was the case, as participants were not informed that they would be excluded from the study based on their responses. In addition, assignment to heavy or light alcohol use groups was performed by median split after all of the data had been collected, so participants would not have been influenced by experimenter beliefs about their level of alcohol consumption. It is also possible that the participants who were heavier/riskier drinkers had differences in their

prefrontal cortical functioning, and thus executive functions, prior to the onset of heavy drinking behaviours. Indeed Lyvers et al. (2011) found that heavy drinking and self-reports of executive function deficits were related to personality traits and genetic factors (e.g. familial alcoholism). Future research using laboratory tests should seek to clarify mediating factors such as this.

In conclusion, there is clearly much scope for further research regarding heavy social drinking and specific executive functions. We found that heavy social drinkers exhibited performance deficits in the executive functions of switching, inhibition and access to semantic memory. Although both groups reported drinking within the UK government's "safe" limits for alcohol consumption, the scores on the AUDIT suggested both groups exhibit hazardous drinking behaviour, especially the heavier alcohol use group. The heavy user group exhibit deficits similar to those seen with chronic heavy use. Future research should investigate the link between social drinking, executive function and control over drinking.

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* Indicates studies identified by the systematic review

Table 1

Summary of executive studies of alcohol users identified in this review

Authors/study, (country), & participants' mean (SD) ages if given	Sample details: Means (M) with (SDs) in brackets in most cases	Mean (SD) estimated Weekly alcohol consumption (UK units)	Executive tasks	Statistical controls for potential inter- group confounds	Relevant findings
<p>Crego et al. (2009) (Spain)</p> <p>Binge drinkers mean age = 18.9 years (0.5 years). Non-binge = 18.7 years (0.5 years).</p>	<p>95 1st year university students.</p> <p>42 binge drinkers (21 female). 53 non-binge (26 female)</p>	<p>Binge drinkers average 18.69 (12.17) drinks in last week. Non-binge average 2.79 (0.3) drinks in the last week.</p>	Visual pairs Continuous performance task (like spatial working memory task).	Gender was incorporated as an independent variable in ANOVA.	No significant effects found on this executive task.
<p>Hartley et al. (2004) (UK)</p> <p>Male non-drinkers = 20.3 (S.E.M. = \pm 0.6) years.</p> <p>Female non-drinkers = 21.4 (S.E.M. = \pm 0.4) years.</p> <p>Male binge drinkers = 21.8 (S.E.M. = \pm 0.3) years.</p> <p>Female binge drinkers = 21.0 (S.E.M. = \pm 0.8) years.</p>	<p>Student sample</p> <p>13 teetotallers (6 male) 14 Binge drinkers (9 male)</p>	<p>Teetotallers alc/week = 0</p> <p>Bingers units of alcohol/week = 22.0 (4.6) units for males and 15.6 (3.2) units for females.</p>	CANTAB SWM ID/ED shift Stockings of Cambridge	ANOVA for age, verbal IQ, BMI, cigarette consumption and alcohol consumption.	<p>SWM: females made more errors. No effects of binge drinking status.</p> <p>For ID/ED: Male bingers made fewer errors than male teetotallers in the ED shift.</p> <p>For SoC: significant effect on the initial planning time, with binge drinkers being significantly slower than teetotallers on two-, four and five-move problems.</p>

Authors/study, (country), & participants' mean (SD) ages if given	Sample details: Means (<i>M</i>) with (SDs) in brackets in most cases	Mean (SD) estimated Weekly alcohol consumption (UK units)	Executive tasks	Statistical controls for potential inter- group confounds	Relevant findings
<p>Heffernan et al. (2004)</p> <p>Excessive alcohol users: 21.4 years (5.33 years).</p> <p>Low dose or alcohol nonusers: 20.9 years (4.73 years).</p>	<p>Student sample: 40 excessive alcohol users, 40 low dose or alcohol nonusers.</p>	<p>Excessive alcohol users: 24.9 (6.53) units.</p> <p>Low dose or alcohol nonusers: 4.87 (4.20) units.</p>	<p>Dysexecutive questionnaire</p>	<p>ANOVAs for age, strategies to aid memory, and tobacco use.</p> <p>Tobacco use used as a covariate, based on a significant main effect for use across groups.</p>	<p>Excessive alcohol users reported a significantly higher number of problems than low dose or alcohol nonusers.</p>
<p>Nederkorn et al. (2009) (Netherlands)</p> <p>Overall sample age 21.05 years (2.31 years). Age data for groups not given.</p>	<p>Student sample.</p> <p>30 light social drinkers (16male)</p> <p>31 Heavy social drinkers (16 male)</p>	<p>Female light drinkers 6.1 (3.4) units/wk.</p> <p>Male light drinkers 5.1 (3.6) units/wk.</p> <p>Female heavy drinkers 17.4 (7.4) units/wk.</p> <p>Male heavy drinkers 30.7 (15.9) units/wk.</p>	<p>Stop Signal Reaction Time (inhibition)</p>	<p>Gender was incorporated as an independent variable in ANOVA.</p>	<p>No main effect of drinking group.</p> <p>Interaction between gender and drinking group indicating that heavy drinking women had poorer response inhibition.</p> <p>For errors, main effect of drinking group as heavy drinkers made more errors than light drinkers.</p>

Authors/study, (country), & participants' mean (SD) ages if given	Sample details: Means (<i>M</i>) with (SDs) in brackets in most cases	Mean (SD) estimated Weekly alcohol consumption (UK units)	Executive tasks	Statistical controls for potential inter- group confounds	Relevant findings
<p>Townshend & Duka (2001) (UK)</p> <p>Overall sample's mean age = 21.5 years (range 18 to 24 years). Age data for groups not given.</p>	<p>University students.</p> <p>16 Heavy Social drinkers</p> <p>16 Light social drinkers</p>	<p>Heavy social drinkers: mean 37.9 (12.2) units/week.</p> <p>Light Social drinkers: mean and SD not specified- but less than 3 units a week.</p>	<p>CANTAB pattern recognition</p> <p>CANTAB spatial recognition</p> <p>CANTAB ID/ED shift task.</p>	<p>Independent t-tests for group differences between light vs. Heavy. Then correlations with alcohol use.</p>	<p>All differences non-significant and no correlations with alcohol use.</p>
<p>Townshend & Duka (2005) (UK)</p> <p>Binge Drinkers: 20.9 years (2.6 years)</p> <p>Non-Binge Drinkers: 20.9 years (2.5 years)</p>	<p>Young social drinker sample:</p> <p>38 Binge Drinkers,</p> <p>34 Non-Binge drinkers.</p>	<p>Binge Drinkers: 33.3 (19) units.</p> <p>Non-Binge Drinkers: 20.5 (11.9) units.</p>	<p>CANTAB match to sample (MTS) visual search.</p> <p>CANTAB spatial working memory</p>	<p>Gender was incorporated into the group design, with further analyses if it interacted with drinking group. Inter-group demographic differences test by <i>t</i>-tests, with variables showing a significant difference then being used as covariates.</p>	<p>Binge drinkers faster in 8 pattern but not 4 pattern condition in MTS task.</p> <p>For SWM, no main effect of binge, but significant binge x gender interaction, with female binge drinkers making more errors than female non-binge drinkers. No inter-group effect for males.</p>

Authors/study, (country), & participants' mean (SD) ages if given	Sample details: Means (<i>M</i>) with (SDs) in brackets in most cases	Mean (SD) estimated Weekly alcohol consumption (UK units)	Executive tasks	Statistical controls for potential inter- group confounds	Relevant findings
<p>Piechatzek et al. (2009) (Germany).</p> <p>Overall sample age = 25.83 years (3.08 years).</p>	<p>Sample of 284 selected from a large epidemiological study of psychopathology development, according to their substance use history. Histories of psychiatric or neurological diagnoses were exclusion criteria.</p>	<p>Not given.</p>	<p>Stroop ID/ED Verbal fluency Non-verbal Fluency Stockings of Cambridge CANTAB SWM</p>	<p>Potential covariates (e.g. age, IQ) were included in regression models.</p>	<p>Alcohol intake was only significantly associated with strategy scores on the SWM task. Alcohol consumption was positively related to better use of strategies.</p>

Table 2

Scores on Background variables and standardised Executive Function Measures.

	Light Alcohol Group		Heavy Alcohol Group	
	Mean	SD	Mean	SD
Units of alcohol consumed in an average week.	6.43	4.74	26.57	13.29
AUDIT score	9.29	4.97	15.33	4.09
State Arousal	22.24	2.90	20.38	2.78
State Anxiety	11.00	2.35	11.95	1.80
State Depression	11.10	2.39	11.48	2.18

Table 3.

Standardised scores on executive function measures.

	Light Alcohol Group		Heavy Alcohol Group	
	Mean	SD	Mean	SD
Computation Span (updating)	0.05	0.96	-0.05	1.09
Word Fluency S-letter	0.43	0.88	-0.42	0.97
Word Fluency C-letter	0.25	0.92	-0.30	1.03
Number-Letter (Switching)	-0.27	0.69	0.31	1.21
RLG- redundancy	-0.08	0.94	0.09	0.77
RLG- alphabetical sequences	-0.19	0.52	0.20	0.96
RLG- repeat sequences	-0.22	0.73	0.23	0.67

Figure 1
The process of the literature search

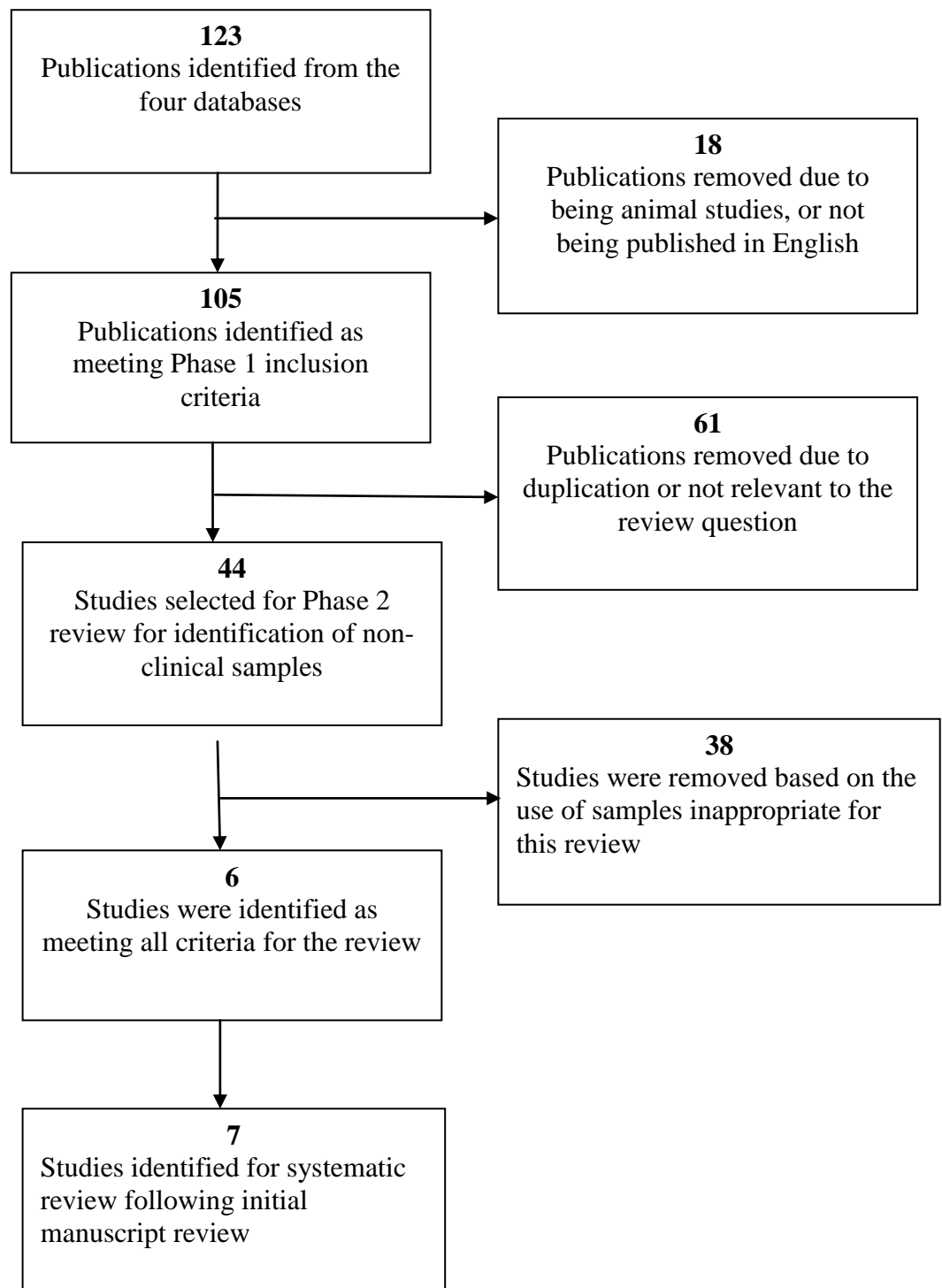


Figure 2

Forest plot for the meta-analysis

