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# Alcohol increases hypnotic susceptibility

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ABSTRACT

One approach to hypnosis suggests that for hypnotic experience to occur frontal lobe activity must be attenuated. For example, cold control theory posits that a lack of awareness of intentions is responsible for the experience of involuntariness and/or the subjective reality of hypnotic suggestions. The mid-dorso-lateral prefrontal cortex and the ACC are candidate regions for such awareness. Alcohol impairs frontal lobe executive function. This study examined whether alcohol affects hypnotisability. We administered 0.8 mg/kg of alcohol or a placebo to 32 medium susceptible participants. They were subsequently hypnotised and given hypnotic suggestions. All participants believed they had received some alcohol. Participants in the alcohol condition were more susceptible to hypnotic suggestions than participants in the placebo condition. Impaired frontal lobe activity facilitates hypnotic responding, which supports theories postulating that attenuation of executive function facilitates hypnotic response, and contradicts theories postulating that hypnotic response involves enhanced inhibitory, attentional or other executive function.

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## 1. Introduction

Hypnotic suggestions give rise to a wide range of interesting experiences and behaviours. Typically these involve a sense of involuntariness, such as when one's arm apparently rises by itself. Alternatively they may comprise the experience of an entirely convincing yet fabricated subjective reality, such as the experience of a mosquito on one's hand. While there may be different underlying mechanisms involved in different types of hypnotic suggestions, and individuals may create the experience in different ways (e.g. see Terhune, Cardeña, & Lindgren, 2011; Woody & Barnier, 2008), a number of general theories have been developed in an attempt to explain hypnotic phenomena. Hypnosis can be construed either as a special state or as a way of responding to suggestions (Kirsch et al., 2011). In terms of the latter, hypnotic responding is a way of respondingin which the sense of volition or reality has been deliberately distorted (whether or not one is in a special state). In terms of the former, it is a state that may facilitate such responding. Here we investigate the effect of a drug state on hypnotic response in order to test different theories of hypnosis.

Although several studies have examined the effects of drugs, including cannabis, psilocybin, diazepam and nitrous oxide on hypnotisability (Kelly, Fisher, & Kelly, 1978; Sjoberg & Hollister, 1965; Whalley & Brooks, 2009), surprisingly none has yet investigated the relationship of alcohol to hypnotic suggestibility. Yet, as we now describe, theories of hypnosis often postulate a role of the frontal lobes in hypnotic responding, and alcohol primarily disrupts frontal lobe functioning.

A number of theories have emphasised the role of the frontal cortex and associated executive functions, such as attention. One broad approach posits that hypnotic phenomena arise from a state of hypofrontality (see Dietrich, 2003) and diminished executive functions such as attention. For example, Woody and Bowers (1994) postulate that hypnotic induction leads to impairment of executive functions, causing actions to be controlled by contention scheduling (i.e. habit). Woody and Sadler

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(2008) review a number of ways in which executive control mechanisms may be disrupted in order to produce hypnotic response. Similarly, Gruzelier (1998, 2006) has proposed that hypnosis results from a state of frontal lobe exhaustion and diminished attentional abilities resulting from extreme concentration during hypnotic induction. Gruzelier and Warren (1993), Kallio, Revonsuo, Hämäläinen, Markela, and Gruzelier (2001), and Farvolden and Woody (2004), found that hypnotic induction reduced letter fluency in high rather than low hypnotisables, although similar effects were not detected on other frontal tasks. Thus, responding hypnotically may involve a specific form of hypofrontality. If these theories are true then alcohol should increase hypnotic responding.

Other theories would predict that the alcohol-induced frontal lobe impairment would reduce hypnotic responsiveness. The theories of both Spanos (e.g. 1986) and Hilgard (e.g. 1986) rely on the functioning of the frontal lobes for hypnotic response to be achieved. Spanos (e.g. Bertrand & Spanos, 1985; Spanos et al., 1982) has demonstrated that hypnotic behaviour can involve overcoming pre-potent responses, which necessarily involves executive functioning. Hilgard's theory relies upon two intact but dissociated executive functions. In fact, Hilgard (1986) argued that maintaining the two dissociated streams itself took executive capacity, because the hypnotic rather than non-hypnotic performance of one of two simultaneous tasks involved more dual task interference (see also Tobis & Kihlstrom, 2010; Wyzenbeek & Bryant, 2012). Similarly Crawford, Knebel, and Vendemia (1998) argue that frontal lobe executive functions are required for hypnotic analgesia. Therefore, since alcohol impairs executive function, alcohol should decrease hypnotic susceptibility by these approaches.

A more recent theory has highlighted the role of metacognition in hypnosis. The cold control theory of hypnosis (Dienes, 2012; Dienes and Perner, 2007; also see Barnier, Dienes, & Mitchell, 2009) explains hypnotic phenomena as the result of a strategic lack of awareness of the intention to perform a particular action. In other words, to respond hypnotically, the subject performs an action while thinking that they were not intending to perform that action: hypnosis essentially involves the lack of accurate higher order thoughts (HOTs) of intending. (Hence 'cold control': intentional control without HOTs.) Take, for example, the hypnotic suggestion that one's arm is stiff and rigid as if splinted, so that it cannot bend. In order to perform the suggestion successfully, the subject might intend to contract the antagonistic muscles of the arm simultaneously to prevent it from bending (about 80% of participants do try to bend, Comey & Kirsch, 1999). The hypnotic aspect is the experience of involuntariness, and cold control posits that this occurs by way of avoiding HOTs of intending, which thus lead to the inaccurate HOT, "my arm has become stiff and rigid by itself and I cannot bend it." Similarly, suggestions for analgesia or amnesia may involve distraction away from pain or the to-be-forgotten material. However, the hypnotic component is the ability to deceive oneself about having intended to do so; that is, by cold control theory this is done by avoiding accurate HOTs of intending. Note that on this theory hypnotic experience does not involve any alteration in first-order abilities (i.e. abilities with the function of dealing only with the world), but is achieved purely metacognitively. Thus, according to cold control, impairment of frontal function would enhance hypnotic response in virtue of the role of the frontal lobes in metacognition.

Higher order thoughts of seeing have been linked to the dorsolateral prefrontal cortex (DLPFC). Lau and Passingham (2006) using fMRI found that the brain region that distinguished reports of "seeing" rather than of "guessing" for equivalent perceptual discrimination was the DLPFC; thus, the DLPFC was not linked to the first order mental state of seeing, but to awareness of seeing. In another study, subjects' self-reported awareness of seeing was disrupted when theta burst TMS was applied to the area, even when first order perception was held constant with and without TMS (Rounis et al., 2010). That is, the disruption found was purely related to HOTs, and not first order perception. Fleming, Weil, Nagy, Dolan, and Rees (2010) also found the individual differences in the accuracy of higher order thoughts about perceiving correlated with grey and white matter volume in the same region.

The neural substrate of accurate higher order thoughts may well extend beyond the DLPFC. The monitoring and cognitive control functions of the anterior cingulate cortex (the ACC) make it a likely co-candidate region for the production of HOTs. Indeed, Woody and Szechtman (2011) found in highly hypnotisable participants that there were greater levels of activation in the ACC during auditory hallucination compared to imagination of the same sounds. That is, the ACC may be involved in determining whether internally generated sensory representations are just that – imagination – or else misrepresented as perceptions.

Alcohol impairs both the DLPFC (Wendt & Risberg, 2001) and the ACC (Ridderinkhof et al., 2002). Consistent with the claim these areas are involved in accurate metacognitive awareness, Sayette, Reichle, and Schooler (2009) found that alcohol compared to placebo decreased people's awareness that they were mind wandering. The DLPFC and ACC not only have executive monitoring functions but also control functions. Thus, the effect of alcohol on these areas can also be shown by the effect of alcohol on tasks that test inhibition of pre-potent response (like the Stop Signal Task, SST; Fillimore & Weafer, 2004), or the ability to resist perseveration (like the letter fluency task; Peterson, Rothfleisch, Zelazo, & Pihl, 1990). For example, Marinkovic, Rickenbacher, Azma, and Artsy (2012) reported reduced activation in ACC bilaterally during the incongruent condition on the colour Stroop task. Similarly, Gundersen, Specht, Grüner, Ersland, and Hugdah (2008) observed decreased activation in ACC and cerebellum during a working memory task following alcohol consumption. With a different variant of a working memory task, Paulus, Tapert, Pulido, and Schuckit (2006) found less activation in the DLPFC in participants who consumed alcohol rather than placebo.

In sum, alcohol impairs control and monitoring functions subserved by the DLPFC and ACC. According to theories postulating that hypnosis involves disruptions in executive control mechanisms (e.g. Woody and Sadler, 2008), alcohol should increase hypnotic responsiveness. According to cold control theory, as alcohol impairs the areas responsible for metacognitive monitoring, alcohol should make it harder to have accurate higher order thoughts of intending, and thereby facilitate hyp-

notic response. On the other hand, theories that emphasize that hypnotic response involves extra executive capacity (e.g. Hilgard, 1986) predict that alcohol should impair hypnotic responding.

It is well known that alcohol produces effects of an altered state of consciousness, i.e. drunkenness, and this may lead to increased expectations of hypnotic responding. Expectancy is a strong predictor of hypnotic response (Braffman & Kirsch, 1999), and indeed, according to response expectancy theory, is the final psychological mechanism by which hypnotic response is achieved (Kirsch, 1985). Theories that postulate some form of diminished frontal lobe functioning, such as cold control theory, predict that the effect of alcohol on hypnotic susceptibility will be observed above and beyond any effects of response expectancy.

The main aim of the study was to determine the effect of alcohol on hypnotic suggestibility, specifically measuring the central element to successful responding: the sensation that actions and experiences "happen by themselves". We administered real or placebo alcohol to medium susceptible participants before they received a series of hypnotic suggestions. (Note: although suggestions typical of susceptibility scales were used, we did not assess an individual's objective responsiveness with a pass/fail, but rather the rated feeling of automaticity, in order to increase sensitivity). Participants rated how strongly they experienced each suggestion. As a manipulation check, the effect of the alcohol on frontal function was determined by the letter fluency task and stop signal task. Before responding to hypnotic suggestions, participants also rated how strongly they expected to respond in order to control expectancy effects.

## 2. Methods

### 2.1. Participants

Participants in this study were 32 undergraduate and postgraduate students aged between 18 and 39 years ($M = 22$, $SD = 5.62$) recruited from the University of Sussex hypnosis screening database. Participants scored in the medium range (4–8 suggestions passed out of 12) on the Waterloo-Stanford Group Scale of Hypnotic Susceptibility, Form C (WSGC; Bowers, 1998). Medium-susceptible participants were selected in order to allow for either an increase or a decrease in hypnotic suggestibility. If alcohol were to decrease hypnotic suggestibility then we might see a floor effect in lows, and if it were to increase it, then we might see a ceiling effect in highs. For study inclusion, minimum alcohol consumption as assessed with the Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978) of 10 units per week and maximum alcohol consumption of 40 units per week was defined. (One unit equals 8 g ethanol.) All participants were in good health, had a body mass index of between 18 and 28, and were not pregnant or breastfeeding. Participants included were not heavy smokers (>20 cigarettes per day) and were able to abstain from smoking for the duration of the test session. Volunteers with current symptoms of mental illness or neurological disease, a history of severe mental illness, drug or alcohol abuse or altered metabolism of alcohol (e.g. impaired liver function or gastroenteritis) were excluded from the study. Ethical approval was received from the University of Sussex ethical committee. Informed consent was obtained from each participant before commencing with the study. All 32 participants completed the study (12 males). Participants were remunerated with course credits or £5 per hour for their participation in the study.

### 2.2. Design

Participants were randomly allocated to an alcohol or placebo condition according to a double blind between-subjects design and administered either an alcohol or placebo beverage. Participants were told that they were to receive a high or low dose of alcohol. The drinks were prepared by a laboratory assistant and administered to the participants by the researcher, who was blind to whether or not alcohol or placebo was being administered.

### 2.3. Materials

#### 2.3.1. REY Auditory Verbal Learning Test (RAVLT)

To assess whether the two groups differed in verbal learning and memory abilities, each participant completed the REY Auditory Verbal Learning Test (RAVLT; Rey, 1964), in which the experimenter read aloud 15 words at a rate of one per second. Participants were required to wait 2 min and then recall as many words from the list as possible.[1]

#### 2.3.2. Letter fluency task

Participants were asked to produce as many words as possible starting with the letters F and S (in a counterbalanced order across conditions) within 1 min. Proper nouns and variants of words already given counted as errors. This task was administered to assess the effects of alcohol on participants' monitoring function, which is required in this task to avoid perseverations. It was scored by subtracting the score of the post-drink test from the score of the pre-drink test to establish that alcohol had impaired the frontal lobe functioning of participants in the alcohol compared to placebo condition.

[1] The groups did not differ significantly on this task and thus this will not be discussed further.

*2.3.3. Drunkenness scale and VASs for alcohol effects*

These scales were used to measure the participants' general feelings of drunkenness and more specific experiences. The drunkenness scale requires participants to indicate how drunk they feel on a scale from 1 (I feel no effect of alcohol) to 9 (So drunk the room is spinning).[2] The Subjective Effects Visual Analogue Scales (VASs; Loeber & Duka, 2009) required participants to indicate the degree to which they experienced each of light-headedness, contentment, stimulation, pleasant glow, irritability, alertness and relaxation, by marking a corresponding line labeled from 1 (I feel no effect of alcohol) to 10 (I feel a strong alcohol effect).

*2.3.4. Hypnotic suggestions*

A total of nine hypnotic suggestions were made to participants following the second word fluency task, approximately 45 min after alcohol administration was finished. The suggestions covered both motor and cognitive types, as well as direct and challenge types (Barnier & Woody, 2008). The suggestions were (in order delivered): that they had a sour taste in their mouth; that their outstretched hands were attracted to each other, making them move together (magnetic hands); to feel that their outstretched right arm was weighed down by holding an imaginary heavy object that they could not keep it up (heavy arm); that a mosquito had landed on their hand and was tickling it; that their arm was so stiff and rigid that they could not bend it (rigid arm); to see two balls out of three placed in front of them (negative hallucination); that their arm was so heavy they could not lift it (arm immobilisation); to forget everything that had happened since they were hypnotised until told that they could remember (post-hypnotic amnesia) and to feel a strong urge to move seats when a clipboard was handed to them.

*2.3.5. Expectancy ratings*

Explicit expectancy ratings were recorded using E-Prime 2.0. Before each hypnotic suggestion was made, participants were asked to report whether or not they expected to experience the suggestion. For example, "If you were given a hypnotic suggestion that your arm would feel so heavy that you would not be able to hold it up, do you expect that your arm would feel heavier than normal." They responded by pressing 'Y' for yes and 'N' for no on a computer keyboard. They were then asked how confident they were about this expectancy (on a scale of 1–4[3]). Yes/no responses were combined with confidence ratings to give a directional "explicit expectancy" scale, ranging from −4 (indicating a strong expectancy not to respond to a suggestion) to +4 (indicating a strong expectancy that one would respond to the suggestion). Additionally, reaction times for yes/no responses were recorded and used as a measure of unconscious expectancy (when explicit ratings are partialled out).

*2.3.6. Subjective hypnotic response ratings*

Following each suggestion, participants were asked to rate how strongly they experienced the suggestion (on a scale of 0–5). For example "On a scale from 0 to 5, how strongly did you feel your hand becoming heavy (where 0 means you felt your arm was no more heavy than normal and 5 means you felt your arm becoming heavy as though you had a heavy object in your hand, pulling it down.).

*2.3.7. Stop Signal Task (SST)*

The SST was administered at the end of the session to check that participants were still generally influenced by alcohol, as response inhibition tends to be impaired under alcohol's influence (Loeber & Duka, 2009; Fillimore & Weafer, 2004). The SST, from the CANTAB (Cambridge Cognition, Cambridge, UK; http://www.camcog.com), assesses response inhibition performance. In each trial, an arrow (go-stimulus) was presented on the screen and the participant was required to press the left or the right button of a two-choice response box as quickly as possible to indicate if the arrow was either right-facing or left-facing. In 25% of the trials, an auditory stop signal (a beep sound) was presented at a variable delay after the go-stimulus. The subject was instructed to withhold their motor response on presentation of the stop signal. Five blocks of 64 trials were presented. The main variable was Stop Signal reaction time (SSRT) a measure of response inhibition (Robbins, 2007), which takes into account reaction time on go trials and is calculated from the length of time between the go stimulus and the stop stimulus when the participant is able to successfully inhibit his or her response in the latter 50% of trials. High SSRT indicates impaired inhibitory motor control.

*2.4. Procedure*

Participants were instructed not to drink alcohol for at least 12 h before the start of the test session. On the day of testing zero blood alcohol concentration was ensured before the start of testing. Baseline breath alcohol concentration was measured using a breathalyser (Lion Alcolmeter SD-400, Lion Laboratories Ltd., UK) and participants completed the Subjective Effects Visual Analogue Scale (VAS) and the drunkenness scale. The REY Auditory Verbal Learning Test (RAVLT; Rey, 1964) and letter fluency task were administered to control for any pre-manipulation differences between the two groups.

[2] 1, Feel no effect of alcohol; 2, Feel the first effects of alcohol; 3, Slightly tipsy; 4, Feeling warm; 5, a bit disinhibited; 6, Very merry; 7, Beginning to feel uncoordinated; 8, Very drunk; hard to focus properly; 9, So drunk the room is spinning and I feel sick.

[3] (1) I am completely guessing, I have no idea whether I would or wouldn't. (2) I am more or less guessing, but I have some feeling I was right. (3) I am pretty sure I am right. (4) I am completely certain.

Participants in the alcohol group were given an alcohol dose of 0.8 g/kg. For a 70 kg person this is equal to about 56 g of pure alcohol. This is equivalent to approximately 2.5 pints of lager or 5 glasses of wine (Weissenborn & Duka, 2003). The alcohol beverage consisted of 90% v/v alcohol diluted with tonic water (Schweppes® Indian Tonic Water) to make up a drink of 500 ml which was mixed with Angostura Bitter® to mask the taste of the alcohol. The placebo beverage consisted of 500 ml tonic water and Angostura Bitter® only. Drinks were divided into 10 portions, and participants consumed the ten portions at 3 min intervals in the presence of the experimenter. Participants were breathalysed 15 min after alcohol consumption and then completed another set of the Subjective Effects Visual Analogue Scales (VASs; Loeber and Duka (2009) and a 9 point drunkenness scale in order to obtain a subjective rating of how 'drunk' they felt. Participants were next given a brief hypnotic induction and nine suggestions. They were asked about their expectancy before each suggestion and subjective response after each suggestion. Following this they were breathalysed before completing the letter fluency task and finally the stop signal task.

## 3. Results

### 3.1. How blind were subjects to condition?

Subjects were told at the end of the experiment that there had been two conditions: alcohol and placebo. Subjects were then asked if they thought they had received alcohol; 86% thought they had. 73% of those in the placebo condition and 100% of those in the alcohol condition, $\chi^2(1) = 5.8$, $p = .08$). A *t*-test indicated that those who believed they had received alcohol rated their drunkenness higher ($M = 3.4$, $SD = 2.1$) than those who did not ($M = 0.25$, $SD = 0.5$), $t(30) = 2.98$, $p = .006$. Participants who believed they had received alcohol also rated their experience of "pleasant glow" higher than those who believe they had not ($M = 5.7$, $SD = 1.4$ and $M = 3.9$, $SD = 2.8$, respectively), $t(30) = 2.14$, $p = .002$. Further, when scores on the scales were averaged both before and after administration of alcohol or placebo, not only did alcohol change subjective feelings, from 2.82 (SD = 0.49) pre-administration to 5.04 (0.79), post administration, $t(13) = 7.17$, $p < .001$, $d = 3.37$, but so did the placebo, from 3.25 (0.75) to 3.81 (0.80), $t(13) = 3.25$, $p = .006$, $d = 0.72$. Nonetheless the change produced by alcohol (0.94, $SD = 1.04$) was detectably different from the change produced by placebo (0.29, $SD = 0.73$), $t(26) = 2.94$, $p = .035$, 1-tailed, $d = 0.45$. Participants who had alcohol reported feeling significantly more lightheaded, $t(30) = 3.21$, $p = .003$, $d = 1.13$ (see Table 1), and more intoxicated, $t(30) = 13.23$, $p < .001$, $d = 1.84$ (see Table 2) compared to those who had placebo.

In sum, while there was a placebo effect, subjects potentially also had some knowledge about condition, and thus it is important to control expectancies in determining alcohol's effect on hypnotic response.

### 3.2. Was enough alcohol administered to affect frontal lobe functioning?

Blood alcohol levels (BAC) at 45 min ranged from 0.55 *promille w/volume* to 0.96 *promille w/volume* in the alcohol group. No participant of the placebo group had a detectable BAC (derived from the breath alcohol level; BrAC) or BrAC.

As expected, alcohol impaired performance on tests of frontal lobe functioning. The alcohol group's decline in performance on the word fluency task ($M = 3.18$, $SD = 2.58$) was greater than the placebo group's ($M = 0.33$, $SD = 1.45$)

**Table 1**
Mean subjective mood ratings (SEM) after administration of alcohol or placebo.

| VAS | Placebo | Alcohol | |
|---|---|---|---|
| Drunkenness | 1.34 (.19) | 4.25 (.23)* | $t(30) = 13.23$, $p < .001$, $d = 2.59$ |
| Light-headed | 1.93 (.53) | 4.5 (.61)* | $t(30) = 3.21$, $p = .003$, $d = 1.12$ |
| Pleasant glow | 4.73 (.34) | 6.12 (.49) | $t(30) = 2.51$, $p = .018$, $d = 0.89$ |
| Irritable | 0.50 (.21) | 1.59 (.44) | $t(30) = 2.14$, $p = .040$, $d = 0.78$ |
| Relaxed | 5.73 (.41) | 6.15 (.29) | $t(30) = 0.83$, $p = .411$, $d = 0.30$ |
| Alert | 4.70 (.43) | 4.5 (.39) | $t(30) = 0.35$, $p = .733$, $d = 0.12$ |
| Stimulated | 4.73 (.27) | 4.91 (.46) | $t(30) = 0.32$, $p = .749$, $d = 0.12$ |
| Contented | 6.17 (.35) | 6.12 (.50) | $t(30) = .08$, $p = .938$, $d = 0.03$ |

* Significant after controlling for familywise error at the .05 level (Hochberg's, 1988 sequential Bonferroni).

**Table 2**
Mean scores on tests of frontal lobe function (SEM*).

| | Placebo | Alcohol | |
|---|---|---|---|
| Letter fluency pre | 15.733 (1.08) | 14.24 (1.01) | $t(30) = 1.01$, $p = .32$ |
| Letter fluency post | 15.53 (0.71) | 10.94 (0.65) | $t(30) = 4.75$, $p < .001$ |
| SST post | 161.09 (15.88) | 255.57 (29.82) | $t(30) = 2.69$, $p < .012$ |

* Standard error of the mean.

**Table 3**
Mean subjective responses to individual hypnotic suggestions (SEM).

| Suggestion | Placebo (SEM) | Alcohol (SEM) | |
|---|---|---|---|
| Rigid arm | 2.53 (.41) | 3.82 (.31) | $t(30) = 2.53$, $p = .017$ |
| Posthypnotic suggestion | 0.73 (.25) | 2.06 (.47) | $t(30) = 2.42$, $p = .022$ |
| Negative hallucination | 0.40 (.16) | 1.71 (.50) | $t(30) = 2.36$, $p = .025$ |
| Heavy arm | 3.73 (.37) | 4.53 (.15) | $t(30) = 2.08$, $p = .047$ |
| Arm immobilisation | 2.60 (.34) | 3.41 (.24) | $t(30) = 1.99$, $p = .056$ |
| Sour taste | 1.67 (.35) | 2.41 (.31) | $t(30) = 1.61$, $p = .119$ |
| Magnetic hands | 3.27 (.32) | 4.00 (.24) | $t(30) = 1.87$, $p = .172$ |
| Posthypnotic amnesia | 1.67 (.39) | 2.24 (.32) | $t(30) = 1.15$, $p = .259$ |
| Mosquito hallucination | 1.07 (.36) | 1.12 (.27) | $t(30) = 0.12$, $p = .91$ |

$t(30) = 2.69$, $p = .014$, $d = 0.87$. Kallio et al. (2004) found a hypnotic induction reduced letter fluency by 30%, similar to the 23% reduction alcohol produced in the current study (from 14.24 to 10.91): That is, we have administered sufficient alcohol to reduce frontal function by the order of magnitude relevant for effects on hypnotic response.

The alcohol group's reaction times ($M = 255.66$, $SD = 122.96$) on the stop signal task were also longer than those of participants in the placebo condition ($M = 161.09$, $SD = 61.5$, $t(30) = 2.69$, $p = .012$, $d = .97$, further indicating alcohol's effect on frontal functioning.

### 3.3. Did alcohol affect hypnotic response?

Table 3 shows the mean subjective response for each suggestion separately. When responses were averaged over suggestions, the alcohol group ($M = 2.81$, $SD = 0.71$) responded subjectively more to hypnotic suggestions than the placebo group placebo group ($M = 1.96$, $SD = 0.75$, $t(30) = 3.27$, $p = .003$, $d = 1.16$) (on a scale that went from 0 to 5). This is the key result of the study. An ANCOVA was performed on subjective hypnotic response between the alcohol group (adjusted $M = 2.70$, $SEM = .19$) and the placebo group (adjusted $M = 1.96$, $SEM = 0.19$) with the hypnotic suggestibility score (WGSC) as a covariate. The difference in hypnotic suggestibility remained significant, $F(1,27) = 7.81$, $p = .009$.

A significant positive correlation was found between expectancy and subjective response to suggestion overall, Pearson's $r = .55$, $p < .001$. So was the effect of alcohol just based on expectancy? The expectancy ratings of the alcohol group ($M = 0.56$, $SD = 0.91$) and placebo group ($M = 0.11$, $SD = 1.28$), did not differ significantly $t(30) = 1.16$, $p = .26$, $d = 0.40$, (on an expectancy scale that went from $-4$ to +4).[4] Crucially, when expectancy was put in as a covariate, the difference between the groups in hypnotic suggestibility remains (adjusted means: alcohol group $M = 2.73$, $SEM = 1.7$; placebo group $M = 2.03$, $SE = 1.6$), $t(29) = 3.57$, $p = .002$). A more formal mediation analysis looking at a range of possible mediators is conducted below.

### 3.4. Direct and indirect measures of expectation

Direct and indirect measures of expectancy were taken. Before each hypnotic suggestion was made, participants were asked to report whether or not they expected to experience the suggestion and how confident they were about this expectancy (on a scale of 1–4) as a direct measure, and the reaction time for the yes/no response was taken as an indirect measure. We would expect that subjects with more confidence in 'no' responses would be less hypnotically suggestible, and vice versa for 'yes' responses. In order to examine the effect of these direct and indirect measures of expectancy on subjective ratings (hypnotic response), for each subject a multiple regression was run with expectation RT and confidence rating simultaneously predicting subjective response for yes and no responses separately. Regression weights (betas) for yes and no should be in opposite directions, as the more confident a subject is in a no response, the less hypnotically responsive they should be, so we reversed the sign for 'no' expectancy response regression weights and averaged across both (for both indirect and direct measures). The mean standardised regression weight for confidence was 0.11, $t(24) = 1.00$, $p = .33$, $d = .51$, indicating that conscious expectancy did not significantly predict subjective response controlling for RT differences. However, the mean standardised regression weight for RT was 0.29, significantly above chance, $t(25) = 2.62$, $p = .015$, $d = .20$, indicating that RT as an indirect measure of expectancy did predict subjective response.

[4] In order to determine if this non-significant result was sensitive a Bayes Factor was used to compare the alternative hypothesis (that expectancy was higher for the alcohol rather than placebo group) to the null hypothesis. A Bayes Factor greater than 3 indicates strong evidence for the alternative over the null; less than a 1/3 indicates strong evidence for the null over the alternative; and anything in between indicates the data are insensitive (Dienes, 2011). First we need to specify what sizes of effect the alternative hypothesis predicts. The raw regression slope of hypnotic response on expectancy was 0.43 ($t = 3.58$, $p = .001$). The difference in hypnotic response between groups was 0.85 units. Thus the change in expectancy needed to produce the observed change in hypnotic response is 0.85/0.43 = 2.0 units. Thus, the predictions of the alternative hypothesis were modelled as a normal with a mean of 2.0 and standard deviation of 1.0 to indicate maximum vagueness in this estimate (see Dienes, 2011, Appendix, for this recommendation; and website for Dienes, 2008, for free online software for Bayes Factors). The Bayes Factor was 0.25; thus the non-significant difference in expectancy between groups is strong support of the null hypothesis. The ANCOVA also shows that expectation did not fully mediate the effect of group. The extent of mediation is more formally analysed below.

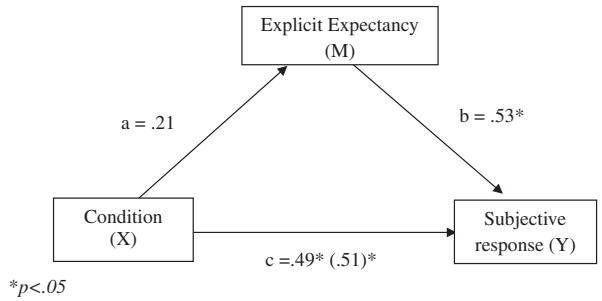


**Fig. 1.** Regression coefficients for the relationship of alcohol/placebo condition with subjective hypnotic response as mediated by explicit expectancy ratings.

### 3.5. Mediation analyses

Measures of explicit and unconscious expectancy were entered into separate mediation analyses to investigate the effect they had on the relationship between alcohol/placebo condition and subjective hypnotic response. In order to measure unconscious expectancies differences between groups, Go trial RTs from the SST were partialled out of 'yes' and 'no' expectancy RTs as a baseline measure of reaction time so as to account for any between-groups differences, such as alcohol causing longer latencies. 'Yes' and 'no' expectancy RTs were regressed separately on SST go trial RTs and the residuals were used as the measure of unconscious expectancies.

Fig. 1 displays mediation analysis for alcohol/placebo condition predicting hypnotic response with explicit expectancy as a mediator. The standardised regression weight predicting hypnotic response from condition partialling out expectancy is "*c*"; the correlation between condition and expectation is "*a*"; and the standardised regression weight predicting hypnotic response from expectancy, controlling condition is "*b*". If $c > 0$, then there is not full mediation; if $ab = 0$ there is no mediation; and if $ab > 0$, then there is some mediation (Woody, 2011). If there is full mediation by a variable, then $c = 0$[5] and if there is no mediation, then $ab = 0$ (see Woody, 2011). Conventional mediation analysis, being based on significance testing, does not provide a systematic method of establishing no meditation, as opposed to partial mediation by establishing if $ab = 0$. The amount of mediation depends on the size of ab. As $c > 0$ in all cases below, the only question is whether there is no mediation or partial mediation, and this depends on determining if $ab = 0$. Thus, we calculated a Bayes Factor, the only known method of determining degree of evidence for a point null hypothesis (Berger & Sellke, 1987).

As the partial correlations were close to the original correlations in each case, we normalised *a*, *b* and *c* with Fisher's *Z*, which has a known standard error. We can use the following formula for the standard error of *ab* in order to test if $ab = 0$ or if $ab > 0$:

$$SE_{ab} = \sqrt{a^2SEb^2 + b^2SEa^2}$$

To represent the prediction of partial mediation for the purposes of calculating a Bayes factor, we used a uniform distribution from zero to an upper limit of the correlation between condition and hypnotic response – because the most the mediated effect can be (*ab*) is the full effect. If the Bayes Factor is <1/3, then it is strong evidence for no mediation. If it is >3, it is strong evidence for partial mediation, and if it is anywhere in-between, then there is insufficient evidence. (Note a limitation of this method is that the distribution of *ab* is not normal, see Mackinnon & Fairchild, 2009; however we use the method here as an approximation so as to use the online Bayes calculator.[6]

As can be seen in Fig. 1, $c > 0$, therefore there is not full mediation. The Bayes Factor for the test of $ab > 0$ as opposed to $ab = 0$ is 0.78. Therefore, the evidence is simply insensitive for indicating whether or not there was partial mediation of the effect of condition on subjective response by explicit expectancy.

As can be seen in Fig. 2, $c > 0$, therefore there is not full mediation of 'no' expectancy RTs on the relationship between alcohol/placebo condition and subjective hypnotic response. The Bayes Factor for the test of $ab > 0$ as opposed to $ab = 0$ is 0.02. Therefore, there was evidence for no mediation by unconscious expectation not to respond to a suggestion.

As can be seen in Fig. 3, $c > 0$, therefore there is not full mediation. The Bayes Factor for the test of $ab > 0$ as opposed to $ab = 0$ is 0.08. Therefore, there was evidence for no mediation by unconscious expectation to respond to a suggestion.

As can be seen in Fig. 4, $c > 0$, therefore there is not full mediation. The Bayes Factor for the test of $ab > 0$ as opposed to $ab = 0$ is 0.13 Therefore, there was evidence for no mediation of condition on subjective response by SST no-go trial RTs (response inhibition), which were used as a measure of frontal lobe functioning.

[5] Therefore, we need to first of all check to see if *c* is significant. If it is not, (and *ab* is non-zero) then there may be partial or full mediation. A Bayes Factor can be used to test $c > 0$ by using an estimate of the full effect as an upper limit of the uniform, in a similar way as for testing *ab*.

[6] We checked the conclusions with a Bayes factor method that involves no violations of distributional assumptions, specifically by using the product of normals to represent the likelihoods, and all conclusions stand. This latter method will be described in a future publication. It is reassuring that they give similar answers.

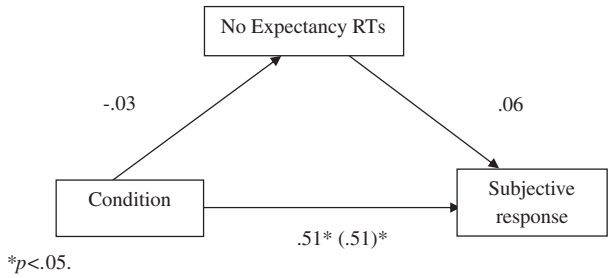


**Fig. 2.** The effect of condition (alcohol or placebo) on subjective hypnotic response with 'no' expectancy RTs as a single mediator.

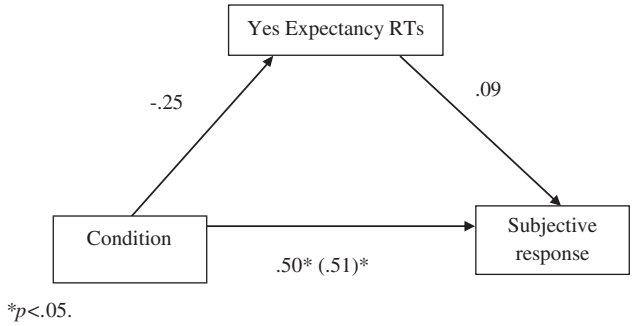


**Fig. 3.** Regression coefficients for the relationship of alcohol/placebo condition with subjective hypnotic response as mediated by 'yes' expectancy RTs.

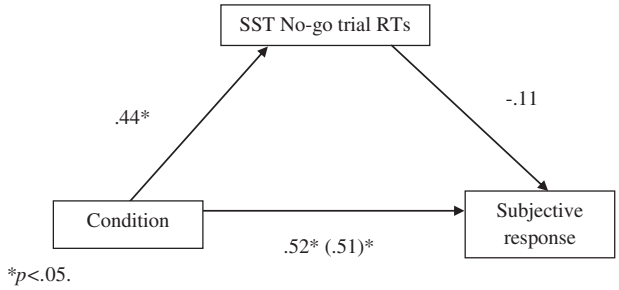


**Fig. 4.** Regression coefficients for the relationship of alcohol/placebo condition with subjective hypnotic response as mediated by SST no-go trial RTs.

## 4. Discussion

In this study we examined the effect of alcohol versus placebo on how compelling the hypnotic experience is. The placebo was convincing as a possible alcoholic drink, although there was a significant difference between the ratings of drunkenness reported between the two groups. The key result was that alcohol consumption increased hypnotic responsiveness compared to placebo. The results conceptually replicate those of Dienes and Hutton (2013) who showed that applying rTMS to the left DLPFC increased subjective ratings of hypnotic experience, compared to stimulation of the vertex.

While explicit expectancy strongly predicted performance, the effect of alcohol on hypnotic suggestibility remained after controlling for explicit expectancy. We measured unconscious as well as conscious expectancies for the first time, and showed that unconscious expectancy could predict hypnotic response above and beyond conscious expectancy. Yet the effect of alcohol could not be fully accounted for by either type of expectancy. Bayesian analysis indicated that while explicit expectancy might have partially mediated the relationship between alcohol condition and hypnotic responding, unconscious expectancies did not mediate at all.

The results also confirmed that alcohol impaired frontal lobe functions, as demonstrated by the alcohol group's decline in performance on the stop signal task and the letter fluency task, consistent with theories postulating a role for hypo- rather than hyper-frontality in hypnotic response. However, performance on the stop signal task did not mediate the relationship between alcohol condition and subjective hypnotic response, which is problematic for the original dissociated control theory (Woody & Bowers, 1994) and related neurophysiological approaches (Gruzelier, 1998; Gruzelier, 2006), which postulate a

generalized hypofrontality producing hypnotic response. However, these findings support theories postulating that only some aspect of diminished frontal lobe functioning is related to hypnotic suggestibility, such as the cold control theory (e.g. Dienes, 2012) and some types of dissociation theory (e.g. second order dissociative control theory; Woody & Sadler, 2008). For example, according to cold control theory, it is only the effect of alcohol on higher order thoughts of intending that is relevant to its effect on hypnotic response to the suggestions used, and not the effect of alcohol on the inhibitory processes measured by the stop–go task. Similarly, on second order dissociated control theory, it is specifically disruption in the monitoring of control processes that leads to hypnotic response, and there is no reason why the stop–go task would measure that aspect of alcohol's influence on the frontal system.

These findings do not, however, rule out the arguments suggesting that sufficient frontal lobe impairment should reduce hypnotisability. For example, the theories of both Spanos (1986) and Hilgard (1977) rely on intact executive functioning. Similarly, cold control does not postulate a state of utter hypofrontality; on this theory executive control is still implemented in order to carry out the cognitive or motor action performed. Thus, if executive function is impaired to the degree that the action could not be performed by executive control, then hypnotic suggestibility would be impaired (Dienes and Perner, 2007; contrast Woody & Bowers, 1994). For moving the hands together, or even imagining a mosquito, it is obvious people have sufficient frontal function after the amount of alcohol we administered to still intend to voluntarily perform these actions and successfully perform them. What is crucial for hypnotic suggestibility to be facilitated is that diminished frontal lobe functioning reduces concomitant higher order thoughts about intentions, allowing for HOTs of not intending to arise (Barnier et al., 2009). Indeed, it may be that performance on suggestions that heavily involve executive functions would be impaired following alcohol consumption (e.g. consider the inhibition of pre-potent responses hypnotically suggested in Bertrand & Spanos, 1985). For example, forgetting the number 4, which involves overcoming habit, may become more difficult according to cold control (Dienes and Perner, 2007) but not dissociated control theories (Woody & Bowers, 1994). We found that alcohol increased responsiveness to a negative hallucination suggestion. This suggestion involves the hypnotic subject avoiding the perception of a clearly visible object, a mental task that one would naturally assume to be an inhibitory task. The increase in the alcohol group's ability on this task despite a reduction in executive functioning suggests that responding to this suggestion does not involve above average inhibitory abilities (cf. Kirsch et al., 2011). It may be people attended away from the third ball without being aware of that intention (cold control) – the special ability of highs may not be in their ability to attend away, which may be normal (Dienes et al., 2009) but in their ability to not know that is what they were doing. We predict that once sufficient alcohol is administered to impair inhibition of prepotent responses under standard conditions, the corresponding response performed hypnotically will also be impaired.

Frontal lobe impairment may be just one of a number of ways of creating a hypnotic experience. Highly hypnotisable subjects differ in the way they create hypnotic experiences. For example, Barber's three-dimensional theory of hypnosis theory suggests that there are three types of hypnotisable subjects: those who are fantasy prone and spend much of their lives having "real-as-real" daydreams; amnesic subjects, who tend to forget life events and hypnotic experiences and subjects who are extremely motivated and have strong expectations about their ability to respond hypnotically (Barber, 1999). McConkey his colleagues (e.g. McConkey et al., 1989) have also identified two types of highly hypnotisables: those who actively construct hypnotic experience and those who are more passive, listening to the suggestions and waiting for the effects to happen to them. Similarly, Terhune et al. (2011; Terhune & Brugger, 2011) showed that highly hypnotisables can be separated into high and low dissociating groups which differ in their performance on executive tasks, and consequently in how they respond to suggestions. Future research could investigate the effect of alcohol on different types of highs.

Although alcohol particularly disrupts the DLPFC and ACC, it also affects a large area of the prefrontal cortex and beyond (Kähkönen, Wilenius, Nikulin, Ollinkainen, & Ilmoniemi, 2003) and so we cannot definitively conclude that the increase in hypnotic suggestibility was specifically due a reduction in metacognition, nor even specifically executive function. In future, specific disruption of DLPFC function could be coupled with a measure of accuracy of higher order thoughts as well as hypnotic responsiveness.

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