UvA-DARE (Digital Academic Repository)

In Vino Veritas? Alcohol, Response Inhibition and Lying

Suchotzki, K.; Crombez, G.; Debey, E.; van Oorsouw, K.; Verschuere, B.

[Link to publication](#)

Citation for published version (APA):

In Vino Veritas? Alcohol, Response Inhibition and Lying

Kristina Suchotzki[1,*], Geert Crombez[1], Evelyne Debey[1], Kim van Oorsouw[2] and Bruno Verschuere[1,2,3]

[1]Department of Experimental Clinical and Health Psychology, Ghent University, Henri Dunantlaan 2, 9000 Ghent, Belgium, [2]Faculty of Psychology and Neuroscience, Maastricht University, Universiteitssingel 40, 6229 ER Maastricht, The Netherlands and [3]Department of Clinical Psychology, University of Amsterdam, Weesperplein 4, 1018 XA Amsterdam, The Netherlands

*Corresponding author: Department of Experimental Clinical and Health Psychology, Ghent University, Henri Dunantlaan 2, B-9000 Ghent, Belgium. Tel.: +32-9-264-94-46; Fax: +32-9-264-64-89; E-mail: kristina.suchotzki@ugent.be

Abstract — **Aims:** Despite the widespread belief that alcohol makes the truth come out more easily, we know very little on how alcohol impacts deception. Given that alcohol impairs response inhibition, and that response inhibition may be critically involved in deception, we expected that alcohol intake would hamper lying. **Methods:** In total, 104 volunteers were tested at a science festival, where they had the opportunity to drink alcohol. Stop-Signal Reaction Times (SSRTs) served as operationalization of response inhibition. Differences in error rates and reaction times (RTs) between lying and truth telling served as indicators of the cognitive cost of lying. **Results:** Higher blood alcohol concentration was related to longer SSRTs, but unrelated to the cognitive costs of lying. **Conclusion:** This study validates previous laboratory research on alcohol and response inhibition in a realistic drinking environment, yet failed to find an effect of alcohol on lying. Implications of these findings and for the role of response inhibition in lying are discussed.

INTRODUCTION

'*In vino veritas*', 'Drunks and children always speak the truth' and 'Alcohol loosens the tongue' are only some expressions of the widespread belief that alcohol makes the truth come out more easily. Yet, there is nearly no research on the relationship between alcohol and lying, which is unexpected considering the substantial number of crimes committed by intoxicated offenders (Sigurdsson and Gudjonsson, 1994; Haggard-Grann *et al.*, 2006).

Theoretical support for the hypothesis that alcohol may hamper lying comes from research showing that alcohol hampers response inhibition. Response inhibition is most often defined as the intentional suppression of dominant, automatic or prepotent responses (Miyake *et al.*, 2000). Experimental laboratory studies have shown that moderate blood alcohol concentration (BAC; 0.04–0.08%) can impair performance in behavioral measures of response inhibition, such as the Stop-Signal task or the Go/No-Go task (Mulvihill *et al.*, 1997; Fillmore and Vogel-Sprott, 1999, 2000; de Wit *et al.*, 2000; Marczinski and Fillmore, 2003; Fillmore *et al.*, 2009; Anderson *et al.*, 2011; Tsujii *et al.*, 2011; Nikolaou *et al.*, 2013; for a review see Fillmore, 2007). Crucially, lying almost by definition involves the inhibition of the truth response. Prolonged reaction times (RTs) and an increased error rate (ER) for lying compared with truth telling have been interpreted as a cognitive cost of the conflict between the prepotent truth response and the deceptive response (Walczyk *et al.*, 2003; Spence *et al.*, 2008; Seymour and Schumacher, 2009; Verschuere and De Houwer, 2011). This claim has been further supported by research showing that lying is accompanied by increased activation in brain regions that are crucially involved in response inhibition tasks (e.g. the right inferior frontal gyrus; Spence *et al.*, 2001, 2008; Aron *et al.*, 2004, 2014; Christ *et al.*, 2009; Gamer, 2011; Vartanian *et al.*, 2013). As there are indications that the effect of alcohol on response inhibition might be mediated by the depressing effects of alcohol on neural activity in the right inferior frontal cortex (Tsujii *et al.*, 2011), one might hypothesize that alcohol intake not only interferes with response inhibition, but also with lying.

A contrasting prediction, namely that alcohol intake improves deception, can be derived from the findings of Karim *et al.* (2010). Inhibiting neuronal activity in the anterior prefrontal cortex (aPFC), a region that has previously been linked to moral cognition (Greene *et al.*, 2001; Moll *et al.*, 2002, 2005), facilitated lying as evidenced by shorter RTs and decreased skin-conductance responses. The authors also observed diminished feelings of guilt to deceive the interrogator after aPFC inhibition and proposed that the facilitation may be caused by a diminished experience of moral conflict. Alcohol impacts on multiple brain areas and has been observed to disinhibit 'immoral' behavior under certain conditions (Bond, 1998; Lyvers, 2000; Leeman *et al.*, 2009), and could therefore also facilitate lying.

There are only a few studies that investigated the impact of alcohol in a lie detection context. Bradley and Ainsworth (1984) studied the effects of alcohol intake on the psychophysiological detection of crime-related information. Alcohol intoxication (BACs around 0.12%) during a polygraph examination did not affect detection accuracy, but intoxication during a preceding mock crime decreased crime memory detection. Yet, O'Toole *et al.* (1994) were unable to replicate the latter finding. These two studies were the first to investigate the influence of alcohol in a forensic 'lie detection' context, but they speak more to the effect of alcohol on memory. More relevant for deception is a study by Kireev *et al.* (2008), in which participants performed the same deception paradigm twice, once sober and once after alcohol intake. In their paradigm, participants freely chose on each trial whether to respond truthfully or deceitfully (i.e. to indicate with one of two buttons correctly or incorrectly the directions of simple arrows) with the purpose to 'deceive' a computer. Results were mixed. RTs for lying were significantly longer than for truth telling in the sober condition, whereas this difference was not significant in the alcohol condition. Yet, neither RTs for truth telling nor RTs for lying differed significantly between the sober and the alcohol condition, and statistical information regarding the crucial interaction between lie/truth and intoxicated/sober was not reported. Using an event-related potential (ERP) measure, Kireev *et al.* (2008) also found a larger N190 for lying compared with truth telling in the sober condition,

but a reversed N190 effect in the alcohol condition. As the N190 is regarded as related to error perception ('error-related negativity'), this finding was taken as an indication that sober participants, but not intoxicated participants, perceived lying as an 'error'. These results fit with the results and the interpretation that alcohol may improve lying by reducing moral conflict (Karim *et al.*, 2010), but should be treated with caution. Although Kireev *et al.* (2008) compared a sober with an intoxicated condition, they did not find significant BAC differences between both conditions and did not report the respective mean BACs. Furthermore, the sample size was small (*n* = 13) and participants could freely choose between truth telling and lying so that there was no possibility to differentiate between intentional lies and behavioral errors.

The goal of the present study was to investigate the relationship between alcohol, response inhibition and lying. To that means, we chose a real-life drinking situation that enabled us to test a large number of volunteers with varying blood alcohol levels. The study therefore aimed not only to elucidate the relationship between alcohol and lying, but also to add to the alcohol and response inhibition literature by investigating in a large sample whether the effects of controlled alcohol intake in laboratory settings generalize to real-life drinking environments, in which participants freely determine their drinking behavior. Response inhibition in our study was measured as the estimated time of stopping a prepotent go-response (SSRT) in the Stop-Signal Task (Vince, 1948; Lappin and Eriksen, 1966; Logan and Cowan, 1984). Lying was measured with the Sheffield Lie Test (Spence *et al.*, 2001; based on the Differentiation of Deception paradigm, Furedy *et al.*, 1988). In this paradigm, one typically observes an enhanced ER and prolonged RTs for lying compared with truth telling. These lie effects ($ER_{lying} - ER_{truth\ telling}$; $RT_{lying} - RT_{truth\ telling}$) were taken as indication of the cognitive cost of lying (Spence *et al.*, 2001; Fullam *et al.*, 2009; Farrow *et al.*, 2010; Verschuere *et al.*, 2011; Debey *et al.*, 2012; Hu *et al.*, 2012; Van Bockstaele *et al.*, 2012). Based on previous laboratory research showing that alcohol impairs response inhibition, we expected higher BACs to be related to longer SSRTs. Based on previous research showing that lying comes at a cognitive cost, we expected to replicate both lie effects (in ER and RTs). Based on the research that implies a crucial role of response inhibition in deception, we expected higher BACs to be related to an increased cognitive cost of lying (i.e. larger ER and RT lie effects). As also habitual alcohol use was found to be associated with impairments in stop-signal performance (Nigg *et al.*, 2006; Lawrence *et al.*, 2009), we included an assessment of problematic drinking behavior (AUDIT). Considering the substantial overlap of the concepts of response inhibition and impulsivity as well as findings that increased impulsivity is implicated in the development and maintenance of substance abuse disorders (de Wit, 2009), we also included a measurement of trait impulsivity (BIS-11).

METHOD

Participants

In total, 104 visitors of the science festival Discovery Day 2012 volunteered to participate in the study. The study was approved by the ethical committee of Maastricht University and all participants provided written informed consent. Data of participants were excluded from data-analyses when participants had reported drug and/or medication use (*n* = 14). Furthermore, we excluded data of participants that exceeded the mean ER plus 2.5 standard deviations in the Stop-Signal Task or the Sheffield Lie Test (*n* = 2). The mean age and gender of the remaining 88 participants can be found in Table 2.

Procedure

Testing took place at two locations of the festival (Rotterdam and Amsterdam) from 9.00 PM to 3.00 AM. The study was advertised as investigating the relation between alcohol and lying, and had been announced on national radio earlier that day. Following the advice of the ethical committee, everyone interested in the study could participate and participants were not selected on the basis of their alcohol consumption. Participants were not encouraged to drink alcohol.

Participants filled out a questionnaire assessing demographic variables (gender and age), feelings of tension, anxiety, intoxication, tiredness and concentration (1–10 Likert scales), drinking behavior on that day (number of alcoholic consumptions and drinking time) and drugs or medication use on that day. Trait impulsivity was assessed with the Barratt Impulsiveness Scale (BIS-11; Patton and Stanford, 1995) and habitual alcohol use was assessed with the Alcohol Use Disorders Identification Test (AUDIT). Testing took place on four computers, which allowed simultaneous testing of four participants. In each location, three experimenters conducted the study. For every participant, the time of testing was noted in order to control for it in statistical analyses as a potential confound. Participants were not allowed to drink during the experiment to ensure a minimum of 15 min (i.e. the duration of both tasks) between the last alcoholic drink and the alcohol test. Everyone first executed the Stop-Signal Task and then the Sheffield Lie Test. Finally, participants were asked to drink a sip of water and BAC was measured with the Dräger Alcotest 6510. The Dräger Alcotest 6510 converts the breath alcohol ratio into blood alcohol concentration (BAC in %). Finally, participants were told their BAC values. If participants were severely intoxicated, they were warned about the consequences of severe alcohol intake and they were advised to stop drinking. Participants were thoroughly debriefed about the purpose and the background of the experiment and received a handout with information and contact details of the experimenter in case they had any further questions.

Stop-signal task

The Stop-Signal Task was programmed and presented with Tscope, a C/C++ library (Stevens *et al.*, 2006). During the task, two types of stimuli (an 'X' or 'O') were presented in white in the center of a black screen. Participants were instructed to indicate with left and right button presses which of the two stimuli they saw ('z' (left) and '/' (right) on a standard QWERTY keyboard). Stimuli and response mappings were counterbalanced across participants. The response deadline was 2000 ms and the inter-trial interval was 300 ms. On 75% of the trials, participants simply had to perform the binary decision as fast as possible (go-trials). Crucially, on 25% of the trials, a signal (a 1000 Hz tone) was presented for 100 ms via a headphone, indicating that participants should try to stop their response. The time interval between the stimulus and the stop-signal (stop-signal delay, or SSD) was

initially set to 250 ms, but adjusted on a trial-to-trial basis. After a successful stop, it was increased by 50 ms, after a failure to stop it was decreased by 50 ms. The test phase consisted of two blocks of 80 trials, with 20 stop trials each (160 trials in total, including 40 stop trials). Test blocks were separated by a self-paced break. As a measure of response inhibition, we calculated the SSRT by subtracting the mean SSD from the mean RT on go-signal trials (Verbruggen *et al.*, 2008). The SSRT is a well-validated measure of response inhibition ability (for reviews see Logan, 1994; Boucher *et al.*, 2007; Verbruggen and Logan, 2008).

Before the actual test, participants practiced the task. In a first practice phase, consisting of eight trials, participants practiced the go-response while ignoring the stop-signals. In a second phase, consisting of 16 trials, participants practiced to inhibit their response on 4 stop-signal trials.

Sheffield lie test

The Sheffield Lie Test was presented with Inquisit 3.0.1. In the Sheffield Lie Test, participants have to answer Yes/No questions both truthfully and deceptively, depending on a color cue. Thirty questions (15 with 'yes' and 15 with 'no' as correct response) were presented verbally via headphones, in random order. For example: 'Is Amsterdam in the Netherlands?',

'Is Amsterdam in Switzerland?'. All questions can be found in Table 1. Each question was presented four times, and had to be answered twice truthfully and twice deceptively. Reminder labels for the possible responses ('Yes'/'No') appeared on the left and right lower part of a black screen and responses had to be given with left and right button presses ('z' (left) and '/' (right) on a standard QWERTY keyboard). The response labels were presented in yellow or blue, and participants were instructed that one color required a truthful response, whereas the other required a lie. The position of the reminder labels and color-assignment were counterbalanced across participants. Participants were instructed to respond as fast as possible. If participants did not respond after 6000 ms, the labels disappeared and the words 'Too slow' were presented centrally on the screen. The inter-trial interval was 200 ms. The test phase consisted of two blocks, with 60 trials each (120 trials in total, including 60 truth and 60 lie trials). Test blocks were separated by a self-paced break. As measure of the cognitive costs of lying, we calculated the ER and RT lie effects by subtracting the mean of the truth telling condition from the mean of the lying condition ($ER_{lying} - ER_{truth\ telling}$; $RT_{lying} - RT_{truth\ telling}$).

Before the actual test, participants practiced the task with 12 different questions. Only during the practice phase, participants received additional feedback after each trial on the correctness of their response.

Table 1. Questions used in the Sheffield Lie Test (translated from Dutch)

Questions requiring 'yes' as correct response	Questions requiring 'no' as correct response
Is water wet?	Is water dry?
Is ice cold?	Is ice warm?
Can birds fly?	Can pigs fly?
Is a crocodile an animal?	Is a computer an animal?
Is Amsterdam in the Netherlands?	Is Amsterdam in Switzerland?
Are giants big?	Are giants small?
Do cars have four wheels?	Do cars have six wheels?
Is an igloo made of ice?	Is an igloo made of stone?
Is sausage meat?	Is salad meat?
Is stone hard?	Is stone soft?
Is fire warm?	Is fire wet?
Is milk white?	Is milk green?
Are bananas yellow?	Are bananas red?
Is grass green?	Is grass blue?
Does a butcher sell meat?	Does a butcher sell bread?

RESULTS

Descriptives

As can be seen in Fig. 1, the distribution of BAC was positively skewed with an overrepresentation of BAC = 0.00% ($n = 31$; $z_{skewness} = 5.93$, $P < 0.001$; $z_{kurtosis} = 4.30$, $P < 0.001$). BACs ranged between 0.00 and 0.15%, with an average BAC of 0.03% (SD = 0.03; Mdn = 0.02). Means and standard deviations of all other assessed variables can be found in Table 2.

Preliminary analysis and manipulation check

Paired sample *t*-tests confirmed that lying ($M = 10.34\%$, SD = 7.43) was associated with a higher ER than truth telling ($M = 6.79\%$, SD = 5.45), $t(87) = 5.48$, $P < 0.001$, $d = 0.58$. [For group comparisons, the standardized mean difference *d*
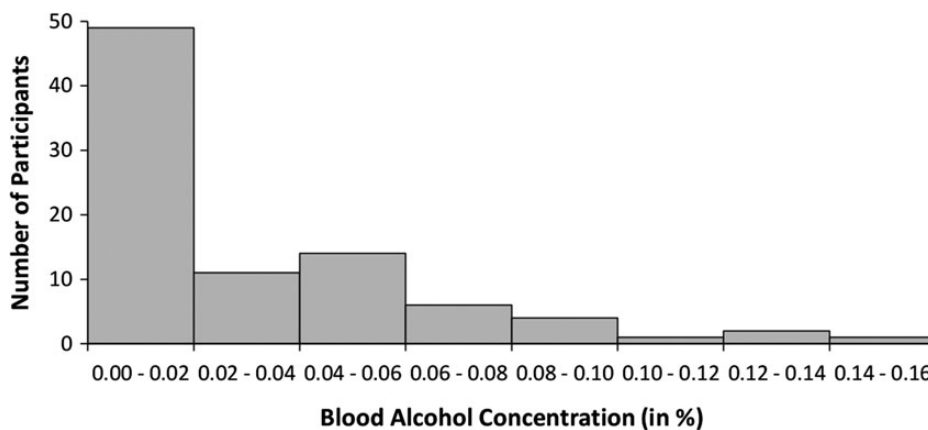

Fig. 1. Distribution of the blood alcohol concentration (in %) in our sample ($n = 88$).

Table 2. Means, standard deviations and correlations (r_s) with BAC

Measure	M	SD	BAC
BAC	0.03	0.03	–
Gender	0.50	0.50	−0.14
Age	28.02	6.24	0.05
Time of testing	230.75	105.20	0.61***
Tension	3.16	2.14	−0.03
Anxiety	1.62	0.80	−0.12
Tiredness	5.05	2.02	−0.10
Concentration	5.38	2.06	−0.11
BIS-11	53.95	9.16	0.11
AUDIT	9.73	4.80	0.53***
Manipulation checks			
Intoxication	3.51	2.22	0.74***
No. consumptions	3.60	2.92	0.81***
Drinking time	2.77	2.40	0.75***

Gender = % female; Time of testing = Time of testing in minutes after 20 h; No. consumptions = Number of alcohol consumptions; Drinking time = Drinking time in hours. *P*-values reported two-tailed.
***$P < 0.001$.

was calculated as measure of effect size, with 0.20, 0.50 and 0.80 as thresholds for 'small', 'moderate' and 'large' effects (Cohen, 1988). When computing *d* for dependent samples, we corrected *d* for inter-correlations (Dunlap et al., 1996; Morris and DeShon, 2002)] After removal of error trials and RT outliers (0.02%; RTs >2.5 SDs from the mean per subject and condition), paired sample *t*-tests confirmed that lying ($M = 3315$ ms, $SD = 326$) was associated with longer RTs compared with truth telling ($M = 3149$ ms, $SD = 293$), $t(87) = 9.19$, $P < 0.001$, $d = 0.98$. Means and standard deviations of SSRT, ER lie effect and RT lie effect can be found in Table 3.

As manipulation check, we computed the correlation between BAC and the feeling of intoxication, the number of alcohol consumptions and the drinking time. Because BAC was not normally distributed, we used Spearman's rho (r_s) as correlation coefficient in all further analyses. Note that r_s also serves as effect size, with 0.10, 0.30 and 0.50 as thresholds for 'small', 'moderate' and 'large' effects. As can be seen in Table 2, higher levels of BAC were related to a higher feeling of intoxication, a larger number of reported alcoholic consumptions and a longer drinking time.

To discriminate between effects of acute alcohol consumption, impulsivity and habitual alcohol use, and to check for possible other confounding variables, we also computed the correlations between BAC and gender, age, time of testing, feelings of tension, anxiety, tiredness, and concentration, the BIS-11, and the AUDIT. As can be seen in Table 2, higher

levels of BAC were related to a later time of testing and a stronger habitual alcohol use. We will therefore control for these factors in our dimensional analyses.

Dimensional analyses

To investigate the link between BAC, response inhibition and the cognitive cost of lying, we computed the correlations between BAC, SSRT, ER and RT lie effect. As can be seen in Table 3, higher levels of BAC were related to higher SSRTs, whereas the correlations with the lie effects were not significant.

To control for the influence of the time of testing on the SSRT scores, we checked whether the time of testing was correlated with any of the feelings during the testing and computed the nonparametric partial correlation between BAC and SSRT. Results revealed that time of testing was only related to the feeling of intoxication, $r_s = 0.58$, $P < 0.001$, but not to any other feeling, all $Ps > 0.15$. The BAC-SSRT relation was still marginally significant after controlling for the time of testing, $r = 0.20$, $P = 0.07$. As higher SSRT scores were related not only to higher BAC levels but also to higher AUDIT scores, we also computed the nonparametric partial correlation between BAC and SSRT to examine whether acute alcohol effects (BAC) were carried by effects of habitual alcohol use (AUDIT). The BAC-SSRT relation remained marginally significant after controlling for the AUDIT scores, $r = 0.21$, $P = 0.07$. A multiple linear regression analysis with BAC predicting SSRT also revealed no significant increase in the prediction when adding AUDIT and BAC × AUDIT to the model. Intercorrelations of all assessed variables can be found in Table S1 of the online Supplementary material.

Categorical analyses

To enable a better comparison of our results with previous research that compared groups of participants that received different doses of alcohol with sober controls, we categorized participants according to their BAC levels. As previous research found effects of alcohol on response inhibition from 0.04% on (Mulvihill et al., 1997; Fillmore and Vogel-Sprott, 1999, 2000; de Wit et al., 2000; Marczinski and Fillmore, 2003; Fillmore, 2007; Fillmore et al., 2009; Anderson et al., 2011; Tsujii et al., 2011; Nikolaou et al., 2013), participants with an alcohol level below 0.04% were categorized as sober controls ($n = 60$), whereas participants with an alcohol level of 0.04% and above were categorized as intoxicated ($n = 28$).

As can be seen in Table 4, independent-sample *t*-tests revealed a significantly longer SSRT for the intoxicated group

Table 3. Means, standard deviations and intercorrelations (r_s) of BAC, dependent variables, time of testing, BIS-11, and AUDIT

Measure	M	SD	BAC	SSRT	ER lie effect	RT lie effect	Time of testing	BIS-11	AUDIT
BAC	0.03	0.03	–						
SSRT	296.29	143.15	0.35**	–					
ER lie effect	3.55	6.08	0.07	0.02	–				
RT lie effect	166.51	170.04	0.08	0.04	0.11	–			
Time of testing	230.75	105.20	0.61***	0.34**	0.10	0.11	–		
BIS-11	53.95	9.16	0.11	0.07	0.12	0.01	0.12	–	
AUDIT	9.73	4.80	0.53***	0.24*	0.17	−0.03	0.27*	0.11	–

Time of testing = Time of testing in minutes after 20 h. *P*-values reported two-tailed.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.

Table 4. Means and standard deviations of different variables for the sober and the intoxicated group and results of the independent *t*-tests

Measure	Sober		Intoxicated				
	M	SD	*M*	SD	*t*	*df*	*P*
BAC	0.01	0.01	0.07	0.03	9.82	30.78	<0.001
Gender	55	0.50	39	0.50	1.89[a]	1[a]	0.17[a]
Age	28.63	7.19	26.70	3.04	1.74	83.66	0.09
Time of testing	203.05	97.01	290.10	98.67	3.90	86	<0.001
Tension	3.08	1.93	3.32	2.57	0.48	86	0.63
Anxiety	1.73	0.89	1.38	0.50	2.22	76.76	<0.05
Tiredness	5.22	1.96	4.69	2.15	1.09	79	0.28
Concentration	5.51	2.12	5.12	1.95	0.80	79	0.43
BIS-11	53.32	8.71	55.40	10.16	0.95	80	0.35
AUDIT	8.69	4.73	12.35	3.96	3.28	79	<0.01
Manipulation checks							
Intoxication	2.53	1.71	5.58	1.70	7.51	79	<0.001
No. consumptions	2.26	2.21	6.38	2.17	7.87	78	<0.001
Drinking time	2.00	2.37	4.38	1.53	5.45	71.41	<0.001
Dependent measures							
SSRT	262.99	99.21	366.47	191.31	2.70	34.07	<0.05
ER lie effect	3.18	5.81	4.34	6.66	0.83	86	0.41
RT lie effect	152.54	168.56	196.47	172.39	1.13	86	0.26

Gender = % female; Time of testing = Time of testing in minutes after 20 h; No. Consumptions = Number of alcohol consumptions. Drinking time = Drinking time in hours. Degrees of freedom are corrected when equality of variances was rejected and differ between measures due to missing values. *P*-values reported two-tailed.
[a]As gender is a categorical variable, Pearson's chi-squared (χ^2) test was used.

compared with the sober control group, $t(34.07) = 2.70$, $P < 0.05$, $d = 0.76$. There were no significant group differences in the ER lie effect, $t(86) = 0.83$, $P = 0.41$, $d = 0.19$, or the RT lie effect $t(86) = 1.13$, $P = 0.26$, $d = 0.26$.

DISCUSSION

In order to investigate the relation between alcohol consumption, response inhibition and lying, the current study was conducted at a science festival where visitors voluntarily consumed alcohol. Such a naturalistic setting comes at the cost of experimental control, but it enabled us to recruit a large number of volunteers with varying blood alcohol levels, without actively administering alcohol to participants or encouraging alcohol consumption. Furthermore, our study complemented and extended previous laboratory research by demonstrating the generalization of alcohol and lie-effects to more realistic samples and settings.

Results of both the dimensional and the categorical analyses revealed that alcohol intake was associated with impaired response inhibition. Our findings thereby validate previous laboratory research that found impaired response inhibition performances after alcohol intake (Mulvihill *et al.*, 1997; Fillmore and Vogel-Sprott, 1999, 2000; de Wit *et al.*, 2000; Nikolaou *et al.*, 2013). This is important as alcohol intake in a laboratory environment differs from realistic drinking environments in many aspects (e.g. instructed vs. spontaneous alcohol consumption, different environmental cues, social factors and reinforcing effects of alcohol intake). Accordingly, a meta-analysis revealed that both pharmacological and expectancy effects of alcohol intake were significantly moderated by the experimental setting (experimental vs. natural vs. bar setting; McKay and Schare, 1999), stressing the need for demonstrations of experimental effects in more realistic environments. The finding that alcohol effects on response inhibition transfer to realistic drinking environments is also relevant for forensic and clinical contexts, as impaired response inhibition has been linked to aggressive behavior and psychological disorders, such as anti-social personality, obsessive-compulsive and attention deficit/hyperactivity disorders (ADHD; Schachar and Logan, 1990; Schachar *et al.*, 1993; Oosterlaan and Sergeant, 1996; Pawliczek *et al.*, 2013).

In line with theories stating that habitual alcohol use is related to poor response inhibition capacities, either by facilitating the development of a dependency or as consequence of long alcohol abuse (Jentsch and Taylor, 1999; Nigg *et al.*, 2006; de Wit, 2009; Lawrence *et al.*, 2009; Courtney *et al.*, 2013), we also found that stronger habitual alcohol use was related to a worse performance in the Stop-Signal task. Our design does not allow to disentangle acute alcohol effects and habitual alcohol use. Yet, the observation that the correlation between BAC and SSRT was still marginally significant when controlling for the AUDIT scores indicates that the observed response inhibition impairments cannot fully be attributed to habitual alcohol use. We also did not observe an association between impulsivity and response inhibition (Reynolds *et al.*, 2006; Dougherty *et al.*, 2008; Caswell *et al.*, 2013), which further suggests that it was the acute alcohol intake that impaired response inhibition in our sample.

Extending previous laboratory research on lying, we replicated the finding of an increased cognitive cost of lying in our sample (Seymour *et al.*, 2000; Walczyk *et al.*, 2003; Spence *et al.*, 2008; Verschuere and De Houwer, 2011). This is important considering the need for more ecologically valid settings in forensic research (National Research Council, 2003; Evans *et al.*, 2009). However, in contrast to our expectations, alcohol consumption was not related to the cognitive cost of lying. To interpret this finding, we have to evaluate whether our null finding may be due to a lack of power. As there is no comparable research to estimate the size of our expected effect of alcohol on the cognitive cost of lying, we used the medium-

sized correlation between the BACs and SSRTs in our sample ($r_s = 0.35$) as an estimate. Assuming the expected relationship in our sample between BACs and lying to be comparable in strength to the relationship between BACs and SSRTs, our experiment had a power of 0.93 to discover this relation. Although we cannot exclude that the size of the actual relation may be lower (e.g. as response inhibition may only be one component influencing the variance of the lie effect), we can deduct that we had reasonable power to detect a medium size effect. Another factor may be the underrepresentation of severe intoxication levels in our sample. Because of ethical reasons, every festival visitor who wanted to participate was included in the study and we did not encourage participants to drink. Although we did find an effect of alcohol on response inhibition and other research has shown that response inhibition is impaired already from moderate intoxication levels on (from 0.04%), it could be that lying is only impaired at higher alcohol levels.

It is possible that hampering effects of alcohol on lying were counteracted by other factors in our experiment. Importantly, motivational effects may have neutralized alcohol effects. It has been shown that alcohol-related impairments can be reduced when inhibition is reinforced and participants are highly motivated (Fillmore and Vogel-Sprott, 1999, 2000; Vogel-Sprott *et al.*, 2001). Advertising our study as investigating the relation of alcohol and lying, we approached participants with the question whether they wanted to find out how well they could lie. Participants also received feedback at the end of the experiment on their 'lying performance' (based on their RT lie effect). Such particular motivation may have neutralized alcohol effects. Finally, it could also be the case that alcohol intake did hamper lying in our experiment, but at the same time facilitated it by decreasing moral conflict (Kireev *et al.*, 2008; Karim *et al.*, 2010). Sober participants may have experienced a stronger moral conflict than participants who were under the influence of alcohol and these two antagonistic effects might have counteracted each other. In that context, it may be interesting to investigate whether the use of more personal, emotionally arousing questions (e.g. Did you ever take drugs? Did you ever cheat?) would change the pattern of results. First, sober participants may experience a higher moral conflict when lying about personal, emotionally arousing questions, compared with when lying about neutral questions. Second, if alcohol intoxication reduces this moral conflict, one may observe a significant facilitation of lying for personal, emotionally arousing questions for intoxicated participants, compared with sober participants.

The present data do not support the role of response inhibition in lying. There was no association between response inhibition and lying, and alcohol did not impact on lying. As such our study may also question the role of response inhibition in lying (Gamer *et al.*, 2012; Verschuere *et al.*, 2012). It should be noted that so far most evidence for the contribution of response inhibition is indirect. Response inhibition has been used to explain differential effects of lying compared with truth telling, as for instance elevated RTs (Seymour *et al.*, 2000; Verschuere and De Houwer, 2011), enlarged activation in brain areas linked to response inhibition (Spence *et al.*, 2001; Schumacher *et al.*, 2010; Vartanian *et al.*, 2013) and stronger ERPs linked to conflict-detection (Johnson *et al.*, 2004, 2005, 2008; Dong *et al.* 2010). More direct evidence of response inhibition during lying is scarce. Duran *et al.* (2010)

found, that when moving a Nintendo Wii Remote to truthful or deceitful 'yes' or 'no' answers displayed on the top of a screen, participants' arm movements revealed stronger response competition for deceitful compared with truthful answers as evidenced by a stronger deviation toward the not-chosen (truthful) response. Hadar *et al.* (2012) found in three experiments larger motor-evoked potentials for the truthful compared with the deceitful response during preparation of a deceitful response and no such response competition during the preparation of a truthful response. But although these findings strengthen the idea that response competition indeed causes the cognitive cost of lying, they do not provide information about the specific type of inhibition needed to resolve this competition. Overcoming the truth response in lying might involve inhibition at an earlier stage than the motor inhibition required in the Stop-Signal task (also referred to as 'action cancelation'; Sebastian *et al.*, 2013). Hence, the inhibition involved in lying may for instance rather resemble 'interference inhibition' (Sebastian *et al.*, 2013), and further deception research should differentiate and compare the subcomponents of inhibition in order to clarify which of those is involved in lying.

To sum up, this field study validates laboratory research on the acute impairing effects of alcohol on response inhibition within a realistic drinking environment. Furthermore, it replicated the increased cognitive costs of lying and provides first information on the relationship between alcohol and lying.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *Alcohol and Alcoholism* online.

REFERENCES

Anderson BM, Stevens MC, Meda SA *et al.* (2011) Functional imaging of cognitive control during acute alcohol intoxication. *Alcohol Clin Exp Res* **35**:156–65.

Aron AR, Robbins TW, Poldrack RA. (2004) Inhibition and the right inferior frontal cortex. *Trends Cogn Sci* **8**:170–77.

Aron AR, Robbins TW, Poldrack RA. (2014) Inhibition and the right inferior frontal cortex: one decade on. *Trends Cogn Sci* **18**:177–85.

Bond AJ. (1998) Drug-induced behavioural disinhibition. *CNS Drugs* **9**:41–57.

Boucher L, Palmeri TJ, Logan GD *et al.* (2007) Inhibitory control in mind and brain: an interactive race model of countermanding saccades. *Psychol Rev* **114**:376–97.

Bradley MT, Ainsworth D. (1984) Alcohol and the psychophysiological detection of deception. *Psychophysiology* **21**:63–71.

Caswell AJ, Morgan MJ, Duka T. (2013) Inhibitory control contributes to 'motor'—but not 'cognitive'—impulsivity. *Exp Psychol* **60**:324–34.

Christ SE, Van Essen DC, Watson JM *et al.* (2009) The contributions of prefrontal cortex and executive control to deception: evidence

from activation likelihood estimate meta-analyses. *Cereb Cortex* **19**:1557–66.

Cohen J. (1988) *Statistical Power Analysis for the Behavioural Sciences*. Hillsdale: Lawrence Erlbaum.

Courtney KE, Ghahremani DG, Ray LA. (2013) Fronto-striatal functional connectivity during response inhibition in alcohol dependence. *Addict Biol* **18**:593–604.

de Wit H. (2009) Impulsivity as a determinant and consequence of drug use: a review of underlying processes. *Addict Biol* **14**: 22–31.

de Wit H, Crean J, Richards JB. (2000) Effects of d-Amphetamine and ethanol on a measure of behavioral inhibition in humans. *Behav Neurosci* **114**:830–7.

Debey E, Verschuere B, Crombez G. (2012) Lying and executive control: an experimental investigation using ego depletion and goal neglect. *Acta Psychol* **140**:133–41.

Dong G, Hu Y, Wu H. (2010) The presentation order of cue and target matters in deception study. *Behav Brain Funct* **6**:63.

Dougherty DM, Marsh-Richard DM, Hatzis ES *et al.* (2008) A test of alcohol dose effects on multiple behavioral measures of impulsivity. *Drug Alcohol Depend* **96**:111–20.

Dunlap WP, Cortina JM, Vaslow JB *et al.* (1996) Meta-analysis of experiments with matched groups or repeated measures designs. *Psychol Methods* **1**:170–7.

Duran ND, Dale R, McNamara DS. (2010) The action dynamics of overcoming the truth. *Psychon Bull Rev* **17**:486–91.

Evans JR, Compo NS, Russano MB. (2009) Intoxicated witnesses and suspects: procedures and prevalence according to law enforcement. *Psychol Public Policy Law* **15**:194–221.

Farrow TF, Hopwood MC, Parks RW *et al.* (2010) Evidence of mnemonic ability selectively affecting truthful and deceptive response dynamics. *Am J Psychol* **123**:447–53.

Fillmore MT. (2007) Acute alcohol-induced impairment of cognitive functions: past and present findings. *Int J Disabil Hum Dev* **6**:115–25.

Fillmore MT, Vogel-Sprott M. (1999) An alcohol model of impaired inhibitory control and its treatment in humans. *Exp Clin Psychopharmacol* **7**:49–55.

Fillmore MT, Vogel-Sprott M. (2000) Response inhibition under alcohol: effects of cognitive and motivational conflict. *J Stud Alcohol* **61**:239–46.

Fillmore MT, Ostling EW, Martin CA *et al.* (2009) Acute effects of alcohol on inhibitory control and information processing in high and low sensation-seekers. *Drug Alcohol Depend* **100**:91–9.

Fullam RS, McKie S, Dolan MC. (2009) Psychopathic traits and deception: functional magnetic resonance imaging study. *Br J Psychiatry* **194**:229–35.

Furedy JJ, Davis C, Gurevich M. (1988) Differentiation of deception as a psychological process: a psychophysiological approach. *Psychophysiology* **25**:683–8.

Gamer M. (2011) Detection of deception and concealed information using neuroimaging techniques. In Verschuere B, Ben-Shakhar G, Meijer E (eds). *Memory Detection: Theory and Application of the Concealed Information Test*. Cambridge: Cambridge University Press, 90–114.

Gamer M, Klimecki O, Bauermann T *et al.* (2012) fMRI-activation patterns in the detection of concealed information rely on memory-related effects. *Soc Cogn Affect Neurosci* **7**:506–15.

Greene JD, Sommerville RB, Nystrom LE *et al.* (2001) An fMRI investigation of emotional engagement in moral judgment. *Science* **293**:2105–08.

Hadar AA, Makris S, Yarrow K. (2012) The truth-telling motor cortex: response competition in M1 discloses deceptive behaviour. *Biol Psychol* **89**:495–502.

Haggard-Grann U, Hallqvist J, Langstrom N *et al.* (2006) The role of alcohol and drugs in triggering criminal violence: a case-crossover study. *Addiction* **101**:100–8.

Hu X, Chen H, Fu G. (2012) A repeated lie becomes a truth? The effect of intentional control and training on deception. *Front Psychol* **3**:488.

Jentsch JD, Taylor JR. (1999) Impulsivity resulting from frontostriatal dysfunction in drug abuse: implications for the control of behavior by reward-related stimuli. *Psychopharmacology* **146**:373–90.

Johnson R, Jr, Barnhardt J, Zhu J. (2004) The contribution of executive processes to deceptive responding. *Neuropsychologia* **42**: 878–901.

Johnson R, Jr, Barnhardt J, Zhu J. (2005) Differential effects of practice on the executive processes used for truthful and deceptive responses: an event-related brain potential study. *Brain Res Cogn Brain Res* **24**:386–404.

Johnson R, Jr, Henkell H, Simon E *et al.* (2008) The self in conflict: the role of executive processes during truthful and deceptive responses about attitudes. *Neuroimage* **39**:469–82.

Karim AA, Schneider M, Lotze M *et al.* (2010) The truth about lying: inhibition of the anterior prefrontal cortex improves deceptive behavior. *Cereb Cortex* **20**:205–13.

Kireev MV, Pakhomov SV, Medvedev SV. (2008) Cerebral mechanisms of error detection during deceptive responses in the normal state and under the influence of alcohol. *Hum Physiol* **34**:141–9.

Lappin JS, Eriksen CW. (1966) Use of a delayed signal to stop a visual reaction-time response. *J Exp Psychol* **72**:805–11.

Lawrence AJ, Luty J, Bogdan NA *et al.* (2009) Impulsivity and response inhibition in alcohol dependence and problem gambling. *Psychopharmacology* **207**:163–72.

Leeman RF, Grant JE, Potenza MN. (2009) Behavioral and neurological foundations for the moral and legal implications of intoxication, addictive behaviors and disinhibition. *Behav Sci Law* **27**:237–59.

Logan GD. (1994) On the ability to inhibit thought and action: a user's guide to the stop signal paradigm. In Carr DDTH (ed). *Inhibitory Processes in Attention, Memory, and Language*. San Diego: Academic Press, 189–239.

Logan GD, Cowan WB. (1984) On the ability to inhibit thought and action—a theory of an act of control. *Psychol Rev* **91**:295–327.

Lyvers M. (2000) 'Loss of control' in alcoholism and drug addiction: a neuroscientific interpretation. *Exp Clin Psychopharmacol* **8**:225.

Marczinski CA, Fillmore MT. (2003) Preresponse cues reduce the impairing effects of alcohol on the execution and suppression of responses. *Exp Clin Psychopharmacol* **11**:110–7.

McKay D, Schare ML. (1999) The effects of alcohol and alcohol expectancies on subjective reports and physiological reactivity: a meta-analysis. *Addict Behav* **24**:633–47.

Miyake A, Friedman NP, Emerson MJ *et al.* (2000) The unity and diversity of executive functions and their contributions to complex 'frontal lobe' tasks: a latent variable analysis. *Cognit Psychol* **41**:49–100.

Moll J, de Oliveira-Souza R, Eslinger PJ *et al.* (2002) The neural correlates of moral sensitivity: a functional magnetic resonance imaging investigation of basic and moral emotions. *J Neurosci* **22**:2730–6.

Moll J, Zahn R, de Oliveira-Souza R *et al.* (2005) Opinion: the neural basis of human moral cognition. *Nat Rev Neurosci* **6**:799–809.

Morris SB, DeShon RP. (2002) Combining effect size estimates in meta-analysis with repeated measures and independent-groups designs. *Psychol Methods* **7**:105–25.

Mulvihill LE, Skilling TA, Vogel-Sprott M. (1997) Alcohol and the ability to inhibit behavior in men and women. *J Stud Alcohol* **58**:600–5.

National Research Council. (2003) The polygraph and lie detection. Committee to review the scientific evidence on the Polygraph. Division of Behavioral and Social Sciences and Education. Washington, DC: The National Academic Press.

Nigg JT, Wong MM, Martel MM *et al.* (2006) Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *J Am Acad Child Adolesc Psychiatry* **45**:468–75.

Nikolaou K, Critchley H, Duka T. (2013) Alcohol affects neuronal substrates of response inhibition but not of perceptual processing of stimuli signaling a stop response. *PLoS One* **8**:e76649.

O'Toole D, Yuille JC, Patrick CJ *et al.* (1994) Alcohol and the physiological detection of deception: arousal and memory influences. *Psychophysiology* **31**:253–63.

Oosterlaan J, Sergeant JA. (1996) Inhibition in ADHD, aggressive, and anxious children: a biologically based model of child psychopathology. *J Abnorm Child Psychol* **24**:19–36.

Patton JH, Stanford MS. (1995) Factor structure of the Barratt impulsiveness scale. *J Clin Psychol* **51**:768–74.

Pawliczek CM, Derntl B, Kellermann T *et al*. (2013) Inhibitory control and trait aggression: neural and behavioral insights using the emotional stop signal task. *Neuroimage* **79**:264–74.

Reynolds B, Ortengren A, Richards JB *et al*. (2006) Dimensions of impulsive behavior: personality and behavioral measures. *Pers Individ Dif* **40**:305–15.

Schachar R, Logan GD. (1990) Impulsivity and inhibitory control in normal development and childhood psychopathology. *Dev Psychol* **26**:710–20.

Schachar RJ, Tannock R, Logan G. (1993) Inhibitory control, impulsiveness, and attention-deficit hyperactivity disorder. *Clin Psychol Rev* **13**:721–39.

Schumacher EH, Seymour TL, Schwarb H. (2010) Brain activation evidence for response conflict in the exclude recognition task. *Brain Res* **1329**:113–23.

Sebastian A, Pohl MF, Klöppel S *et al*. (2013) Disentangling common and specific neural subprocesses of response inhibition. *Neuroimage* **64**:601–15.

Seymour TL, Schumacher EH. (2009) Electromyographic evidence for response conflict in the exclude recognition task. *Cogn Affect Behav Neurosci* **9**:71–82.

Seymour TL, Seifert CM, Shafto MG *et al*. (2000) Using response time measures to assess 'guilty knowledge'. *J Appl Psychol* **85**:30–7.

Sigurdsson JF, Gudjonsson GH. (1994) Alcohol and drug intoxication during police interrogation and the reasons why suspects confess to the police. *Addiction* **89**:985–97.

Spence SA, Farrow TF, Herford AE *et al*. (2001) Behavioural and functional anatomical correlates of deception in humans. *Neuroreport* **12**:2849–53.

Spence SA, Kaylor-Hughes C, Farrow TF *et al*. (2008) Speaking of secrets and lies: the contribution of ventrolateral prefrontal cortex to vocal deception. *Neuroimage* **40**:1411–8.

Stevens M, Lammertyn J, Verbruggen F *et al*. (2006) Tscope: A C library for programming cognitive experiments on the MS Windows platform. *Behav Res Methods* **38**:280–6.

Tsujii T, Sakatani K, Nakashima E *et al*. (2011) Characterization of the acute effects of alcohol on asymmetry of inferior frontal cortex activity during a Go/No-Go task using functional near-infrared spectroscopy. *Psychopharmacology* **217**:595–603.

Van Bockstaele B, Verschuere B, Moens T *et al*. (2012) Learning to lie: effects of practice on the cognitive cost of lying. *Front Psychol* **3**:526–52.

Vartanian O, Kwantes PJ, Mandel DR *et al*. (2013) Right inferior frontal gyrus activation as a neural marker of successful lying. *Front Hum Neurosci* **7**:616.

Verbruggen F, Logan GD. (2008) Response inhibition in the stop-signal paradigm. *Trends Cogn Sci* **12**:418–24.

Verbruggen F, Logan GD, Stevens MA. (2008) STOP-IT: Windows executable software for the stop-signal paradigm. *Behav Res Methods* **40**:479–83.

Verschuere B, De Houwer J. (2011) Detecting concealed information in less than a second: response latency-based measures. In Verschuere B, Ben-Shakhar G, Meijer E (eds). *Memory Detection: Theory and Application of the Concealed Information Test.* Cambridge: Cambridge University Press, 46–63.

Verschuere B, Spruyt A, Meijer EH, Otgaar H. (2011) The ease of lying. *Conscious Cogn* **20**:908–11.

Verschuere B, Schuhmann T, Sack AT. (2012) Does the inferior frontal sulcus play a functional role in deception? A neuronavigated theta-burst transcranial magnetic stimulation study. *Front Hum Neurosci* **6**:284.

Vince MA. (1948) The intermittency of control movements and the psychological refractory period. *Br J Psychol Gen Sect* **38**:149–57.

Vogel-Sprott M, Easdon C, Fillmore M, Finn P, Justus A. (2001) Alcohol and behavioral control: cognitive and neural mechanisms. *Alcohol Clin Exp Res* **25**:117–21.

Walczyk JJ, Roper KS, Seemann E, Humphrey AM. (2003) Cognitive mechanisms underlying lying to questions: response time as a cue to deception. *Appl Cogn Psychol* **17**:755–74.