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No association of goal-directed and habitual control with alcohol consumption in young adults

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ABSTRACT

Alcohol dependence is a mental disorder that has been associated with an imbalance in behavioral control favoring model-free habitual over model-based goal-directed strategies. It is as yet unknown, however, whether such an imbalance reflects a predisposing vulnerability or results as a consequence of repeated and/or excessive alcohol exposure. We, therefore, examined the association of alcohol consumption with model-based goal-directed and model-free habitual control in 188 18-year-old social drinkers in a two-step sequential decision-making task while undergoing functional magnetic resonance imaging before prolonged alcohol misuse could have led to severe neurobiological adaptations. Behaviorally, participants showed a mixture of model-free and model-based decision-making as observed previously. Measures of impulsivity were positively related to alcohol consumption. In contrast, neither model-free nor model-based decision weights nor the trade-off between them were associated with alcohol consumption. There were also no significant associations between alcohol consumption and neural correlates of model-free or model-based decision quantities in either ventral striatum or ventromedial prefrontal cortex. Exploratory whole-brain functional magnetic resonance imaging analyses with a lenient threshold revealed early onset of drinking to be associated with an enhanced representation of model-free reward prediction errors in the posterior putamen. These results suggest that an imbalance between model-based goal-directed and model-free habitual control might rather not be a trait marker of alcohol intake *per se*.

Keywords alcohol, goal-directed, reinforcement learning.

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INTRODUCTION

The underlying mechanisms of developing alcohol dependence are not fully resolved despite extensive research over the past decades (e.g. Jensen, Johnson & Redish 2008; Huys *et al.* 2016). Among numerous theoretical approaches, a dual-systems account has been used to explain the development of alcohol dependence (Everitt & Robbins 2013). In this account, alcohol consumption is assumed to be initially goal-directed, which is characterized by knowledge of the contingency between an action (e.g. alcohol intake) and its consequence (e.g. relaxation and euphoria) and an incentive (motivational) value of this consequence. However, it has been argued that with successive repetitions alcohol consumption may first become stimulus-driven and dissociated from its actual consequences, referred to as habitual, and later on compulsive (Tiffany 1990; Everitt & Robbins 2013). In general, these dual-systems accounts hypothesize goal-directed and habitual control to concur and be implemented in separate but interacting and/or competing neural circuits (Dayan & Dolan 2013; Huys *et al.* 2014).

The standard approach to investigate goal-directed and habitual behavior experimentally is by using outcome-devaluation paradigms (e.g. Adams & Dickinson

1981; de Wit *et al.* 2007). Goal-directed control can adapt behavior to changes in the value of an outcome before experiencing the action–outcome association, whereas habitual control needs to experience the devalued outcome before being able to adapt. The distinction between goal-directed and habitual choices maps onto a theoretical distinction between prospective model-based (MB) and retrospective model-free (MF) valuation. The 2-Step task, a two-stage Markov decision problem (Daw *et al.* 2011), operationalizes this distinction and putatively allows the two components to be measured in humans (Daw, Dayan & Niv 2005; Dayan & Dolan 2013; Friedel *et al.* 2014; Gillan *et al.* 2015).

In MF reinforcement learning (RL), subjective values for state-action pairs are updated by reward prediction errors (RPEs), which encode the difference between expected and received outcomes (Barto & Sutton 1998; Dickinson & Schultz 2000). This updating process happens when an action–outcome association is experienced and typically needs multiple repetitions to change state-action values and thereby action policies. Therefore, MF RL shares its retrospective, inflexible, but computationally cheap nature with habitual behavioral control. In contrast, MB RL builds an internal model of the environment and plans actions by searching the potential combinations of future actions and outcomes. Via changes to the model, it can flexibly adapt to changes in contingencies and values along the paths of the internal model. These qualities match the operant definition of goal-directed control.

Reward prediction errors result in a phasic activation of dopamine midbrain neurons (Schultz 1997; D'Ardenne *et al.* 2008) as well as dopamine-innervated target areas such as the ventral striatum (vS) and ventromedial prefrontal cortex (vmPFC; Daw *et al.* 2011). Although these phasic signals conform to exacting detail with MF theory predictions (for a review, see Huys *et al.* 2014), the RPE signals in vS also incorporate MB valuations providing a path by which MB predictions can be incorporated retrospectively into MF predictions (Daw *et al.* 2011; Gershman, Markman & Otto 2014; Jones, Sadacca & Schoenbaum 2016).

There are suggestions that the balance between habitual and goal-directed control might be shifted towards habitual behavior in alcohol-dependent patients. Sjoerds *et al.* (2013) used an outcome–devaluation task. Although there was no behavioral evidence for a shift (patients just performed worse in all conditions), there was a suggestive decreased activation in vmPFC and vS during putatively goal-directed and increased activation of the putamen during putatively habitual decisions in the patients. In the Two-Step task, Sebold *et al.* (2014) reported an impairment of MB decision-making after losses in alcohol-dependent patients compared with healthy control participants. Gillan *et al.* (2016) also reported a decrease in MB decision-making to be associated with alcohol use disorder identification text scores. However, Voon *et al.* (2014) found no difference between detoxified alcohol-dependent patients and healthy controls. Of note, all of these results test decision-making without reference to the abused substance and as such speak to a generalized shift in decision-making rather than one limited to the setting of the substance (Everitt & Robbins 2013).

Alterations in patients could either be a consequence of prolonged alcohol abuse and corresponding neurobiological adaptations (Volkow *et al.* 2004; Heinz *et al.* 2009) or reflect a predisposition for aberrant decision-making preceding the development of hazardous drinking behavior. Another possible explanation combines both aspects: Aberrant decision-making may lead to early and numerous encounters with drugs of abuse, and their high reward value leads to fast habitization of drug seeking and consumption including neurobiological adaptations in cortico-basal ganglia circuits. This might shift the balance further toward aberrant decision-making processes (cf. Sjoerds *et al.* 2013; Story *et al.* 2014).

We aimed to investigate the association of MB and MF decision-making with alcohol consumption before prolonged alcohol misuse could have led to severe neurobiological adaptations. Therefore, we sampled 18-year-old social drinkers, assessed their alcohol consumption, and had them perform the Two-Step task. We hypothesized that a shift towards MF habitual and away from MB goal-directed behavior and neural correlates thereof would be associated with greater alcohol consumption. In particular, we tested whether participants with (1) stronger MF or (2) weaker MB control during Two-Step and (3) stronger MF RPE-related blood-oxygen level-dependent (BOLD) signals of vS and vmPFC or (4) weaker MB signatures there are associated with (1) greater alcohol consumption in general and, specifically; with (2) earlier onset of drinking; (3) higher average alcohol intake; (4) the presence of binge drinking and more frequent and heavy binge-drinking events; (5) higher scores on drinking-related questionnaires; and (6) elevated levels of blood markers for liver function and alcohol consumption.

MATERIALS AND METHODS

Participants and procedure

Two hundred one 18 year-old male social drinkers completed the first assessment of a longitudinal functional magnetic resonance imaging (fMRI) study (ClinicalTrials.gov identifier: NCT01744834). They were

randomly sampled from the population of 18 year-old men of two German cities (Berlin and Dresden) by the respective local registration office. Subjects who responded to the invitation letter were screened via telephone. Exclusion criteria were a history of or current neurological or mental disorders (except for nicotine dependence and alcohol abuse), left-handedness, and contra-indications for MRI. Participants had to have normal or corrected-to-normal vision. Women were not included because they show decreased rates of risky alcohol consumption compared with men (Kraus & Pabst 2008). An additional inclusion criterion was for participants to have had at least two drinking occasions in the past 3 months.

Participants came in twice. At the first appointment, they gave written informed consent and were interviewed using the Composite International Diagnostic Interview (CIDI; Jacobi *et al.* 2013; Pfister & Wittchen 1997) to assess mental disorders according to the German version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; Saß *et al.* 2003). Further, participants completed several questionnaires. They returned for the second appointment approximately 9 days later (SD = 16 days) to complete the Two-Step task (Daw *et al.* 2011) during fMRI. Blood samples for analysis of alanine transaminase (ALT), aspartate transaminase (AST), gamma-glutamyl transferase (γ-GT) and phosphatidylethanol (PEth) were drawn on the first (Berlin) or second (Dresden) appointment. This study was approved by local ethics committees of Technische Universität Dresden and Charité Universitätsmediz in Berlin.

Behavioral analyses are based on 188 subjects. Participants were excluded because of CIDI diagnosis of alcohol dependence (n = 1), alcohol abstinence in the past year though stated otherwise during telephone screening (n = 2), positive drug screening on the day of the fMRI assessment (n = 7), and missing Two-Step data due to technical issues (n = 3). Effect size estimates of previous studies regarding model-free/-based control and alcohol range from $|d|$ = .06 (Voon *et al.* 2014) over $|d|$ = .12 (Gillan *et al.* 2016) to $|d|$ = .53 (Sebold *et al.* 2014) for which we would have a power to identify an association of $(1 - \beta)$ = .07 , $(1 - \beta)$ = .12, and $(1 - \beta)$ = .94, respectively (with n = 188 and α = .05). To check whether exclusion criteria influenced results, all behavioral analyses were repeated with all available data (n = 198).

Measures of goal-directed and habitual behavioral control

The Two-Step task consisted of 201 trials; each of which was composed of two subsequent binary choices (Fig. 1).

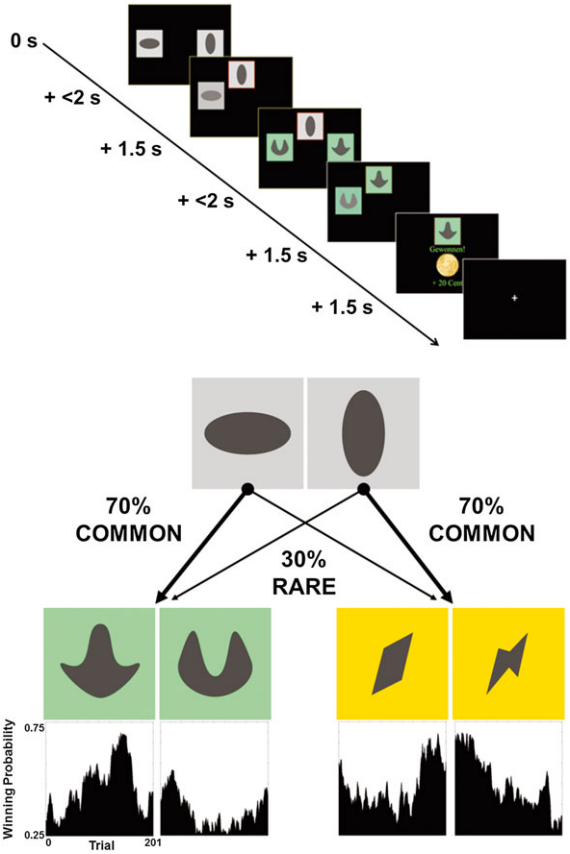

Figure 1 Upper panel: Temporal sequence of one trial of the Two-Step task starting with presentation of the two gray first-stage stimuli followed by a response phase of maximum 2 seconds. Then second-stage stimuli were presented (either green or yellow pair), followed by another response phase, outcome presentation and finally an inter-trial interval with an exponentially distributed jitter of 1–7 seconds. Lower panel: Schematic view of the design of the Two-Step task displaying the choices on the first stage (gray stimuli) and second stage (green and yellow stimuli); displayed below the second-stage stimuli are the corresponding winning probabilities of each second-stage stimulus and their change during the course of the 201 trials of the experiment

First-stage stimuli were always the same two gray boxes. Choice of one of them led with a probability of 70 percent (common transition) to one colored pair of second-stage stimuli and with 30 percent (rare transition) to the other (vice versa for the alternative first-stage stimulus). Participants were informed about the transition structure and that transition probabilities stay fixed during the experiment. Each second-stage stimulus led to reward (20 Cent) with a probability between 25 percent and 75 percent, which was slowly changing during the course of the experiment according to Gaussian random walks [the exact same random walks as in the original publication by Daw *et al.* (2011), were used]. With this setup, participants had to constantly update the utilities of the second-stage stimuli. Updating the values of second-stage stimuli relies on MF learning as there is no

further transition to another state. Therefore, MB and MF control had the same second-stage values but produced different values at the first stage. Choices at the first stage were modeled as a mixture of MF and MB control: MF control increased the probability of repeating a choice at the first stage after being rewarded at the second stage regardless of the transition type of the respective trial; MB control computes action values by weighting the values of possible future states with the probability to reach this state. Hence, MB control is sensitive to which transition had occurred. Participants were paid out the collected rewards of a randomly chosen third of all trials and were told so before the experiment.

Choice data were analyzed using hierarchical logistic mixed-effects regression implemented in the lme4 package (version 1.1–10; Bates *et al.* 2015) in R (version 3.2.2; R Development Core Team, 2008). Repetition of first-level choice was predicted by previous trial's outcome (rewarded versus unrewarded) and transition probability (common versus rare). Both factors and their interaction were taken as random effects across subjects. A significant main effect of outcome indicated a MF strategy, whereas a significant interaction of outcome and transition probability indicated MB control (Daw *et al.* 2011). To test for associations with alcohol consumption, we included measures of drinking behavior as additional between-subject factors in the regression analysis. In addition, scores for MF (MF_{score}) and MB control (MB_{score}) were derived from the individual probabilities to repeat first-stage choice (stay probabilities). These scores are calculated according to the respective assumed choice pattern in MF and MB control [(MF_{score} = P(stay | rewarded common) + P(stay | rewarded rare) − P(stay | unrewarded common) − P(stay | unrewarded rare); MB_{score} = P(stay | rewarded common) − P(stay | rewarded rare) − P(stay | unrewarded common) + P(stay | unrewarded rare); Sebold *et al.* 2014]. Furthermore, choice data were fitted by the computational model introduced by Daw *et al.* (2011), which assumes a hybrid controller using goal-directed and habitual choice strategies. In the model, goal-directed choices were accounted for by MB RL, assuming correct weighting of expected outcomes with expected transition probabilities. The habitual learning system was implemented as MF state-action-reward-state-action (λ) temporal-difference learning (Niranjan & Rummery 1994). Both systems were assumed to contribute to behavioral choice according to the relative weight parameter ω, which varies between fully MF (ω = 0) and fully MB (ω = 1) choice [see Supporting Information (SM1.1) for details]. There were six further parameters of choice behavior modeled, but due to our specific focus on goal-directed and habitual control, we did not analyze these here. We applied a logistic transformation to ω (creating ω_{log}) to adhere to normal distribution assumptions during model fitting and parametric statistical testing. Individual estimates of ω_{log} were used as indicator for the balance of MF and MB control in addition to MF_{score} and MB_{score}. Model comparisons replicated the superiority of a hybrid controller over pure MF and pure MB strategies for the whole sample. Individually, 74 percent (n = 139) subjects showed model fits better than chance.

Measures of alcohol consumption

To characterize participants' drinking behavior, we used information acquired with the CIDI (Pfister & Wittchen 1997; Jacobi *et al.* 2013): age of first drink (i.e. drinking a whole alcoholic beverage), age of first time being drunk, estimated average alcohol consumption in the past year (g alc/day), average alcohol consumption per drinking occasion in the past year (g alc), age of first binge-drinking event, number of binge-drinking events lifetime and average alcohol consumption per binge-drinking event in the past year (g alc). Binge drinking was defined as the consumption of at least five drinks (≥60 g alc) on one occasion. To increase reliability of the single CIDI items as indicators of alcohol drinking behavior and to account for their high intercorrelations (Table 1), we calculated a sum score ($Drink_{score}$) from the z-scaled CIDI items with higher values indicating greater alcohol consumption (see SM1.2 for details). Seventy-four percent of the sample (n = 139) reported at least one lifetime binge-drinking event. Binge drinkers and non-bingers can be seen as two meaningful subgroups within our sample of social drinkers systematically differing in their alcohol consumption (S5) and were, therefore, compared regarding measures of goal-directed and habitual control.

Additionally, we used blood markers for alcohol intake and liver function (AST, ALT, γ-GT and PEth) and several questionnaires to characterize drinking behavior: the Alcohol Dependence Scale (ADS; Horn *et al.* 1984), Obsessive Compulsive Drinking Scale (OCDS-G; Ackermann & Mann 2000), and adapted forms of the Family Tree Questionnaire (Mann *et al.* 1985) and the alcohol-related section of the Family History Assessment Module (Rice *et al.* 1995). Using Family Tree Questionnaire and Family History Assessment Module, participants were classified as family history positive if they had at least one first-degree alcohol-dependent relative fulfilling three or more lifetime DSM-IV-TR criteria or had any treatment of alcohol dependence: 3.7 percent of our sample were considered family-history positive. Because of this small proportion, family history was not included in our analyses. $Drink_{score}$ correlated highly significant with each other measure of alcohol consumption

(Bonferroni corrected for multiple comparisons (105 tests), all *ps* < .0005) except for the blood markers AST, ALT, γ-GT and PEth (all *ps* > .045; Table 1).

Behavioral statistical analyses

To examine associations between the multiple measures of goal-directed and habitual behavioral control (ω_{log}, MF_{score} and MB_{score}) and of alcohol consumption (CIDI measures including $Drink_{score}$, ADS sum score, OCDS-G sum score and blood markers), we first performed a multi-variate analysis of variance (MANOVA) with measures of drinking behavior ($Drink_{score}$, ADS sum score, OCDS-G sum score and blood markers) as dependent and measures of goal-directed/habitual behavioral control (ω_{log}, MF_{score} and MB_{score}) as independent variables. We used MANOVA because our multiple outcome measures characterizing drinking behavior are intercorrelated, and by using a multi-variate approach, we control the familywise error rate. This analysis was repeated with measures of impulsivity as independent variables. The sum score of the Barratt Impulsiveness Scale short form (BIS-15; Kübler, Meule & Vögele 2011) and the Impulsivity subscale of the Substance Use Risk Profile Scale (SURPS; Woicik *et al.* 2009) were also included in behavioral analyses. Thereby, we tested the association between measures of alcohol consumption and measures of impulsivity, which were previously related to alcohol dependence and onset of consumption (Stanford *et al.* 2009; Jurk *et al.* 2015). Testing the association of alcohol consumption and impulsivity was used as demonstration that our analytic approach was sensitive to detecting associations in our data. In addition, we selected the best predictors of drinking behavior (operationalized with $Drink_{score}$) with an elastic net analysis, performed with the glmnet package (version 2.0–2; Friedman, Hastie & Tibshirani 2010) implemented in R [see Supporting Information (SR1.4)]. This type of analysis selects predictors in order to build a regression model explaining as much variance of the outcome as possible with the least necessary number of predictors. Measures of goal-directed/habitual control and impulsivity were entered as predictors to test whether one construct is superior to the other in predicting $Drink_{score}$. Next, we used a correlational approach. Exact Kolmogorov–Smirnov tests implied violation of the assumption of normality for most measures of goal-directed/habitual control and alcohol consumption (Table 2). Therefore, reported correlation coefficients are Spearman's ρ, which was shown to have smaller alpha error rate and higher power than Pearson's r in case of non-normal variables and large sample sizes (Bishara & Hittner 2012). Last, we compared binge drinkers and non-bingers and the four-risk groups regarding WHO criteria of alcohol consumption (WHO 2000) in regard to their measures of goal-directed/habitual behavioral control. In response to comments of the reviewers, we additionally examined whether high self-reported impulsivity was associated with increased habitual or decreased goal-directed behavioral control and neural correlates thereof as reported recently (Deserno *et al.* 2015). Thus, we correlated self-report measures of impulsivity (BIS-15) with measures of habitual/goal-directed control and neural correlates thereof.

All analyses regarding data distribution, correlations and MANOVAs were performed with SPSS 23.0 (2015, IBM SPSS Statistics for Windows: IBM Corp., Armonk, NY, USA).

Functional magnetic resonance imaging data acquisition and analysis

Imaging data were obtained using three-Tesla whole-body MRI scanners (Magnetom Trio, Siemens, Erlangen, Germany) equipped with a 12-channel head coil located at the Neuroimaging Center, Technische Universität Dresden, and the Charité Universitätsmedizin Berlin. For fMRI, a standard T2*-weighted echo-planar imaging (EPI) sequence (TR = 2410 ms; TE = 25 ms; flip angle: 80°; voxel size: 3 × 3 × 2 mm (1 mm gap); FOV: 192 × 192 mm; in-plane resolution: 64 × 64 pixels) was obtained comprising 42 transversal slices in descending order, orientated approximately 25° to the anterior commissure–posterior commissure line. Moreover, a structural T1-weighted magnetization-prepared rapid gradient echo (MPRAGE) image was obtained (TR = 1900 ms, TE = 2.26 ms, flip angle: 9°, voxel size: 1 × 1 × 1 mm, FOV: 256 × 256 mm).

Functional magnetic resonance imaging preprocessing and data analyses were performed with Statistical Parametric Mapping software (SPM8; London, UK: Wellcome Department for Imaging Neuroscience) implemented in Nipype Version 0.9.2 (Gorgolewski *et al.* 2011) and Matlab R2014a (2014. Natick, MA: The MathWorks Inc.). Preprocessing included correction for differences in slice acquisition times with reference to the middle slice, motion correction via realignment of each slice to the first, correction for field inhomogeneities with a voxel displacement map computed from acquired field maps, coregistration of the mean EPI image to the individual MPRAGE image, segmentation and normalization of the individual MPRAGE image to Montreal Neurological Institute space and applying these normalization parameters to the distortion-corrected EPI images, simultaneously resampling EPI images to 2 × 2 × 2 mm, and spatially smoothing the EPI images with a Gaussian kernel of 8 mm full-width-half-maximum. During first-level analyses, a high-pass filter of 128 s width was applied.

Table 1 Intercorrelation of measures of alcohol consumption (n = 188).

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*
	Drink $_{score}$	*Age of first drink*	*Age of first time drunk*	*Estimated alcohol consumption in past year (g/day)*	*Alcohol consumption in past year (g/drinking occasion)*	*Age of first binge-drinking episode*	*Number of binge-drinking episodes lifetime*	*Alcohol consumption per binge-drinking episode (g)*	*ADS Sum Score*	*OCDS-G Sum Score*	*AST*	*ALT*	*γ-GT*	*PEth*
1	1	−.50***	−.617***	.707***	.713***	−.422***	.843***	.756***	.477***	.274***	−.036	.129	.082	.140
2		1	.538***	−.187*	−.121	0	−.227**	−.186*	−.233**	−.060	−.073	−.111	−.129	−.117
3			1	−.311***	−.276***	.300***	−.282***	−.286***	−.317***	−.144	−.036	−.115	−.042	−.037
4				1	.728***	−.034	.669***	.557***	.384***	.378***	−.009	.026	.008	.375***
5					1	−.025	.695***	.632***	.420***	.249***	−.014	.011	−.043	.149
6						1	−.362***	.040	−.142	.046	.002	−.127	.037	.114
7							1	.646***	.443***	.302***	−.027	.124	.046	.118
8								1	.405***	.213**	−.079	.043	.060	.132
9									1	.489***	.018	−.049	−.088	.154
10										1	.096	.041	−.019	.162*
11											1	.566***	.124	.079
12												1	.37***	.049
13													1	.124
14														1

*$P < .05$; **$P < .01$; ***$P < .001$ (two-tailed). Note: All correlations are Spearman's ρ.

Table 2 Demographic information, descriptive statistics of measures of goal-directed/habitual control and alcohol consumption of participants included in analyses (n = 188; see Table S1 for these data of the complete sample).

	n	*Min*	*First quartile*	*Median*	*Third quartile*	*Max*
Descriptive statistics of sample						
Age	188	18.07	18.24	18.33	18.50	18.93
Years in school	187	4	11	12	12	15
Measures of goal-directed/habitual control						
ω[a]	188	0.00	0.20	0.59	0.80	1.00
MF_{score}	188	−0.42	−0.04	0.08	0.21	0.85
MB_{score}	188	−0.34	0.06	0.24	0.49	1.21
Measures of alcohol consumption						
CIDI measures						
$Drink_{score}$	188	−8.21	−3.54	−0.35	1.61	17.52
Age of first drink[a]	188	9	14	14	15	18
Age of first time drunk[a]	180	10	15	16	17	18
Estimated alcohol consumption in past year (g/day) [a]	188	0.00	3.21	6.43	15.43	112.50
Alcohol consumption in past year (g/drinking occasion)[a]	188	18	45	54	90	342
Age of first binge-drinking episode[a]	131	14	16	16	17	18
Number of binge-drinking episodes lifetime[a]	131	1	4	10	20	150
Alcohol consumption per binge-drinking episode (g)[a]	139	63	90	117	135	450
Questionnaire measures						
ADS sum score[a]	181	0	2	4	7	30
OCDS-G sum Score[a]	183	0	1	3	5	18
Blood markers						
AST (μKat/l)[a]	183	0.17	0.35	0.40	0.48	2.51
ALT (μKat/l)[a]	182	0.11	0.27	0.35	0.45	1.59
γ-GT (μKat/l)[a]	183	0.13	0.23	0.27	0.33	0.89
PEth[a]	158	10	10	60	60	1180
Measures of impulsivity						
BIS-15 sum score	185	18	27	30	34	45
SURPS Impulsivity[a]	186	5	9	10	11	17

[a]Exact Kolmogorov–Smirnov test implied non-normal distribution of this measure ($P < .05$). Note: n occasionally differs from 188 (or 139 in binge drinking-related measures, respectively) due to single missing data points. ADS = Alcohol Dependence Scale; ALT = alanine transaminase; AST = aspartate transaminase; BIS-15 = Barratt Impulsiveness Scale (short form); Drinkscore = score of drinking behavior from CIDI measures of alcohol consumption; γ-GT = gamma-glutamyl transferase; MBscore = score of model-based control; MFscore = score of model-free control; OCDS-G = Obsessive Compulsive Drinking Scale; ω = balance between model-free and model-based control; PEth = phosphatidylethanol; SURPS = Substance Use Risk Profile Scale.

Model-based fMRI analyses are based on 146 subjects. Neuroradiologists screened each T1-weighted MPRAGE image for anatomical findings leading to exclusion of five participants. Additionally, participants were excluded because of missing field maps (n = 3), ghost artifacts in EPI after preprocessing (n = 4), non-remediable failure of coregistration (n = 2) or normalization (n = 7) and extensive motion during fMRI (n = 21; >3 mm translation or 3° rotation volume-to-volume) resulting in a sample size of n = 146 for fMRI analyses. We computed RPEs for each participant. RPEs are non-zero at the onsets of second-stage and outcome presentation (Daw *et al.* 2011). Therefore, we modeled BOLD signals at these timepoints by two parametric modulators obtained from the computational model. MF RPE (RPE_{MF}) and MB RPE time series were derived for both timepoints under the assumption of fully MF (ω = 0) and fully MB (ω = 1) control, respectively. To capture unique trial-variance in RPEs associated with the MB but not the MF system, we used the difference between MF and MB RPEs ($RPE_{\Delta MB}$) as regressor. At the second stage, there is no further transition to another stage, and MB learning reduces to pure MF learning. That is why $RPE_{\Delta MB}$ is zero at outcome presentation. We set up individual fMRI statistics according to Daw *et al.* (2011; see SSM2.1 for details). For repetition of their analyses, we validated the task setup with region of interest (ROI) analyses in anatomically defined masks of bilateral vS and vmPFC (SM2.2 and Fig. S2); reported activations were deemed significant at $P_{FWE} < .05$ for the peak voxel. To test our hypotheses that neural correlates of MF and MB control are associated with alcohol consumption, mean activation in the same ROIs were correlated with measures of drinking behavior (trading-off spatial resolution to reduce the number of tests performed). Additionally, exploratory whole-brain analyses were performed to test for associations outside the a priori defined ROIs. For these analyses, statistical thresholds were set to $P_{uncorr.} < .001$, $k \geq 50$,

and results were deemed significant with $P_{FWE} < .01$ on cluster level. All fMRI analyses included a dichotomous variable for site of investigation as covariate to control for possible center effects.

RESULTS

Sample characteristics

The sample consisted of 188 young male adults. Table 2 summarizes the distribution of sociodemographic information and relevant measures of goal-directed/habitual control and alcohol consumption. According to criteria for risk of alcohol consumption published by the WHO (World Health Organization 2000) for comparative research purposes, this sample can be characterized as follows: regarding average consumption on a single drinking occasion in the past year, 21.8 percent fall into the low-risk (1–40 g alc), 31.9 percent in the medium-risk (41–60 g alc), 30.3 percent in the high-risk (61–100 g alc) and 16.0 percent in the very-high-risk category (101 + g alc); regarding average alcohol consumption per day in the past year, 96.8 percent have to be characterized as having low-risk (1–40 g alc), 2.1 percent as having medium-risk (41–60 g alc) and 1.1 percent as having high-risk (61 + g alc) alcohol consumption. Eight participants (4.3 percent) fulfilled DSM IV-criteria of alcohol abuse: 73.9 percent reported at least one occasion of binge drinking. Levels of blood markers were below the cut-off value for pathological levels in 91.5 percent (AST; cut-off 0.835 μKat/l), 93.1 percent (ALT; cut-off 0.835 μKat/l) and 100 percent (γ-GT; cut-off 1.002 μKat/l) of the sample. PEth values were available for 158 participants only, because collection started 3 months after the start of Two-Step data collection. Of available PEth data, 31.6 precent were negative (i.e. <20 ng/ml) suggesting no or very low-alcohol consumption in the preceding 2 weeks; in 46.8 percent, values were positive but too low to be exactly measurable (i.e. 20–100 ng/ml) indicating low-alcohol intake. For these participants, PEth values were set to 10 and 60, respectively. Consequently, PEth was treated as ordinal data. Thirteen participants (8.2 percent) had PEth values >210 ng/ml, which was suggested to be the threshold between moderate drinking and alcohol misuse (Wurst *et al.* 2015).

Thirty participants (16 percent) reported to currently be regular smokers. Exact Mann–Whitney *U*-test showed no differences in measures of goal-directed/habitual control between smokers and non-smokers. Also, smoking status had no significant effect on the results of analyzing stay probabilities with the logistic regression. In addition, we compared participants with an individual better-than-chance model fit ($n = 139$) with the non-fitters regarding the measures of alcohol consumption with exact Mann–Whitney *U*-test and found no significant differences. Furthermore, there were no significant correlations between individual log-likelihoods and measures of alcohol consumption (all Spearman's $|\rho| < .135$, all Ps $> .067$) except for correlations with the three blood markers (AST: $\rho = .156$, $P = .035$; ALT: $\rho = .174$, $P = .019$; γ-GT: $\rho = .168$, $P = .023$).

Behavioral results

First, we analyzed behavioral choice tendencies of participants to find evidence for MF and MB control. Therefore, we performed a logistic regression to analyze how previous trial's transition type from first to second stage (common versus rare) and final outcome (reward versus no reward) affected the probability to repeat the same choice at first stage in the current trial. Participants had a higher probability to repeat a first-stage choice after having been rewarded in the previous trial (significant main effect of outcome), which indicates MF control strategies. The probability to repeat a first-stage choice was also increased after rewarded trials with common transition and unrewarded trials with rare transition (significant interaction effect of outcome and transition type). This interaction effect indicates MB behavioral control. Additionally, this analysis yielded a significant main effect of transition with repetition probability being generally higher after common compared with rare transition trials (all Ps $< .001$; see SR1.2 and Fig. 3).

Second, we investigated the relationship between measures of goal-directed/habitual control and alcohol consumption. Therefore, we first included $Drink_{score}$ as additional between-subjects factor in the logistic regression analysis of choice repetition. This yielded no significant effects of $Drink_{score}$ while preserving the aforementioned main and interaction effects (see SR1.2). Then, we used MANOVA with measures of goal-directed/habitual control (ω_{log}, MF_{score} and MB_{score}) as independent and measures of alcohol consumption ($Drink_{score}$, ADS sum score, OCDS-G sum score, AST, ALT, γ-GT and PEth) as dependent variables. MANOVA is a multi-variate approach bypassing the multiple comparisons problem we face with our multitude of dependent and independent variables. This analysis yielded no significant associations of alcohol consumption measures with ω_{log} $F_{(7,\ 142)} = 1.685$, $P = .117$, $\eta_p^2 = .077$; MF_{score} $F_{(7,\ 142)} = .646$, $P = .717$, $\eta_p^2 = .031$; or MB_{score} $F_{(7,\ 142)} = 1.491$, $P = .175$, $\eta_p^2 = .068$. Next, we correlated measures of goal-directed/habitual control (ω_{log}, MF_{score} and MB_{score}) with measures of alcohol consumption. The associations of main interest between $Drink_{score}$ and each measure of model-free/-based control did not reach significance (Table 3 and Fig. 2). Besides

Table 3 Results of correlations between measures of alcohol consumption and behavioral measures of goal-directed/habitual control, mean extracted ROI BOLD responses to RPE_{MF} and $RPE_{\Delta MB}$ and measures of impulsivity.

	ω	MF_{score}	MB_{score}	RPE_{MF}		$RPE_{\Delta MB}$		*BIS-15*	*SURPS*
				vS	*vmPFC*	*vS*	*vmPFC*	*SUM*	*IMP*
$Drink_{score}$	−.067	.000	−.004	−.019	.014	−.058	−.023	.256***	.246***
Age of first drink	−.011	.042	.057	−.184*	−.143	−.063	−.008	−.125	−.263***
Age of first time drunk	.066	.052	.048	−.044	−.011	−.040	.059	−.182*	−.155*
Estimated alcohol consumption in past year (g/day)	−.070	−.071	.038	−.101	.021	−.105	−.048	.088	.116
Alcohol consumption in past year (g/drinking occasion)	−.026	−.081	.101	−.087	−.006	−.038	−.018	.133	.081
Age of first binge-drinking episode	.098	−.033	.019	.075	.040	.076	.047	−.156	−.126
Number of binge-drinking episodes lifetime	−.033	.038	.044	.001	.047	−.090	−.035	.232**	.179*
Alcohol consumption per binge-drinking episode (g)	−.064	.096	−.018	−.015	.048	.035	.059	.210**	.245***
ADS sum score	−.061	.007	.029	.006	.115	−.040	−.099	.211**	.298***
OCDS-G sum score	.000	−.011	.031	.088	.182*	.021	.073	.223**	.228**
AST	.015	.015	−.047	−.025	.059	−.008	−.042	.039	.165*
ALT	−.072	.061	−.080	.003	.029	.010	.030	−.018	.159*
γ-GT	−.066	−.011	−.160*	−.074	−.089	−.075	−.005	−.205**	−.092
PEth	.041	−.048	.052	−.091	−.016	−.019	.005	−.150	.005

*$P < .05$; **$P < .01$; ***$P < .001$ (two-tailed). Note: All correlations are Spearman's ρ. ADS = Alcohol Dependence Scale; ALT = alanine transaminase; AST = aspartate transaminase; BIS-15 = Barratt Impulsiveness Scale (short form) with SUM, Sum score; Drinkscore = score of drinking behavior from CIDI measures of alcohol consumption; γ-GT = gamma-glutamyl transferase; MBscore = score of model-based control; MFscore = score of model-free control; OCDS-G = Obsessive Compulsive Drinking Scale; ω = balance between model-free and model-based control; PEth = phosphatidylethanol; SURPS = Substance Use Risk Profile Scale with IMP, Impulsivity subscale; vS = ventral striatum; vmPFC = ventromedial prefrontal cortex.

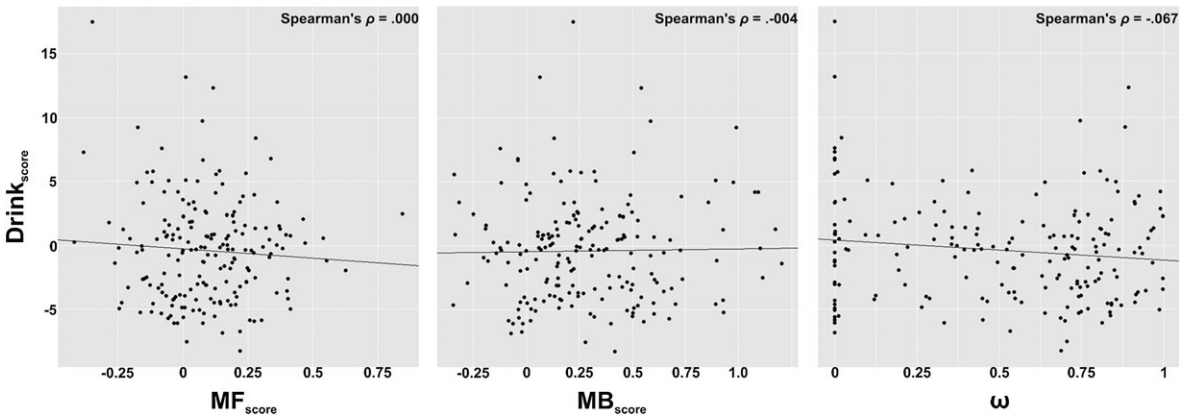

Figure 2 Scatterplots of $Drink_{score}$ with the three measures of goal-directed/habitual control: the score for model-free (MF_{score}) and model-based (MB_{score}) choice behavior stay probabilities and the balance parameter from the hybrid-controller computational model (ω). Note that for displaying purposes and better interpretability, ω is used instead of ω_{log}, but this does not influence the rank–order correlation

this, these analyses yielded a significant negative association of γ-GT with MB_{score} (Spearman's $\rho = -.160$, $P = .031$; Table 3). However, this finding did not survive Bonferroni correction for multiple comparisons (42 tests). No further correlation on the behavioral level reached significance (all $Ps > .168$).

Since binge drinkers and non-bingers can be seen as meaningful subgroups in this sample showing numerous differences in drinking behavior (Table S5), we compared the measures of goal-directed/habitual control between these groups using Exact Mann–Whitney *U*-test. These analyses yielded no significant differences between binge drinkers and non-bingers with regard to ω, MF_{score} or MB_{score} (all $Ps > .125$; Table S5). In addition, we compared measures of goal-directed/habitual control between the four-risk groups

regarding average consumption on a single drinking occasion in the past year (WHO 2000). Adding WHO risk group as a fixed between-subjects factor in the logistic regression of stay probabilities did not yield any significant effect of WHO risk group while preserving aforementioned main and interaction effects. Non-parametric Kruskal–Wallis test showed no differences between the WHO risk groups in ω, MF_{score} or MB_{score} (all $\chi^2_{(3)} < 2.584$, $Ps > .460$).

In contrast to MF an MB control, measures of impulsivity (BIS-15 sum score and SURPS impulsivity subscale) showed the anticipated associations with drinking behavior. Correlational analyses yielded several significant results indicating that earlier and heavier alcohol consumption is associated with higher scores of impulsivity (Table 3). To directly compare the associations of measures of goal-directed/habitual control and impulsivity with measures of drinking behavior, we set up a MANOVA including both groups of predictors. This analysis corroborated previous results by revealing significant associations with measures of impulsivity but not goal-directed/habitual control [ω_{log}: $F_{(7,\ 139)} = 1.954$, $P = .066$, $\eta_p^2 = .090$; MF_{score}: $F_{(7,\ 139)} = .779$, $P = .606$, $\eta_p^2 = .038$; MB_{score}: $F_{(7,\ 139)} = 1.365$, $P = .225$, $\eta_p^2 = .064$; BIS-15 sum score: $F_{(7,\ 139)} = 2.660$, $P = .013$, $\eta_p^2 = .118$, SURPS impulsivity subscale: $F_{(7,\ 139)} = 2.518$, $P = .018$, $\eta_p^2 = .113$]. We then used an elastic net analysis (Friedman *et al.* 2010) to select the best predictors of $Drink_{score}$ among the measures of goal-directed/habitual control and impulsivity thereby directly comparing their respective relation to participants' drinking behavior. This analysis corroborated the findings insofar as no measure of goal-directed/habitual control was selected as predictor, but both measures of impulsivity were (see SR1.4).

All behavioral analyses were repeated with all available data ($n = 198$) to check whether exclusion criteria influenced the results. Participants, which had previously been excluded had higher $Drink_{score}$, ADS sum score, and OCDS-G sum score and reported lower age of first drink, first time drunk and higher average alcohol consumption (exact Mann–Whitney *U*-test, all $Ps < .019$). Nevertheless, results did not change with inclusion of these subjects.

Functional magnetic resonance imaging results

With fMRI data, we first tested the main effects of interest, namely, BOLD correlates of RPE_{MF} and $RPE_{\Delta MB}$. Separate one-sample *t*-test of fMRI contrasts for RPE_{MF}, and $RPE_{\Delta MB}$ were performed as ROI as well as exploratory whole-brain analyses. In addition, we tested the conjunction null hypothesis (Nichols *et al.* 2005) of RPE_{MF} and $RPE_{\Delta MB}$ being correlated with the BOLD responses in the same regions. BOLD responses in vS and vmPFC were associated with RPE_{MF} as well as $RPE_{\Delta MB}$ at $P_{FWE} < .05$ (Fig. 3 and Table S7–9). This replicates the finding of the original study that there are

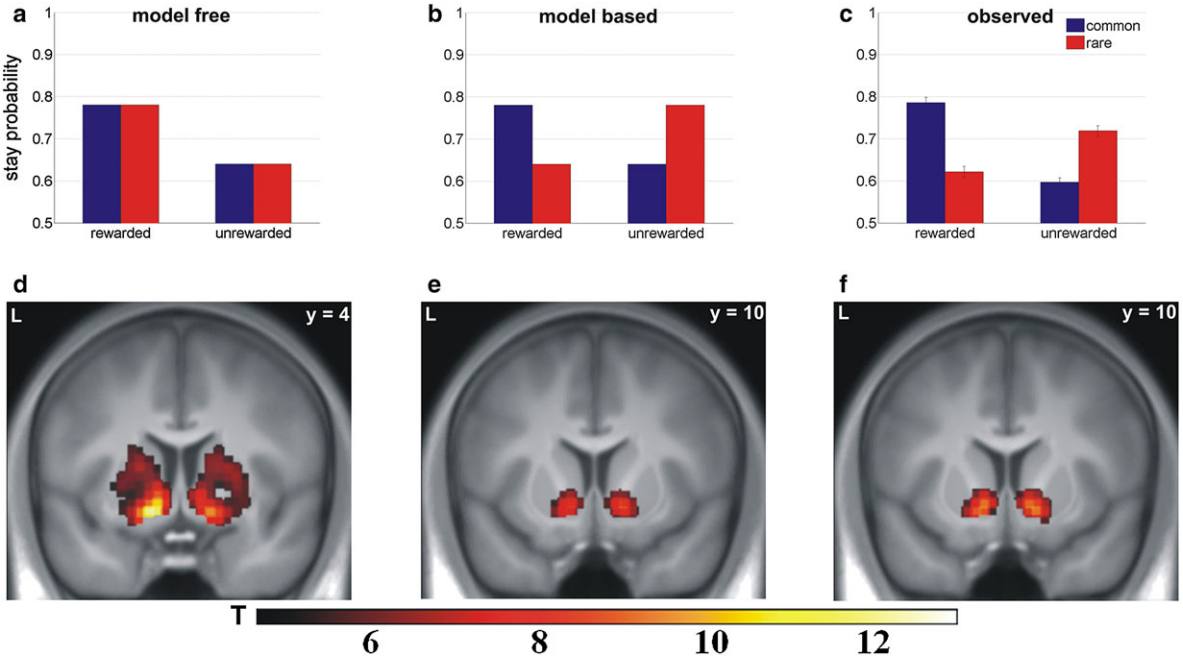

Figure 3 Upper panel: Stay probabilities in hypothetical cases of pure (a) model-free and (b) model-based control. (c) Observed stay probabilities in our sample resemble a mixture of model-free and model-based behavioral control with a tendency towards model-based control. Error bars indicate SEM. Lower panel: Striatal BOLD correlates of (d) RPE_{MF} and (e) $RPE_{\Delta MB}$ and (f) their conjunction. Displayed at $P_{FWE} < .05$; $4.64 < T < 12.5$; whole brain analyses

signatures of MB evaluation in the ventral striatal BOLD response to RPEs, the 'signal most associated with model free RL' (Daw *et al.* 2011, p. 1210).

Finally, we tested whether alcohol consumption is associated with neural representations of RPE_{MF} and $RPE_{\Delta MB}$. We first correlated measures of alcohol consumption with extracted mean activation in vS and vmPFC ROIs. This analysis revealed significant associations of BOLD responses to RPE_{MF} in vS with age of first drink ($\rho = -.184$, $P = .026$) and in vmPFC with OCDS-G sum score ($\rho = .182$, $P = .031$; Table 3). Similar to the significant correlation of γ-GT and MB_{score} on the behavioral level, these correlations did not survive Bonferroni correction for multiple comparisons (28 tests). A MANOVA with measures of drinking behavior as dependent and mean extracted ROI activation as independent variables yielded no significant results (all $Fs_{(7,108)} < .865$, all $Ps > .537$, all $\eta_p^2 s < .053$). Next, exploratory whole-brain regression analyses were performed by testing the relationships of RPE_{MF} and $RPE_{\Delta MB}$ with drinking measures. A negative association between BOLD responses to RPE_{MF} and age of first drink was revealed in a cluster in left putamen, pallidum and insula ($t_{(1,143)} = 4.017$, $k = 608$; Fig. 4 and Table S10), which corresponds to $r = .319$ in the peak voxel of this cluster. This cluster also involves voxels, which are included in our mask of vS explaining the significant correlation of BOLD responses to RPE_{MF} in vS with age of first drink. No further measure of alcohol consumption showed an association with BOLD responses to RPE_{MF} or $RPE_{\Delta MB}$.

Next, we compared neural representations of RPE_{MF} and $RPE_{\Delta MB}$ between binge drinkers and non-bingers. Exact Mann–Whitney *U*-test comparing extracted mean ROI activations in RPE_{MF} and $RPE_{\Delta MB}$ contrasts in vS and vmPFC yielded no significant differences between binge drinkers and non-bingers (all $Ps > .414$; Table S11). Additionally, no significant differences were observed in exploratory whole-brain two-sample *t*-test comparing BOLD responses to RPE_{MF} and $RPE_{\Delta MB}$, respectively, between binge drinkers and non-bingers.

In addition, we examined the relation between measures of impulsivity and goal-directed/habitual control. There has been evidence that high-impulsive subjects have a 'subtle accentuation of model-free control' on a behavioral level and reduced lateral pre-/orbitofrontal MB signals during Two-Step (Deserno *et al.* 2015, p. 5). In our sample, we found no significant associations between BIS-15 subscales or Sum score with λ, ω, MF_{score}, MB_{score}, as well as neural correlates of MF and MB control in vS and vmPFC ROIs (see Table S6 for results of correlations) or 10 mm-spheres around the three peaks in lateral pre-/orbitofrontal cortex reported in Deserno et al. (2015).

DISCUSSION

We investigated the association between goal-directed and habitual behavioral control during an RL task and alcohol consumption in healthy social-drinking young adults. The overall finding of our study is that there were no significant associations of measures of goal-directed or habitual control and alcohol consumption. On the behavioral level, there were no significant associations between stronger habitual or weaker goal-directed control with (1) greater alcohol consumption in general; (2) earlier onset of drinking; (3) higher average alcohol intake; (4) the presence of binge drinking and more frequent and heavy binge-drinking events; (5) higher scores on drinking-related questionnaires; or (6) elevated levels of blood markers for liver function and alcohol consumption, except for a small correlation between MB behavior and γ-GT. On the neural level, stronger representation of MF RPE in vS and vmPFC and weaker MB signatures in these representations were also not significantly associated with measures of alcohol consumption. However, both ROI and exploratory whole-brain analyses revealed that participants, who

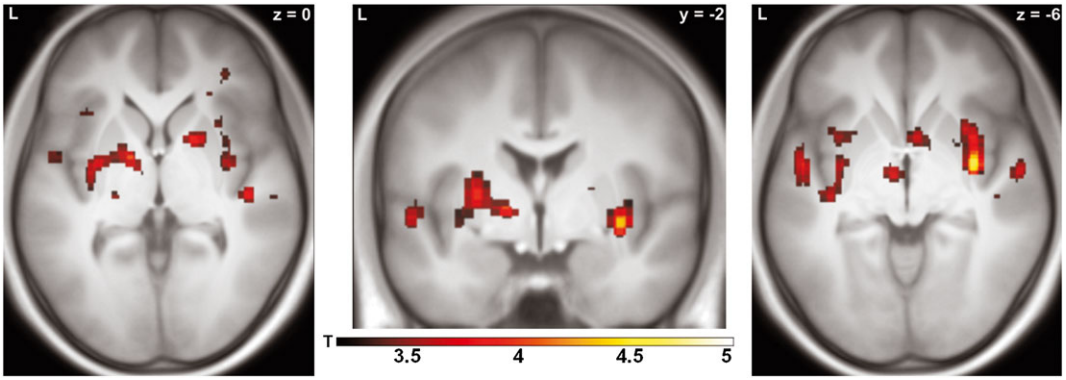

Figure 4 Negative association between BOLD response to RPE_{MF} and age of first drink. Displayed at $p_{uncorr.} < .001$; $3.15 < T < 5$; whole brain analyses

reported earlier onset of drinking, showed a stronger correspondence between BOLD signals in the putamen and RPE_{MF}.

We found that on a behavioral level greater alcohol consumption at age 18 was not associated with stronger MF habitual or weaker MB goal-directed behavior. This suggests that favoring habitual over goal-directed control during decision-making might not be a predisposing vulnerability factor for alcohol consumption *per se*. However, generalization is limited because we deliberately excluded subjects with rare drinking patterns among young men, namely, complete alcohol abstinence or early alcohol dependence. We did this in order to avoid ceiling and floor effects in alcohol consumption over time, to increase the variety of possible drinking trajectories during the follow-up interval and to not include participants who already had severe neuroadaptations due to pathological alcohol intake. Therefore, although this sample is appropriate to investigate drinking trajectories longitudinally, variance in alcohol consumption at the cross-sectional level is limited by design. This might have contributed to the lack of associations reported here. Repeating the behavioral analyses with the subjects excluded due to positive drug screenings or extreme alcohol consumption patterns increased variance in alcohol consumption but failed to alter the results. Furthermore, despite the limited variance in drinking behavior, we did find a robust association of alcohol consumption with impulsivity. Impulsivity has often been associated with substance abuse and is thought to increase liability for addiction (Redish *et al.* 2008; Dalley, Everitt & Robbins 2011). It, therefore, seems unlikely that the current null results with respect to learning variables are due to a lack of variance in alcohol consumption. It certainly suggests that the relation between alcohol consumption and the degree of goal-directed/habitual behavioral control is negligible in comparison to the relation with impulsivity.

Another group has recently also investigated the association between goal-directed/habitual control and alcohol consumption. Investigating a large sample of 1413 participants with an internet-based on-line version of the Two-Step task, Gillan *et al.* (2016) reported a negative association between Alcohol Use Disorders Identification Test scores and MB control. At first glance, our null finding in this regard seems to be in contrast to their result, but the association found in their sample was rather small; and the sample size in our study is too low to detect associations of this magnitude: So the results of Gillan *et al.* (2016; $|d|=.12$) and our study point to a weak association. Howsoever, web-based assessments seem to be a valuable approach to reach more participants and should be used in future studies to complement face-to-face assessments.

Two further studies used the Two-Step task in cohorts of alcohol-dependent patients after cessation of alcohol use and control groups. One of them found a significantly lower magnitude of MB control in patients compared with control participants (Sebold *et al.* 2014), while the other did not (Voon *et al.* 2014). This discrepancy can partly be resolved: first, the difference between alcohol-dependent patients and control participants in Sebold *et al.* (2014) was not significant when controlling for processing speed, in which these groups differed significantly. Second, alcohol-dependent patients in the study of Sebold *et al.* (2014) were abstinent for about 2 weeks, while patients in Voon *et al.* (2014) were abstinent from alcohol for 2 weeks to 1 year and revealed a correlation of longer duration of abstinence with more MB control. Taken together, an imbalance in goal-directed/habitual control does not seem to increase liability for alcohol dependence substantially. If goal-directed control as measured with the Two-Step task is indeed reduced in alcohol-dependent subjects, this might rather emerge during the course of prolonged, excessive alcohol use and, like other cognitive alterations, might be reversible after cessation of alcohol consumption.

As a side issue, we examined the relation between impulsivity and behavioral control during the Two-Step task and found no evidence of a behavioral or neural association. Both impulsivity and the balance between goal-directed and habitual control have been proposed as possible vulnerability factors for addiction (Redish *et al.* 2008) and were hypothesized to interact (Story *et al.* 2014; Deserno *et al.* 2015). Nevertheless, our data do not support this hypothesis. However, rejecting this hypothesis in general on the basis of our results would be premature. Impulsivity is a broad, multi-facetted construct, and research on finer levels of abstraction is warranted to investigate this issue further. Possibly, high motor impulsivity might lead to often favoring fast habit-like actions over slowly forward-planned actions, or high delay discounting might lead to more frequent choices of temporally proximal rewards leading to faster habitization of actions due to more frequent reinforcements (Story *et al.* 2014).

The association of the neural representation of MF RPEs with onset of drinking was predominantly localized in the posterior putamen, an area previously related to the representation of values learned by MF RL (Dayan, Dolan & Wunderlich 2012; Lee, O'Doherty & Shimojo 2014), habit learning, and control of habitual behavior in healthy (Balleine, O'Doherty & Tricomi 2009) and alcohol-dependent subjects (Sjoerds *et al.* 2013). The putamen receives extensive input from the dopaminergic midbrain nuclei (Haber & Knutson 2010), whose output (Schultz 1997) and BOLD response (D'Ardenne *et al.*

2013) have been shown to represent RPEs and be causal for learning (Steinberg *et al.* 2013). However, this enhanced representation of MF error signals did not translate into stronger MF habitual behavioral control during the Two-Step task. This might indicate a compensatory mechanism by which subjects with early onset achieve the same balance between MF and MB control despite stronger neural representation of MF values. This could work via downregulation of functional connectivity between posterior putamen and vmPFC, where MF and MB values are thought to be integrated (Lee *et al.* 2014). Alternatively, a change of the neural representation of MF values might precede a measurable change of MF behavioral control. The longitudinal design of this study will address this question. In addition, this finding will have to be replicated in future studies—just like the association of OCDS-G scores with the mean BOLD response to RPE_{MF} in vmPFC—to decrease the risk of interpreting a false-positive finding.

Interestingly, acute alcohol administration has been shown to reduce goal-directed control in a devaluation task (Hogarth *et al.* 2012). This could lead to habitual control taking over in acute alcohol intoxication and, thereby, increase the probability of choosing previously rewarded actions such as consuming even more alcohol. This provides a possible explanation for out-of-control binge drinking. Hence, in terms of searching for predictors of alcohol consumption at this age, individual volatility or state dependence of the balance between both control systems under acute alcohol may yield better predictive properties for drinking patterns.

There are limitations of this study: First, because of our exclusion criteria, this sample is not representative for the whole population of young adults. This reduces generalizability of our results. Second, we examined participants after they started drinking alcohol rather than before. Both factors preclude us from conclusively ruling out aberrant decision-making as a predisposing risk factor of hazardous alcohol use, although our results strongly suggest that any present association might be negligible.

In summary, we investigated the relationship of goal-directed and habitual control and alcohol drinking behavior in young adult social drinkers. Results did not confirm our hypothesis that an imbalance between goal-directed and habitual control favoring habitual behavior was associated with greater alcohol consumption on a cross-sectional level. These results favor the view that a transition from goal-directed to habitual control as proposed by theoretical work (Everitt & Robbins 2013) occurs during later steps on the path to an alcohol use disorder rather than being a trait marker for alcohol use *per se*.

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CONFLICT OF INTEREST

The authors of this article declare no conflict of interest.

AUTHORS CONTRIBUTION

AH, MAR, QJMH and MNS were responsible for the study concept and design. SN, NB, MS, LS and SKP contributed to the acquisition of self-report, behavioral and fMRI data. QJMH and DJS provided the computational modeling framework. SN performed the behavioral and fMRI analyses. NBK, DJS, NB, MS, DKM, LS, QJMH and MNS assisted with data analysis and interpretation of findings. SN drafted the manuscript. NBK, DJS, NB, MS, SKP, AH and MNS provided critical revision of the manuscript for intellectual content. All authors reviewed the content and approved the final version for publication.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Figure S1. ROI masks. Ventral striatum in red, vmPFC in blue

Table S 1. Demographic information and descriptive statistics of drinking measures in the full sample ($N = 201$).

Table S2. Descriptive statistics of 2-Step parameters ($n = 188$)

Table S3. Intercorrelation of measures of goal-directed/habitual behavioral control ($n = 188$)

Table S4. Coefficients of 17 predictors of $Drink_{score}$ for λ_{min} and λ_{1SE}

Figure S2. Mean squared error (MSE) of elastic net model $\pm$ standard deviation for different values of log(λ). Digits above figure show included number of predictors. First vertical line from the left depicts the value of λ with the smallest MSE (λ_{min}), second vertical line depicts largest λ within one standard error (λ_{1SE}) which represents the sparser and more conservative model

Table S5. Results of exact Mann–Whitney *U*-tests comparing measures of goal-directedness and drinking behavior between binge-drinkers and non-bingers

Table S6. Correlation of BIS-15 subscales and Sum score with behavioral and neural measures of goal-directed/habitual control

Table S7. Correlation of BOLD response with RPE_{MF} in whole-brain analyses

Table S8. Correlation of BOLD response with $RPE_{\Delta MB}$ in whole-brain analyses

Table S9. Results of conjunction analysis of RPE_{MF} and $RPE_{\Delta MB}$ in whole-brain analyses

Table S10. Correlation of BOLD response with RPE_{MF} in whole-brain analyses covarying negatively with age of 1st drink

Table S11. Results of exact Mann–Whitney *U*-tests comparing extracted mean ROI BOLD response between binge-drinkers and non-bingers