Model-Based and Model-Free Decisions in Alcohol Dependence

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Abstract

Background: Human and animal work suggests a shift from goal-directed to habitual decision-making in addiction. However, the evidence for this in human alcohol dependence is as yet inconclusive. **Methods:** Twenty-six healthy controls and 26 recently detoxified alcohol-dependent patients underwent behavioral testing with a 2-step task designed to disentangle goal-directed and habitual response patterns. **Results:** Alcohol-dependent patients showed less evidence of goal-directed choices than healthy controls, particularly after losses. There was no difference in the strength of the habitual component. The group differences did not survive controlling for performance on the Digit Symbol Substitution Task. **Conclusion:** Chronic alcohol use appears to selectively impair goal-directed function, rather than promoting habitual responding. It appears to do so particularly after nonrewards, and this may be mediated by the effects of alcohol on more general cognitive functions subserved by the prefrontal cortex.

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Introduction

Substance dependence is characterized by maladaptive choices that contrast with the subjects' explicitly stated desires, such as the failure to abstain despite the desire to quit drinking. This inflexible and compulsive behavior in addiction suggests a failure or disruption of several components of the underlying decision-making systems.

Two well-defined components of decision-making have been computationally characterized. On the one hand, a flexible, goal-directed, model-based planning sys-

tem explicitly considers the consequences of actions [1]. It is 'model-based' in that it relies on a model of the world (in simple experiments often just the action-outcome contingencies), and then deduces from this model the best sequence of actions. Although it is computationally costly, requiring the explicit consideration of future outcomes, it can be immediately sensitive to environmental changes and lead to rapid behavioral adaptation. In parallel, a more rigid habitual system repeats actions that were in the past associated with reward [2, 3]. The learning process that leads to habits relies on iterative updates of expectations through putatively dopaminergic [4–6] prediction errors, but unlike the goal-directed system does not rely on an explicit model. Hence, it is termed model-free. Because it relies on iterative updating, it is also slow, requiring substantial and repeated experience before behavioral adjustments. Tasks that require rapid shifting between behavioral strategies have been used to distinguish model-based and model-free components behaviorally and neurobiologically in humans and animals. Examples of such tasks are devaluation or motivational shift experiments, whereby the signature of habitual responding is a continued responding for an outcome that is no longer desired. This is reminiscent of drug taking persisting in the face of negative consequences.

Numerous studies in animals have shown that drugs of abuse shift the balance towards habits. Studies using for instance devaluation paradigms [4–7] have shown persistence of responding increasing with alcohol. The processes that speed up habituation have been suggested to also facilitate transformation into compulsions [8–13]. Work in humans with addictions has also shown evidence of inflexible choices, even in terms of non-drug-related rewards such as monetary gains. For instance, alcohol- [14] and stimulant-dependent [15–17] patients show impairments in shifting their responses in probabilistic reversal learning and fail to adapt their responses after errors in stop signal tasks [18, 19]. Moreover shifts towards automatic action tendencies temporally precede relapses [20] and can be trained to improve treatment outcome [21]. There have also been attempts to directly examine how substance dependence affects the relationship between goal-directed and habitual control in humans. A satiety devaluation paradigm did not reveal evidence of habitual responding for either cigarettes or chocolate in smokers, suggesting that not all responses for drug-related stimuli are necessarily habitual in drug users [22]. Interestingly, however, alcohol expectancy [23] and acute alcohol administration [24] did reduce the effect of satiety devaluation, suggesting that alcohol can specifically impair goal-directed decisions. Indeed, the goal-directed system is likely to depend on the kind of cognitive processes that are known to be impaired by alcohol [25, 26]. Sjoerds et al. [27] used an instructed devaluation in a slip of action tasks which involves complex relationships between stimuli, outcomes and responses. They found evidence for a shift towards habits in alcohol-dependent patients, which was accompanied by an increased functional magnetic resonance imaging signal in habit-related areas like the posterior putamen [28] and a decreased signal in goal-directed ventromedial prefrontal and anterior putamen areas [29–31], which parallels findings in animals [32].

Devaluation experiments involve single, sudden and large changes. On the one hand, these changes are obvious to human subjects and such salient changes might therefore lead to potent re-engagement of flexible goal-directed behavior. On the other hand, the slip of action task is very complex, and the performance of goal-directed decisions might be hampered by impairments affecting upstream cognitive functions including working memory [33]. We here use a third type of task [31] that has been developed based on computational arguments about the statistical efficiency of habitual and goal-directed systems [3]. It examines the relative contributions of habitual and goal-directed choices using continuous, subtle valuation shifts rather than few large or instructed ones. This task is also particular in that it allows us to compare the consequences of gains and nongains. Furthermore, given the sensitivity of goal-directed choices to cognitive load [25] and the recently reported importance of other cognitive measures [33, 34], we also examine whether any effects of alcohol dependence on the structure of decision-making might be accounted for by differences in more general cognitive measures.

Methods

Twenty-six recently detoxified alcohol-dependent patients (5 females) and 26 healthy comparison subjects (5 females) participated in this study. Demographic and clinical group characteristics of the final sample are outlined in table 1. Groups were matched for age, gender and years of education. All participants were examined for past and present psychiatric disorders using the Screening Version of the Structured Clinical Interview for DSM-IV [35]. All patients fulfilled DSM-IV criteria for alcohol dependence without axis I comorbidity. The days of alcohol abstinence before study participation in the patient group ranged from 2 to 39 with a mean of 15.1 ± 10.4 days. All healthy controls had no current or past major psychiatric disorder. All participants had normal or corrected-to-normal vision, and were also screened for neurological diseases. After detailed verbal and written instruction on the procedures of the study, participants gave their

Table 1. Demographic and clinical characteristics of 26 patients and 26 matched controls

	Number		Mean		t values	p
	ALC	HC	ALC	HC		
Age, years	26	26	44.0	43.3	$t_{50} = 0.304$	0.76
Education, years	26	25	11.0	11.5	$t_{49} = 1.39$	0.17
Verbal IQ (WST)	25	26	100.5	106.0	$t_{49} = 1.92$	0.061
Cognitive speed (DSST)	25	24	59.0	69.9	$t_{47} = 2.18$	*0.035*
Verbal working memory (DS)	25	25	6.8	7.5	$t_{48} = 1.06$	0.29
Executive functioning (TMT-A)	19	24	33.5	32.4	$t_{41} = 0.32$	0.75
Executive functioning (TMT-B)	19	24	74.5	72.3	$t_{41} = 0.18$	0.86
Verbal memory (word list)	19	24	8.2	8.9	$t_{41} = 1.14$	0.26

Each group included 5 females. Comparisons are based on independent-sample t tests. p values indicating statistical significance at a level less than 0.05 are displayed in italics.

ALC = Alcohol-dependent patients; HC = healthy controls; verbal IQ assessed by German vocabulary test (Wortschatztest [39]); cognitive speed assessed by Digit Symbol Substitution Test (DSST) from Wechsler WAIS-R [40]; verbal working memory assessed by digit span (DS) backwards test [40]; executive functioning assessed by trail making test (TMT) A and B [41]; verbal memory assessed by word list from the Consortium to Establish a Registry for Alzheimer's Disease [42].
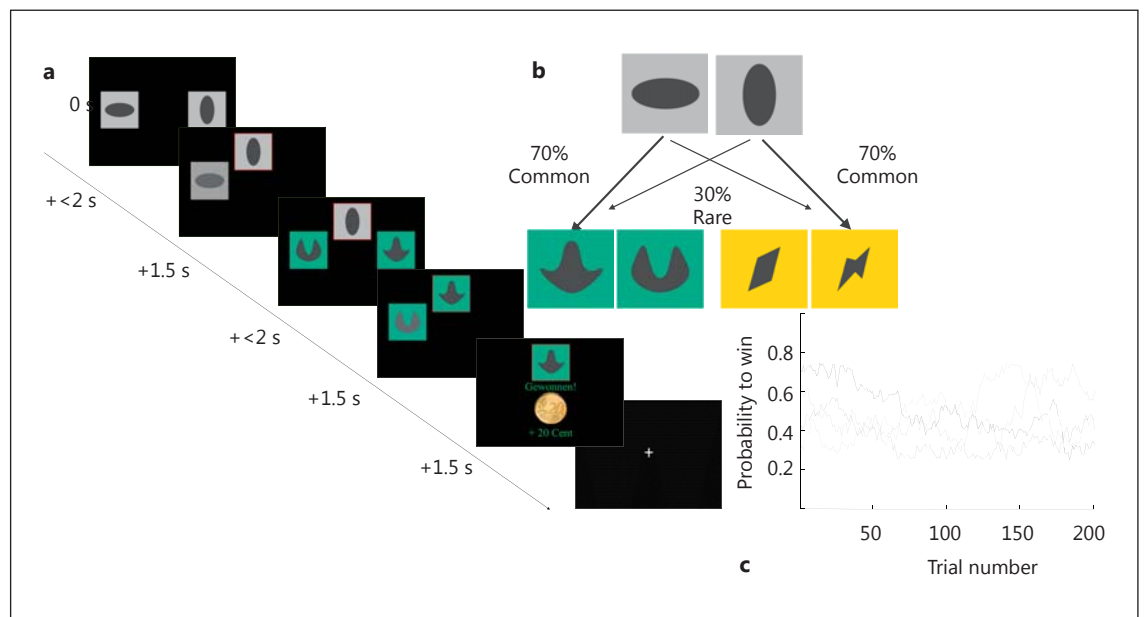

Fig. 1. a Trial configuration for the experiment. Each trial consisted of 2 different stages and each stage involved a choice between 2 stimuli. In the first stage, subjects chose between 2 abstract stimuli on a gray background. The chosen stimulus was highlighted by a red frame and moved to the top of the screen, where it remained visible for 1.5 s; at the same time, the other stimulus faded away. Subjects then reached a subsequent second stage. Here subjects saw 1 of 2 further pairs of colored stimuli and again chose between these. The monetary outcome following this second-stage choice (gain or no gain of 20 cent) was then presented centrally on the screen. **b** One pair of colored second-stage stimuli occurred commonly (on 70% of trials; 'common trials') after choice of one first-stage gray stimulus, while the other second-stage pair was equally strongly associated with the other first-stage stimulus (common). On the remaining 30% of trials, the chosen first-stage option resulted in a transition to the other second-stage stimulus pair (rare). **c** Reinforcement probabilities for each second-stage stimulus changed slowly and independently according to gaussian random walks with reflecting boundaries at 0.25 and 0.75. Win probabilities are displayed as a function of trial number, according to Daw et al. [31].

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Fig. 2. First-stage behavior as a function of reward and frequency of the previous trial for pure model-free versus pure model-based strategies. A pure model-free decision-making strategy would lead to a main effect of reward (**a**), but no effect of or interaction with frequency, while a pure model-based decision strategy would lead to an interaction between reward and frequency without a main effect of reward (**b**).
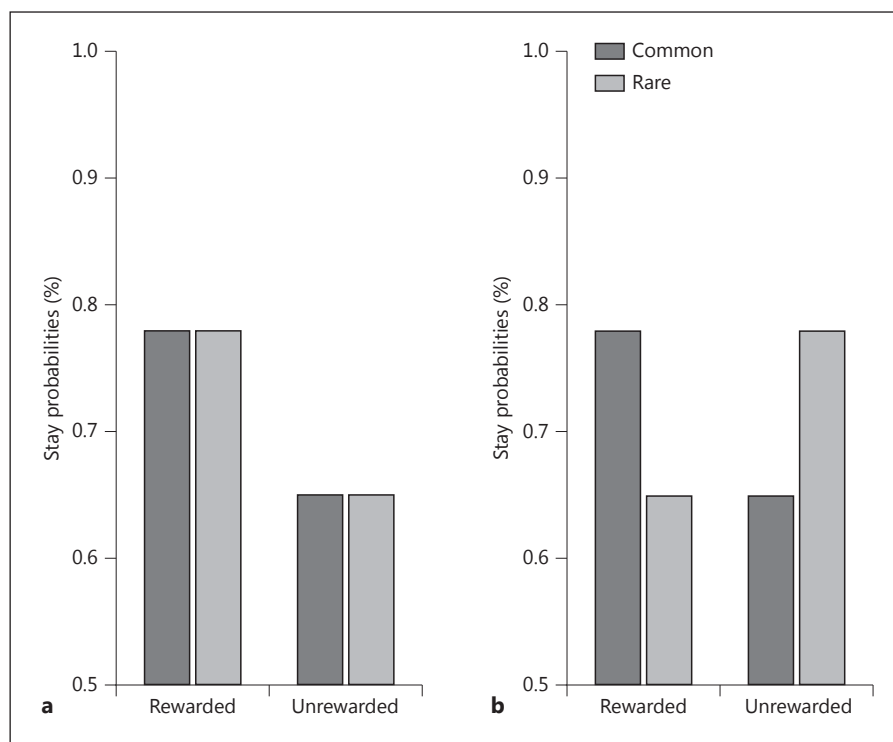
written informed consent. The study was approved by the local ethics committee of Charité Universitätmedizin Berlin and Universitätklinikum Dresden.

Task and Procedure

All participants underwent neuropsychological testing including standard tests assessing crystallized intelligence, cognitive speed, memory and executive functioning (table 1). Participants additionally performed the 2-stage Markov decision task as described by Daw et al. [31] in 2011 (see fig. 1 for a detailed task description).

The task was reprogrammed in MATLAB, using the Psychophysics Toolbox extensions [36, 37] and used different stimuli. Prior to the experiment, participants were explicitly informed about the task structure. Critically, the very detailed subject instructions were carefully translated from the English version. Participants were told that the transition matrix from first-step choices to second stages would stay constant and that the selection of one stimulus on the first stage would lead to a particular stimulus pair on the second stage more often than it would lead to the other second-stage stimulus pair. Participants were instructed that second-stage reward probabilities were independent of each other and would slowly change over the course of the experiment. Participants were familiarized with the paradigm before the task by performing a shortened version of the paradigm (50 trials) with different reinforcement probabilities and a different stimulus set. They were instructed to maximize their monetary outcome throughout the experiment. Participants' overall payout was EUR 13 plus the accumulated reward of one third of all trials. The maximal payout was limited to EUR 20. The task consisted of 201 tri-

als. Trials were separated by an exponentially distributed inter-trial interval, ranging between 1 and 7 s. The maximum response time was 2 s for first- and for second-stage choices. If participants failed to make a response in this time window, the German phrase for 'too slow!' appeared on the screen for 2 s, and the trial was aborted. The two stimuli at the first and second stage were assigned randomly between left and right from trial to trial.

Behavioral Analysis

We performed a simplified analysis focusing only on first-stage choices. Model-based and model-free strategies predict different patterns of first-stage choices. A model-free strategy predicts purely reinforcement-guided action selection: first-stage choices should be repeated after a previous trial's second-stage choice had resulted in reward whereas a first-stage switch should occur after a previous trial had ended up being not rewarded. Thus, model-free action selection should occur irrespectively of whether the transition to the second stage in the previous trial was a common or a rare one (fig. 2a). In contrast, model-based action selection includes the consideration of the task structure in its transition. This results in an inverted response behavior following rare trials. Consider a trial in which a first-stage response uncharacteristically results in a second-stage stimulus pair to which it usually does not lead (rare) and in which the second-stage selection is then rewarded. Model-based action selection would then predict a decreased probability of choosing this first-stage stimulus again, as the selection of the first-stage stimulus that has initially not been chosen has a higher likelihood of leading to the rewarded second-stage stimulus pair (common; fig. 2b).
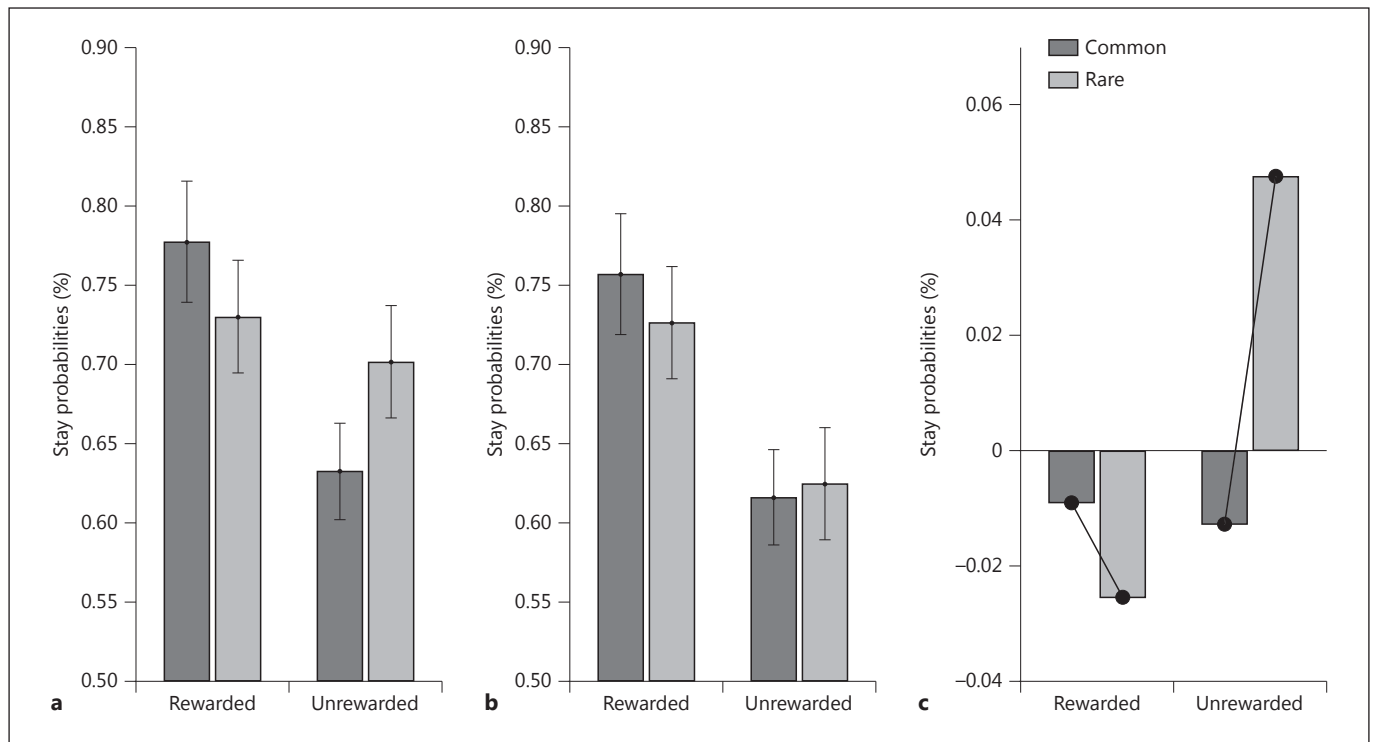
Fig. 3. Observed first-stage behavior as a function of reward and frequency of the previous trial for healthy controls (**a**) and alcohol-dependent patients (**b**). Error bars represent standard errors of means. **c** Standardized differences in stay probabilities between healthy controls and alcohol-dependent patients.

The major aim of this investigation was to specifically assess whether alcohol-dependent patients displayed a shift from model-based to model-free control. For this purpose we calculated 2 individual scores, one for model-based and one for model-free control. Individual model-free scores reflected the individual main effect of reward (% reward common + % rewarded rare – % unrewarded common – % unrewarded rare), whereas individual scores for model-based control reflected the interaction between transition frequency and reward (% reward common + % unrewarded rare – % rewarded rare – % unrewarded common). We computed a 1-tailed t test in each group (which compares both scores in each group against zero), in order to test whether both decision-making systems were evident within each group.

We then conducted independent-samples t tests to assess our a priori hypotheses that model-free contributions would be larger in alcohol-dependent patients than in healthy controls and conversely that model-based contributions would be larger in healthy controls than in alcohol-dependent patients. As we had explicit hypotheses on the direction of within-group and between-group effects of both model-free and model-based control we tested all comparisons regarding these hypotheses against a 1-tailed criterion.

In order to assess whether the neuropsychological variable that differed between groups (Digit Symbol Substitution Test, DSST) had an influence on model-free or model-based control we first regressed the outcome variables (total earnings and individual model-based scores) onto the neuropsychological variable, and performed independent-samples t tests on the residuals. All statistical analyses were performed using MATLAB version R2007a [38].

Results

Participants

Participant characteristics are shown in table 1. Patients and controls were matched for age, sex and education. Nevertheless, there was a significant difference in one measure of cognitive functioning (DSST, $p < 0.05$), and groups showed a tendency towards differing on verbal IQ (German vocabulary test, Wortschatztest: $p = 0.061$).

Participants rarely missed any responses (mean = 1.8%, SD = 2.43%). Missed trials were omitted from analysis. Patients and controls did not differ in their average stay/switch behavior at the first stage (no group difference in overall probability of stay/switch $t_{50} = 0.65$, $p = 0.52$).
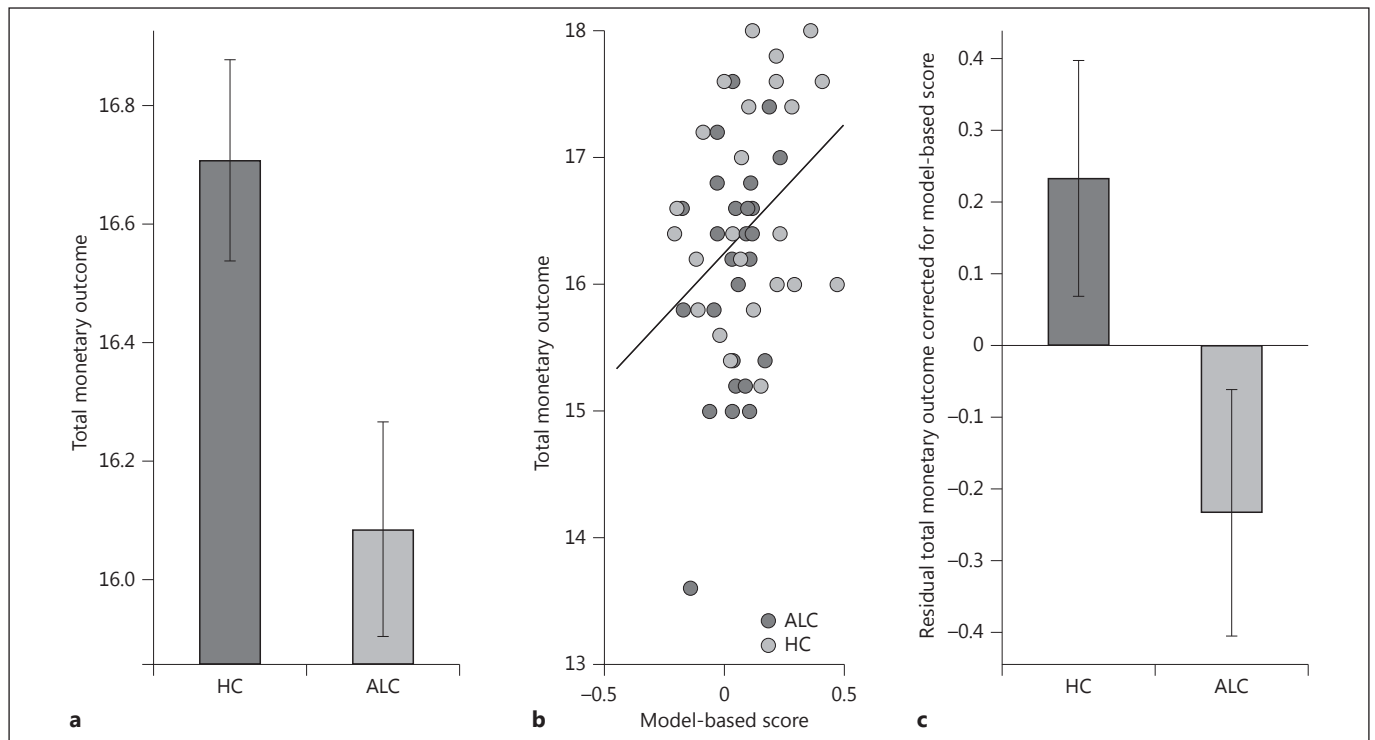
Fig. 4. Monetary outcome. HC = Healthy controls; ALC = alcohol-dependent patients. **a** Group differences in total monetary outcome. **b** Correlation between total monetary outcome and individual model-based scores. **c** Total monetary outcome corrected for differences in model-based scores.

Model-Free Choices

Both groups showed significant model-free influences in their choice behavior (model-free scores greater than zero, alcohol-dependent patient 1-tailed t test, $t_{25} = 5.03$, $p < 0.001$; healthy control 1-tailed t test, $t_{25} = 4.1$ $p < 0.001$). Thus, subjects tended to choose the same first-stage stimulus when rewarded in the previous trial, but switched to the opposing first-stage stimulus when not rewarded. Against our prediction, patients did not show a stronger model-free component (individual model-free scores alcohol-dependent patients vs. healthy controls 1-tailed t test, $t_{50} = 1.08$, $p = 0.14$).
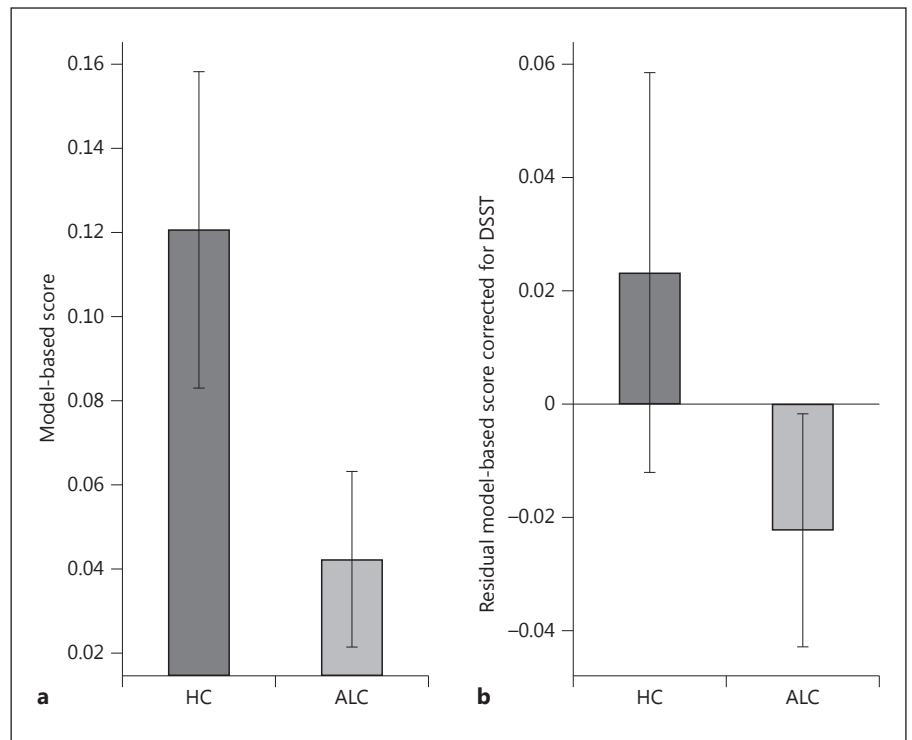
Model-Based Choices

Goal-directed components were present in both groups (individual model-based score greater than zero; 1-tailed tests: healthy control $t_{25} = 3.32$, $p < 0.001$, alcohol-dependent patient $t_{25} = 1.95$, $p < 0.05$). Hence, switches tended to occur when the outcome of the previous trial was a reward, but the transition rare, or when the outcome was a nonreward and the transition a common one. Healthy controls used significantly more model-based strategies than alcohol-dependent patients, as indicated by between-group differences in individual model-based scores ($t_{50} = 1.90$, $p < 0.05$, 1-tailed).

Figure 3 displays the stay probabilities for healthy controls (fig. 3a), alcohol-dependent patients (fig. 3b) and between-group differences in stay probabilities for all 4 trial types (fig. 3c). Visual inspection suggests that the groups mainly differed in stay probabilities after unrewarded trials. In order to test for differential between-group differences in model-based choice behavior for unrewarded and rewarded trials, we calculated individual model-based scores for unrewarded and rewarded trials separately and used independent-sample t tests to compare these between groups. These post hoc t tests confirmed that controls modulated their responses after losses according to a model-based strategy (i.e. were sensitive to transition frequency) more than patients (1-tailed test: $t_{50} = 2.27$, $p < 0.05$). However, model-based control after rewarded trials was not greater in controls than patients (1-tailed test: $t_{50} = 0.57$, $p = 0.28$). Thus, the difference in terms of model-free/model-

Fig. 5. Group differences in model-based scores (**a**) and model-based scores corrected for DSST (**b**). HC = Healthy controls; ALC = alcohol-dependent patients.

based choices between controls and patients was driven by a failure of goal-directed control after nonrewards in patients.

On average, subjects earned EUR 16.4 (total range 13.60–18.00). Patients earned less money than controls (2-tailed t test, $t_{50} = 2.53$, $p < 0.05$, fig. 4a). As the optimal response strategy exploits the structure of the task, model-based responding was expected to improve the overall outcome. Individual model-based scores were indeed positively correlated with the total monetary outcome (Pearson's r = 0.33, $p < 0.05$; fig. 4b). As expected, total outcome was not related to the score for model-free behavior (Pearson's r = –0.13, p = 0.36). However, the group differences in earnings remained marginally significant after correcting for model-based scores ($t_{47} = 1.96$, p = 0.06), indicating that goal-directedness only partially accounted for between-group differences in total monetary outcome (fig. 4c).

Effect of Cognitive Measures on Model-Based Behavior
Despite carefully matching for education, groups differed on the DSST – a measure of cognitive speed (table 1). We therefore asked whether the apparent group differences in model-based reasoning might instead be explained by group differences in the DSST. To correct

for the DSST, we again calculated 1-tailed independent-samples t tests after regressing out individual DSST scores. Although patients continued to be less model based, this correction removed the significant group difference ($t_{47} = 1.13$, p = 0.13; fig. 5a: group differences in model-based scores, fig. 5b: model-based scores corrected for DSST).

Discussion

The current study suggests a disruption of model-based choice behavior in alcohol-dependent patients. Importantly, we found no difference in the strength of model-free choice tendencies, and the disruption appears to be present after nonreward outcomes only. Despite carefully matching patients for educational level, patients and controls differed on one measure of cognitive speed. Patients were less model based than controls even after controlling for this, but the difference was no longer significant (p = 0.13).

The results are in line with theories suggesting a shift from controlled, goal-directed (outcome-guided) to automatic, habitual (stimulus-guided) decision-making in substance dependence [8–11]. They also speak to the critical question of whether chronic drug intake affects the

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goal-directed or the habitual system, or both [13, 43]. Crucially, inflexible, habitual action tendencies in substance dependence could derive from either an overactive habit system or an underactive goal-directed system or a change in the balance between these two systems [44]. Nonsequential tasks, such as devaluation paradigms, cannot address this question as they do not allow specifications on whether habitual action tendencies rather rely on amplified stimulus-response associations (habit system) or on an impairment to update or modify action-outcome contingencies (goal-directed system), or a combination of both. Thus, we here used a Markov decision task which allows precise specifications on the individual contribution of each system on decision-making. The results presented here suggest mainly an effect on goal-directed choices, particularly after nonrewards. This is in line with data showing orbitofrontal [45, 46] and action-outcome impairments in substance dependence [13] and with accounts emphasizing the importance of reasserting goal-directed control after losses or punishment [7, 47–49]. Interestingly, administration of L-dopa in this task selectively enhances goal-directed action selection on unrewarded trials [50]. This may speak to the profound alterations in the dopaminergic system in addiction [51–53]. Learning from nonrewards is known to rely on D_2 receptors in the indirect, inhibitory loop of the basal ganglia [54, 55]. While this has mainly been examined in the context of habitual, prediction-error-based learning, Frank's model also provides routes for influences on the prefrontal cortex via internal actions that update working memory [56].

The study identified a difference in the DSST between patients matched for educational level. This finding is in line with other studies demonstrating deficits in the DSST in alcohol-dependent patients [57, 58] and addiction disorders are well known not only to relate to striatal changes, but also decreased activation in the prefrontal cortex [53]. This may hint at an effect of alcohol on more general measures of cognitive functioning [58]. Goal-directed control has also been associated with prefrontal areas [27, 59, 60]. This raises the question of whether drugs directly affect goal-directed choices or do so only secondarily to their effects on other, more general cognitive functions. It will be important to also correct carefully for other measures of global function, such as working memory [25]. Indeed, confounding factors from even further afield may play a role, too. Stress, for instance, is known to affect the deployment of the goal-directed system [61] by decreasing activity of prefrontal regions [62], and it is not entirely implausible that patients may have felt more stressed during the experiment than controls. Moreover, it has been demonstrated that trait impulsivity is linked to decreased goal-directed control [63]. Given the finding that self-reported impulsivity tends to be increased in substance-dependent subjects [64, 65], this might be another potential mediator for the effects reported here.

We did not find a difference in model-free learning. Very detailed computational theories map phasic dopamine signals decisively onto model-free learning [66–69]. Given the impact of drugs of abuse on dopamine [70], the absence of this finding is rather striking. However, the failure to observe an effect on model-free learning is a negative effect. Similarly, the study was powered to detect a group difference in either of the two components, but not a difference between them, and we therefore did not ask whether the difference in the model-based component was larger than the difference in the model-free component. Hence these findings need to be treated with caution and require replication. Furthermore, while animal studies have convincingly shown a shift towards model-free choices in addiction, and indeed with alcohol [4, 5, 7], there have been only limited investigations to substantiate this in humans so far [23, 27]. One reason may be that it has been difficult to measure these two systems in humans and often has required rather laborious tasks involving extended training [71, 72]. While the current task is more subtle and measures devaluation in a more continuous way, one caveat is that it is not clear whether it fully differentiates between goal-directed and habitual components at a neurobiological level [31]. Moreover, it is yet unclear how different paradigms that have been designed to investigate dual-control mechanisms in humans indeed do examine a common psychological and neurobiological construct. Thus, further research should investigate within-subject correlations between performance in different dual-control tasks, such as paradigms that devalued outcomes by satiation [24, 61, 71] or by omission [72] and sequential learning tasks such as the 2-step task [31] or even more complex Marcov decision tasks [73].

We found that patients earned slightly less than controls. As a goal-directed strategy earns more, we initially thought this would be explained by the measure of goal-directedness. This was not so, suggesting that there might be further factors, possibly beyond the current model-free/model-based account affecting subjects' performance on the task. However, this need not necessarily be the case and in the future could be addressed by fitting the computational models of Daw et al. [31] and examining specifically the softmax terms.

Finally, it is yet unclear whether disruption in model-based control in alcohol-dependent subjects is caused by excessive chronic alcohol intake or rather reflects a predisposition to alcohol abuse. Evidence for alcohol-induced disruption of goal-directed behavior comes from demonstrations of devaluation insensitivity after chronic alcohol intake in rodents [4] and humans revealing disruption of goal-directed behavior [24] and inhibitory top-down control [74] after acute alcohol administration. As it has been shown that impairments in cognitive control serve as a vulnerability marker for an increased risk for substance dependence [65, 75–77], further research should use longitudinal designs in order to answer this chicken-and-egg question.

In conclusion, we have shown rather selective and specific effects of chronic alcohol intake on model-based reasoning, and highlighted that this might arise from the impact of chronic alcohol intake on more general cognitive functions. Further research should pay detailed attention to how impairments in the goal-directed system are related to prefrontal and cognitive functioning.

Acknowledgment

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