

Model-Based and Model-Free Control Predicts Alcohol Consumption Developmental Trajectory in Young Adults: A 3-Year Prospective Study

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ABSTRACT

BACKGROUND: A shift from goal-directed toward habitual control has been associated with alcohol dependence. Whether such a shift predisposes to risky drinking is not yet clear. We investigated how goal-directed and habitual control at age 18 predict alcohol use trajectories over the course of 3 years.

METHODS: Goal-directed and habitual control, as informed by model-based (MB) and model-free (MF) learning, were assessed with a two-step sequential decision-making task during functional magnetic resonance imaging in 146 healthy 18-year-old men. Three-year alcohol use developmental trajectories were based on either a consumption score from the self-reported Alcohol Use Disorders Identification Test (assessed every 6 months) or an interview-based binge drinking score (grams of alcohol/occasion; assessed every year). We applied a latent growth curve model to examine how MB and MF control predicted the drinking trajectory.

RESULTS: Drinking behavior was best characterized by a linear trajectory. MB behavioral control was negatively associated with the development of the binge drinking score; MF reward prediction error blood oxygen level-dependent signals in the ventromedial prefrontal cortex and the ventral striatum predicted a higher starting point and steeper increase of the Alcohol Use Disorders Identification Test consumption score over time, respectively.

CONCLUSIONS: We found that MB behavioral control was associated with the binge drinking trajectory, while the MF reward prediction error signal was closely linked to the consumption score development. These findings support the idea that unbalanced MB and MF control might be an important individual vulnerability in predisposing to risky drinking behavior.

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According to a recent cross-national study, the mean lifetime prevalence of alcohol use among the world's population is 80%. The average lifetime prevalence of alcohol use disorder (AUD) is 10.7% of that population (1), which indicates that AUD develops in only a portion of the population. Current theories about the predisposing factors of AUD point to trait impulsivity, anxiety, genetic factors, and novelty seeking along with their neural correlates [reviewed in (2–4)]. It is widely accepted that compulsive drug-seeking behavior involves a transition from choices based on action-outcome (goal-directed) to those based on stimulus-response (habitual) control (5–7). The imbalance of goal-directed and habitual control frequently results in compulsive drinking behavior (8), as tested in a cross-sectional design. As of yet, whether this imbalance predisposes to risky alcohol use in a longitudinal design remains untested.

Previous studies investigated how unbalanced goal-directed and habitual control was associated with compulsive drinking using the two-step sequential decision-making task (9). Developed from the reinforcement learning

framework, the two-step task assesses habitual and goal-directed behavior via model-free (MF) and model-based (MB) control, respectively. To elaborate, MF control computes and updates the action value based on the reward prediction error (RPE) signal, which has been linked to the dopaminergic neurons in the midbrain (10) and the blood oxygen level-dependent signal in the ventral striatum (VS) (11–13). In contrast, MB control examines all possible pairs of actions and outcomes based on decision trees (14), and it is sensitive to the structure of the task (9). Accordingly, MB prediction error reflects the surprise on entering a new state given the expectation based on the task model (12,13). To compare the two systems, MF control bases decisions on previously selected actions and is therefore inflexible, whereas MB control has more flexibility with respect to in-depth planning, but is more computationally expensive.

As evidenced by poor performance in a reversal learning task, patients with AUD were found to have an impaired MB control system. This was illustrated by behavioral deficits when challenged to integrate alternative choice options in flexible

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decision making (15). When associating AUD with the imbalance of MB and MF control, recently detoxified patients with AUD (3 weeks on average) were shown to use less MB strategy compared with healthy control subjects in a preliminary sample (16). Sebold *et al.* (17) further explored this topic with the full sample and attempted to predict treatment outcomes in recently detoxified patients with AUD with performance on the two-step task. Although MB behavioral control did not predict rates of relapse in the full sample, patients who relapsed showed reduced neural activation in the medial frontal cortex for MB control compared with healthy control subjects and patients who abstained. Conversely, Voon *et al.* (18) examined a detoxified AUD group with varying periods of abstinence (2 weeks to 1 year) and found no differences in strategies between the AUD group and the healthy control group. Nevertheless, a link likely exists between AUD and unbalanced MB and MF control.

Similar associations were also detected in nonclinical populations. Reduced MB control has been associated with binge drinking behavior (19) and the number of AUD symptoms in a large general population sample (20). A small study ($N = 20$) did not find reduced MB control in participants with a positive family history of alcohol dependence (21). Even though the previously mentioned studies demonstrated an association between alcohol consumption, binge drinking, and number of AUD symptoms with unbalanced MB and MF control cross-sectionally, it is not yet clear whether an imbalance between MB and MF control predisposes to risky alcohol use and AUD or evolves from repetitive alcohol consumption. We sought to clarify whether impairments in MB reasoning are a predisposing factor for risky alcohol use using a longitudinal design and a larger sample size. We were specifically interested in early risky alcohol use and binge drinking because they typically evolve as intermediate states during the transition from occasional social drinking into compulsive alcohol use. In our study, MB and MF control were assessed by the two-step task in a community sample of 18-year-old men. Their alcohol drinking behavior was recorded over the course of 3 years, from ages 18 to 21 years, considering that alcohol consumption in this sample is legally allowed at age 16, i.e., when risky drinking behavior typically escalates (22–25). Risky drinking during this period also leads to an increased chance of developing AUD during later stages of life (8). If MB and MF control could predict risky drinking trajectory during this period, it could then be considered one of the more crucial factors that predispose to pathological drinking.

Previously, we reported no association between MB and MF control and alcohol drinking behavior at age 18 in this sample (26). The current study investigated whether the two-step task performance at age 18 would predict the alcohol drinking developmental trajectory over the 3-year follow-up. We included both behavioral and neural predictors from the two-step task. For the neural predictors, we used both MB and MF RPE signals in the VS and ventromedial prefrontal cortex (vmPFC), as both regions have been shown to compute a mixture of the two RPE signals (9,26). Regarding the drinking behavior, we primarily constructed two drinking trajectories with latent growth curve models: a binge drinking score assessed by the quantity of alcohol intake per drinking occasion and a consumption score assessed by the sum of the first

three items of the Alcohol Use Disorders Identification Test (AUDIT). We hypothesized first that behavioral and neural correlates of MB control would be negatively associated with alcohol drinking trajectories over 3 years. Although previous studies have failed to find a clear association between MF control and AUD or risky alcohol use, a shift from MB to MF control could still be a predisposing factor—i.e., it could promote development of risky alcohol use and ultimately AUD and may not necessarily be maintained or identifiable by the time AUD has developed. Therefore, we further tested the hypothesis that behavioral and neural correlates of MF behavioral and neural control in the two-step task at baseline were associated with a steeper increase in alcohol drinking trajectory.

METHODS AND MATERIALS

Participants and Procedure

This study was part of a longitudinal prospective study to identify learning and decision-making mechanisms underlying dysfunctional alcohol consumption during early young adulthood in a community sample (ClinicalTrials.gov identifier: NCT01744834). Only men were recruited owing to the higher prevalence of risky drinking behavior in men compared to women. The recruitment procedure and inclusion/exclusion criteria are described in Section S-1 in the Supplement. At baseline, 201 participants completed the Munich-Composite International Diagnostic Interview (27,28) according to the German version of DSM-IV (29). Additionally, the participants performed the two-step task in the magnetic resonance imaging (MRI) scanner and partook in a cognitive ability assessment that examined working memory, processing speed, and crystallized intelligence (see details in Section S-3 in the Supplement).

Thereafter, all participants who completed the baseline assessment were invited to 6 follow-up evaluations over the course of the next 3 years. Regarding the key drinking behavior assessments, participants were asked to complete the AUDIT questionnaire online or to send the completed questionnaire via post every 6 months starting from the first follow-up. However, the AUDIT questionnaire was not available for the baseline assessment at age 18. The Munich-Composite International Diagnostic Interview was conducted in person at age 18 and via telephone at ages 19, 20, and 21.

Alcohol Drinking Assessment

We constructed the drinking trajectories with two variables of interest. The average alcohol intake per drinking occasion (grams of alcohol/occasion; binge drinking score) during the past year from the Munich-Composite International Diagnostic Interview assesses the amount of alcohol consumed on a typical drinking occasion. This variable was used as a proxy for binge drinking behavior or heavy drinking episodes (30,31). The AUDIT consumption score was used as second variable to construct drinking trajectories. The AUDIT consumption score assesses the frequency of drinking, the alcohol consumption in a typical drinking occasion, and the frequency of binge drinking. Further information on the rationale of choosing these two variables and descriptive statistics are given in Section S-2 in the Supplement, Table S1, and Figure S1.

In addition, we regressed the two variables against time points (modeled as categorical variables) to identify how the drinking behavior developed over the 3 years on the group level. To inspect the individual developmental trajectories, the individual intercepts and slopes (latent variables from the latent growth curve modeling [LGCM] model, which is described below) were extracted and plotted as histograms. The correlation between the two drinking variables was also calculated whenever they were assessed at the same time point (at ages 19, 20, and 21). Moreover, we also tested the correlation between the two individual intercepts and slopes of the binge drinking and consumption score trajectories. These correlation tests would then indicate whether the two variables assessed different aspects of drinking behavior and followed different developmental trajectories. Descriptive statistics of additional drinking variables are displayed in [Table S3](#).

Two-Step Paradigm

The two-step sequential decision-making task was performed in the MRI scanner ([Figure 1](#)).

Two-Step Data Analysis

Two-Step Behavioral Predictors. As suggested by Daw *et al.* (9), who originally described the two-step task, the pure MF agent tends to ignore the structure of the task by repeating

the first-stage choice after being rewarded on making the second-stage choice. Conversely, the pure MB agent considers the transition structures. The MF agent is thus sensitive to the effects of receiving a reward, as he chooses to stay after reward trials and switch after omission trials. The MB agent makes decisions based on the reward by transition interaction effect, as he tends to stay after rewarded common trials but switch after rewarded uncommon trials (and vice versa for the omission trials). It was suggested in our previous article that the participants adopted a combination MB and MF strategy (26). Therefore, we calculated two scores (MB_{score} , MF_{score}) for each individual according to his first-stage stay probability (P) across all trials. The purpose of these scores was to measure the degree that participants behaved like the pure MB and the pure MF agents. The two scores were then used as behavioral predictors for the alcohol drinking developmental trajectory. Specifically, they were calculated as follows:

$$MF_{score} = P(\text{stay}|\text{rewarded common}) + P(\text{stay}|\text{rewarded rare}) - P(\text{stay}|\text{unrewarded common}) - P(\text{stay}|\text{unrewarded rare})$$

$$MB_{score} = P(\text{stay}|\text{rewarded common}) - P(\text{stay}|\text{rewarded rare}) - P(\text{stay}|\text{unrewarded common}) + P(\text{stay}|\text{unrewarded rare})$$

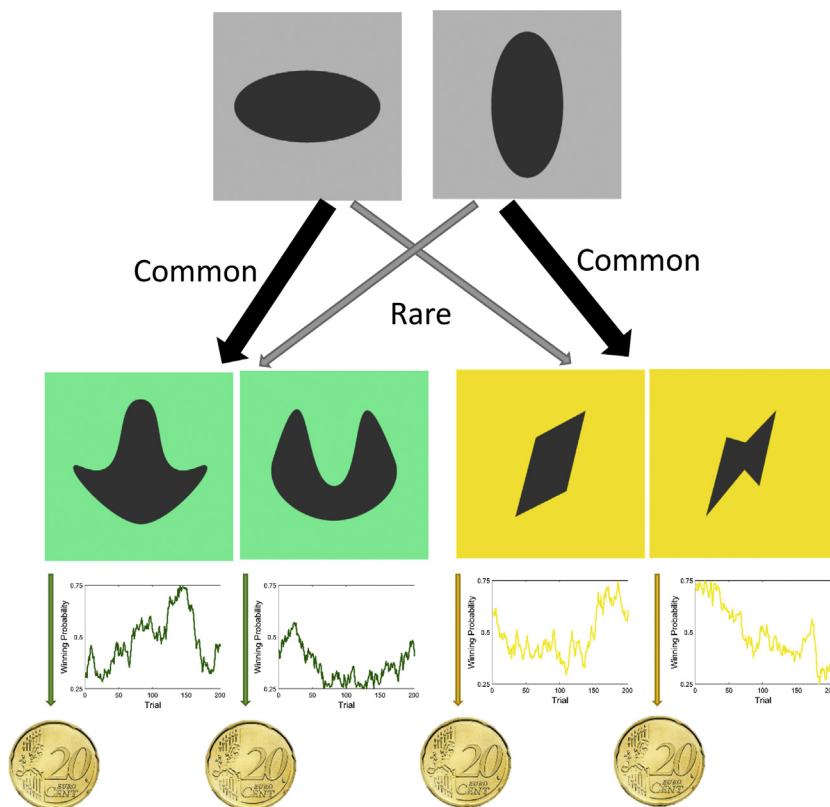


Figure 1. The two-step paradigm. The two-step sequential decision-making task (9) was performed in the magnetic resonance imaging scanner. The functional magnetic resonance imaging data acquisition and preprocessing procedures are described in detail in [Section S-5](#) in the [Supplement](#). The task consisted of 201 trials in total. In the first stage, the same pair of gray boxes with oval shapes inside were shown. Participants were asked to select one of these boxes within 2 seconds. The choice between the two first-stage stimuli would then lead to one of the second-stage pairs: the common transition (with a probability of 70%) or the rare transition (with a probability of 30%). The transition probability from the first to the second stage was fixed throughout the task, and participants were informed about this. In the second stage, one of the two pairs of stimuli were presented (either yellow or green) based on the first-stage choice and the transition probability. Participants were again asked to select one of the second-stage colored stimuli within 2 seconds. The selected second-stage stimulus led to the monetary reward of 20c (€0.20) with a reward probability ranging from 25% to 75%, which was slowly changing across the experiment according to Gaussian random walks. In exchange for their time and cooperation, participants were paid according to the total monetary rewards acquired in one third of the trials that were randomly drawn from all trials.

Two-Step Neural Predictors. A total of 146 participants were included in the final fMRI analysis after quality control [same as in Nebe *et al.* (26)]. The fMRI first-level model is the same as our baseline report; one onset regressor was specified for the second-stage onset, with MB RPE and MF RPE modeled as two parametric modulators (see details in Section S-6 in the Supplement). To assess the neural correlates of MB and MF RPE signal, we performed one-sample *t* tests on both MB and MF RPE parametric regressors on the second level. Consistent with previous studies (9,26), two regions of interest were specified: the bilateral vmPFC and bilateral VS (based on meta-analyses; see Section S-6 in the Supplement). Both the VS and vmPFC have been suggested to compute a mixture of MB and MF RPE signals, and these cannot be disentangled in the two-step task (9). It was for this reason that the mean parameter estimates within the two regions of interest were extracted separately for both MB and MF RPE parametric regressors. The four neural predictors were then applied to predict the alcohol drinking developmental trajectory.

LGCM Analysis

LGCM offers an elegant framework to model both intraindividual and interindividual change over time (32). Traditional approaches, such as analysis of variance, treat individual differences as variances. In contrast to analysis of variance, though, LGCM additionally models the intraindividual change. As a multilevel model, intraindividual change in drinking behavior with respect to time was modeled on the first level. Thus, one intercept and one slope can characterize an individual's drinking behavior when a linear developmental trajectory is assumed. Different individual drinking developmental trajectories can be identified accordingly. Based on our hypothesis, individual drinking trajectories were modeled for the aforementioned variables: gram/occasion and AUDIT consumption score. Additionally, a model comparison was performed between quadratic and linear models to decide if adding a quadratic term to the model would improve the model fit. It was found that the model fit of the quadratic trajectory models was worse than the linear trajectory models (see details in Section S-7 in the Supplement). Therefore, we tested only the predictors from the two-step task in the linear trajectory models.

At the second level, predictors of interest could be included in the model. The model would then decide whether these predictors are associated with the interindividual differences in their developmental trajectories, as indicated by the individual intercepts and slopes from the first level (when the linear trajectory model fits better than the quadratic model). Our model included six predictors of interest: MB and MF behavioral scores and MB and MF neural signals in the VS and the vmPFC, respectively. Freely estimated covariances were allowed between MB and MF behavioral scores, between MB neural signals, and between MF neural signals. The two models of interest are displayed in Figure 2. In addition, executive functions (33) and impulsivity (21,34) are thought to be associated with two-step task performance. We also found associations between the MB behavioral control, working memory capacity, and processing speed in our sample (see Section S-3 in the Supplement and Table S2), but no

association was found between the two-step predictors and impulsivity measured by the Barratt Impulsiveness Scale sum score (26). Nevertheless, to control for the potential effect of these factors on the models, these variables were included separately in the two models to check whether they had an effect. Additionally, because we previously reported that low MB control is associated only with increased risk for relapse in patients with AUD with high alcohol expectancies (17), we explored whether such an interaction between MB control and alcohol expectancies also existed in our sample. The detailed analyses and results are shown in Section S-11 in the Supplement.

RESULTS

Drinking Trajectories

According to the two linear regressions of the two drinking scores against time points, the AUDIT consumption score did not change with time on the group level ($\beta = -0.06$; $p = .26$), but the binge drinking score (gram/occasion) significantly decreased over time ($\beta = -8.54$; $p = 3.81 \times 10^{-7}$). However, as can be seen in the trajectory plots and the histograms of individual intercepts and slopes (Figure 3), individuals exhibited different developmental trajectories within the 3-year time course even without overall significant changes. The two drinking scores correlated with each other early on but tended to develop independently over time (correlations shown in Figure 3).

LGCM Results

The AUDIT consumption score model (Figure 2A) demonstrated a good model fit ($\chi^2_{48} = 81.12$, $p = .002$, comparative fit index = 0.956, root mean square error of approximation = 0.072, standardized root mean square residual = 0.078). The path parameter estimates are shown in Table 1. Among the predictors, we found that the MF VS signal was positively associated with a change in AUDIT consumption score over time (slope), while MF vmPFC activation was positively associated with AUDIT consumption score in the 6-month follow-up (intercept). The association between MF behavioral score and slope was also positive, but this effect was only marginal ($p = .055$).

The binge drinking model displayed in Figure 2B showed a good model fit as well ($\chi^2_{27} = 50.26$, $p = .004$, comparative fit index = 0.935, root mean square error of approximation = 0.077, standardized root mean square residual = 0.084). As displayed in Table 1, we found that the two-step MB behavioral score was negatively associated with the developmental trajectory (slope) of the gram/occasion variable over the past year. The four neural predictors and the MF behavioral score did not show significant associations with either the intercept or the slope of the gram/occasion during the last year. Additional exploratory analyses with alcohol expectancies showed that only individuals with high expectations of the positive reinforcing effect of alcohol showed the negative association (see details in Section S-11 in the Supplement).

Additionally, the individual latent intercepts and slopes were extracted from the two models and plotted against the significant predictors for the purpose of illustration (Figure 4). The

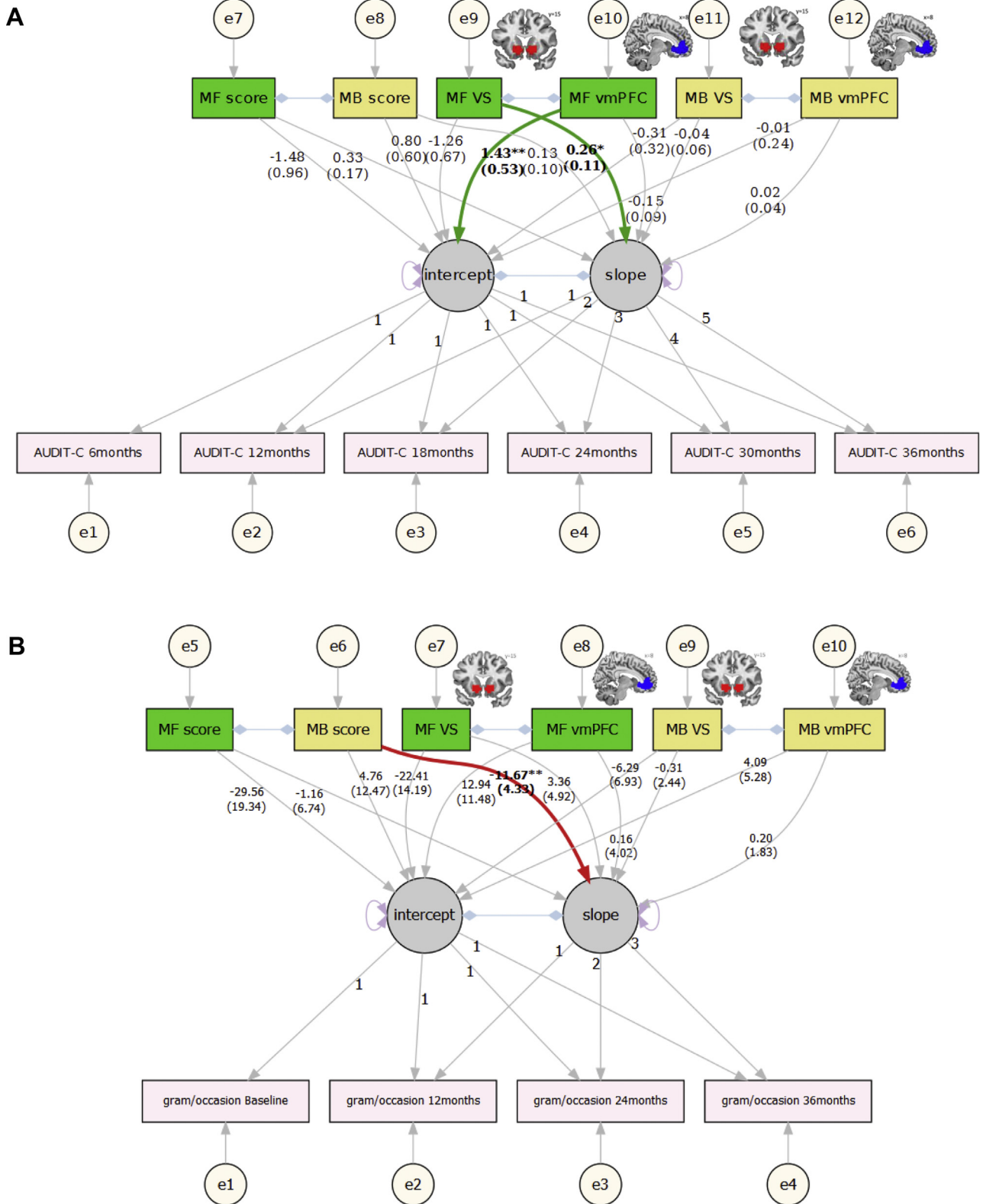


Figure 2. Latent growth curve modeling structure. Alcohol Use Disorders Identification Test consumption score (AUDIT-C) model **(A)** and gram/occasion model **(B)**. The intercept and slope were modeled as the latent variables. All the other variables were observed from the data. The loadings from the intercept and slope to the drinking variables were fixed with values shown in the figure, indicating the linear trajectory. All the other paths including regressions,

control variables—executive functions and impulsivity score—neither changed the model estimates nor showed significant associations with the intercepts or slopes. The detailed results are shown in [Tables S5 and S6](#).

DISCUSSION

With a large community sample, we found that an unbalanced MB and MF control assessed by the two-stage sequential decision-making task at the age of 18 predicted the developmental trajectories of the binge drinking and consumption scores during young adulthood. Specifically, MB behavioral control was associated with less increase or more decrease in the developmental trajectory of binge drinking behavior. Concerning the consumption score assessed by the AUDIT questionnaire, the neural MF RPE signal in the vmPFC and the VS predicted a higher starting point and steeper increase/flatter decrease over time, respectively. All the identified associations had medium effect sizes (explaining 15%–23% of variance). We thus conclude that a bias away from MB and toward MF control may represent a critical mechanism predisposing toward risky alcohol drinking during young adulthood.

Interestingly, we found that MB and MF control predict different aspects of drinking behavior. The binge drinking trajectory (i.e., slope of gram/occasion variable) was negatively associated with the MB behavioral score. Binge drinking has recently been related to deficits in executive functions, such as poor inhibitory control during adolescence and young adulthood (35,36). Moreover, young binge drinkers are comparable to patients with severe AUD in their executive control abilities (37); binge drinking has also been suggested to be a consequence of the effect of alcohol on the brain networks underlying inhibitory control in young adults (38). Consistent with a previous study (33), the MB behavioral score in our sample was also associated with several facets of executive function, including processing speed, working memory capacity, and verbal intelligence. However, these executive functions per se neither predicted the drinking trajectory nor affected the model estimates. Taken together, the MB score may be closely linked to executive function but explains additional variance of binge drinking behavior. It is worth mentioning that in our sample binge drinking decreased between the ages of 18 and 21. High MB control may work as a protective mechanism by further decreasing binge drinking over time.

Notably, the MB neural signal was not associated with binge drinking behavior. On one hand, this may be due to the noise in the neural signals, which might not reliably capture the trial-by-trial MB control. On the other hand, MB behavior was not necessarily guided by the MB RPE defined in the current computational model, which was also pointed out by Daw *et al.* (9). The MB control in the current task tracks transition

probabilities and immediate rewards. Another way of defining the MB control posits that the state prediction error signal can be tracked by examining future planning and calculating cumulative future rewards (12,13). However, whether this type of MB prediction error signal was computed or associated with the MB behavioral control cannot be tested with the current task design. Owing to this discrepancy, the MB behavioral predictor may capture different aspects of the MB control and predict future binge drinking behavior better than the neural signals.

The consumption score trajectory, assessed by the first three items of the AUDIT questionnaire, was predicted by the MF RPE signals in the VS and the vmPFC. Additionally, a weaker positive association between the MF behavioral score and the development (i.e., slope) of risky alcohol use was identified at a trend level. So far, only a limited number of factors associated with alcohol consumption have been identified that are not specific to binge drinking. Two cross-sectional electroencephalography studies have found associations between higher alcohol consumption with attenuated feedback-related negativity amplitudes (39,40) and a feedback-locked P3 component (39). These two event-related components indicated the RPE signals after receiving rewards. Intriguingly, neither the feedback-related negativity nor the P3 component was found to be related to binge drinking behavior when tested with the same balloon analog risk task as used in Soder *et al.* (40). Therefore, we propose that the consumption score, but not specifically binge drinking, may be associated with aberrant RPE processing in the brain. In line with this, higher gray matter volume in the caudate nucleus at age 14 was found to predict a steeper increase in AUDIT score over a 5-year period (41). Although the intercept of binge drinking and the AUDIT consumption score were positively correlated, we did not observe an association between the MF vmPFC signal and the binge drinking intercept. This suggests that the frequency of drinking as well as of binge drinking assessed with the AUDIT in addition to mere amount of drinking per occasion may also play an important role.

Essentially, our results were intrinsically consistent, though indicated by different parameters. Lower MB or higher MF control indicated riskier drinking trajectories. As discussed above, MB and MF control seem to predict different facets of drinking behavior. The associations between MB control and drinking were significant only for the behavioral indicator, whereas the association between MF control were significant only for the respective neural signatures. One explanation is that some predictors might not have been identified because effect sizes are smaller (e.g., MF behavioral score) or are due to lower reliability compared with the ones identified. The MB neural signal, for example, may be noisy, which means that an even larger sample would be required to discern any effects. Furthermore, MB control could also be promoted by more

variances, and covariances were freely estimated from the model. Latent growth curve modeling was modeled within the structural equation model framework using the lavaan package in R (45). The lavaan package allows for the handling of missing data with full information maximum likelihood, which estimates a likelihood function for each individual based on the available information. This method is suggested to be unbiased, as the missing data are assumed to be random (46). For path estimation, as indicated by the green paths, the model-free (MF) ventromedial prefrontal cortex (vmPFC) and the MF ventral striatum (VS) signals were positively associated with the intercept and slope, respectively, in the AUDIT-C model; the model-based (MB) behavioral score was negatively associated with the slope of the gram/occasion (binge drinking) model. The standardized estimates are displayed in [Table 1](#).

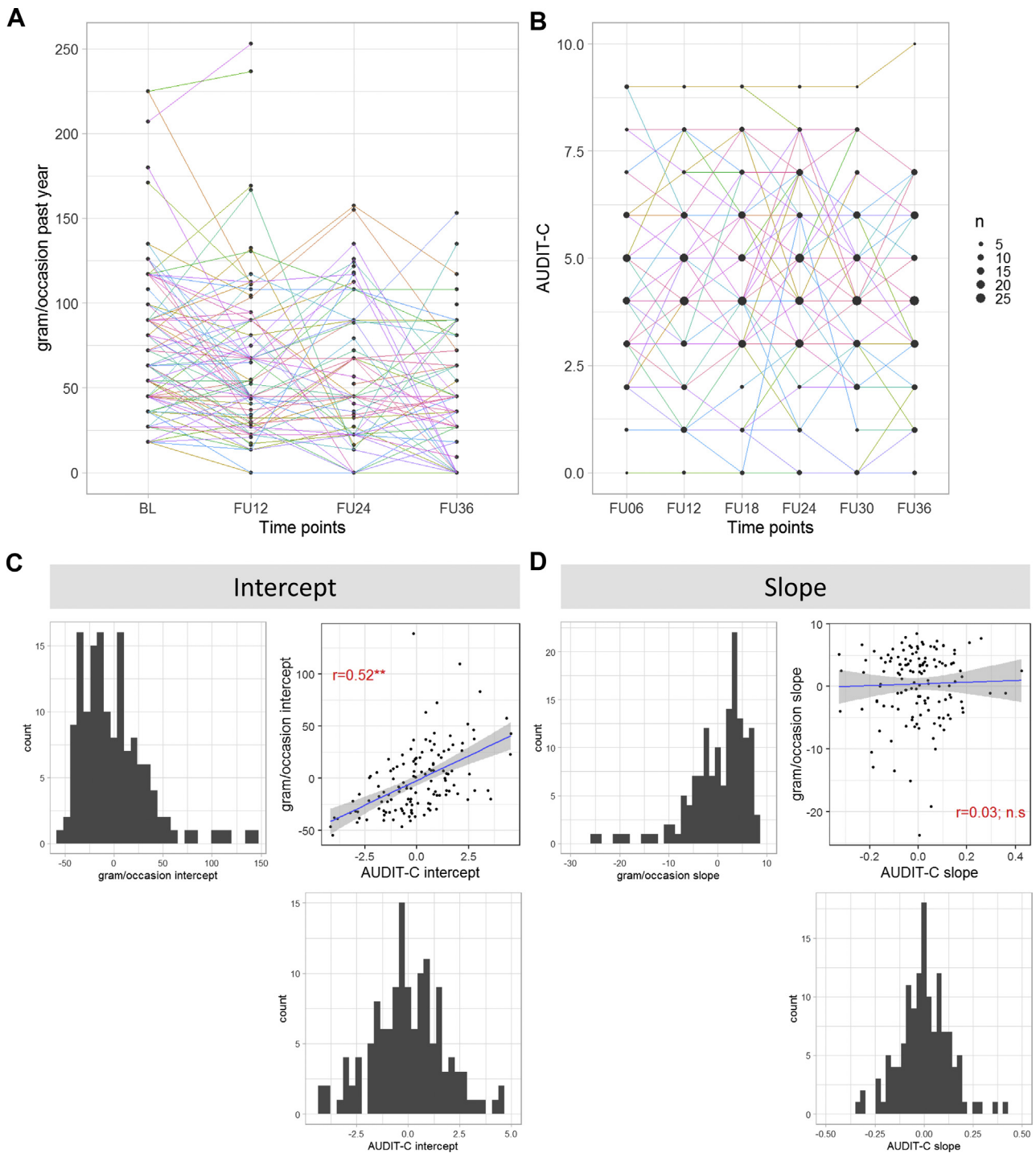


Figure 3. (A, B) Individual trajectories (indicated by different colors) of gram/occasion and Alcohol Use Disorders Identification Test consumption score (AUDIT-C) variables across different time points. Individual differences in the developmental trajectories within the 3 years can be seen. The measure of gram/occasion during the last year yielded 4 time points; the AUDIT-C was assessed every 6 months after baseline (BL) and yielded 6 time points (FU06, FU12, FU18, FU24, FU30, FU36). A total of 146 participants with valid data were included in the gram/occasion trajectory. We further excluded 13 participants who lacked valid AUDIT assessments over the 3 years. The correlation between the gram/occasion variable and AUDIT-C was moderate at age 19 ($r_{77} = .49$, $p = 5.07 \times 10^{-6}$), strong at age 20 ($r_{74} = .61$, $p = 4.58 \times 10^{-9}$), but low at age of 21 ($r_{97} = .29$, $p = 3.46 \times 10^{-2}$). (C, D) The individual intercepts from the 2 drinking models showed a significant correlation ($r_{131} = .52$, $p = 1.35 \times 10^{-10}$), but the 2 slopes were not correlated ($r_{131} = .03$, $p = .73$). n.s., not significant.

Table 1. LGCM Results

	MF/MB	Path	Estimate (Unstandardized)	SE	Estimate (Standardized)	z	p Value	Effect Size ^a (r ²)
AUDIT Consumption Score								
Intercept	MF	Behavioral score	-1.476	0.960	-0.156	-1.537	.124	2.4%
		VS signal	-1.257	0.669	-0.239	-1.880	.060	5.7%
		vmPFC signal	1.428	0.534	0.341	2.675	.007 ^b	15.3%
	MB	Behavioral score	0.801	0.600	0.139	1.337	.181	3.6%
		VS signal	-0.307	0.322	-0.131	-0.952	.341	1.7%
		vmPFC signal	-0.011	0.243	-0.006	-0.044	.965	0.0%
Slope	MF	Behavioral score	0.327	0.171	0.302	1.918	.055	12.4%
		VS signal	0.259	0.113	0.429	2.286	.022 ^c	22.9%
		vmPFC signal	-0.151	0.093	-0.314	-1.628	.104	9.9%
	MB	Behavioral score	0.131	0.104	0.198	1.268	.205	6.2%
		VS signal	-0.035	0.057	-0.130	-0.618	.537	1.7%
		vmPFC signal	0.018	0.042	0.090	0.419	.675	2.0%
Binge Drinking Score (Grams Alcohol/Drinking Occasion) Past Year								
Intercept	MF	Behavioral score	-29.560	19.335	-0.151	-1.529	.126	2.3%
		VS signal	-22.413	14.188	-0.202	-1.580	.114	4.1%
		vmPFC signal	12.944	11.481	0.144	1.127	.260	3.8%
	MB	Behavioral score	4.755	12.465	0.039	0.381	.703	0.8%
		VS signal	-6.291	6.927	-0.129	-0.908	.364	1.7%
		vmPFC signal	4.094	5.278	0.113	0.776	.438	2.7%
Slope	MF	Behavioral score	-1.159	6.735	-0.030	-0.172	.863	0.1%
		VS signal	3.359	4.918	0.152	0.683	.495	4.1%
		vmPFC signal	0.157	4.017	0.009	0.039	.969	0.3%
	MB	Behavioral score	-11.662	4.329	-0.483	-2.694	.007 ^b	23.3%
		VS signal	-0.309	2.435	-0.032	-0.127	.899	0.1%
		vmPFC signal	0.196	1.828	0.027	0.107	.915	0.6%

AUDIT consumption score model fit: $\chi^2_{48} = 81.12, p = .002, CFI = 0.956, RMSEA = 0.072, SRMR = 0.078$; Binge drinking score past year model fit: $\chi^2_{27} = 50.26, p = .004, CFI = 0.935, RMSEA = 0.077, SRMR = 0.084$.

AUDIT, Alcohol Use Disorders Identification Test; CFI, comparative fit index; MB, model-based; MF, model-free; LGCM, latent growth curve modeling; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual; vmPFC, ventromedial prefrontal cortex; VS, ventral striatum.

^aEffect size is displayed as the percent of explained variance (r²). Correlation coefficients (r) were converted from the standardized coefficient according to Peterson and Brown 2005 (47) by using the equation $r = \beta + .05\lambda$, where λ equals 1 when $\beta > 0$ and equals 0 when $\beta < 0$.

^b $p < .01$.

^c $p < .05$.

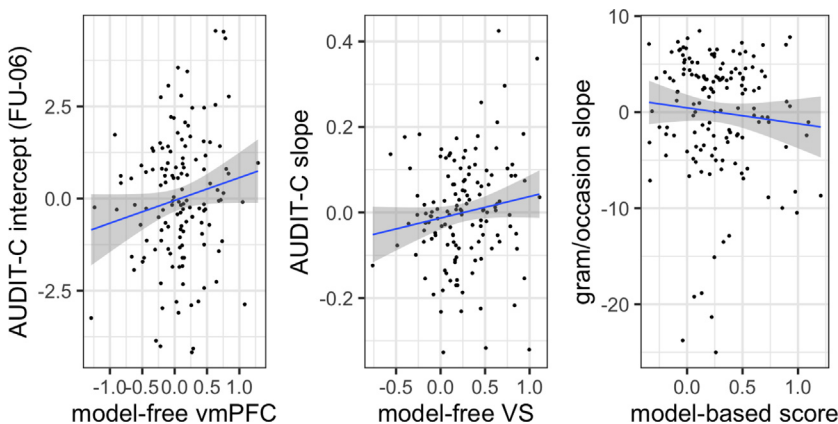


Figure 4. Illustration of the significant paths from latent growth curve modeling. The model-free neural reward prediction error signal in the ventral striatum (VS) predicted higher Alcohol Use Disorders Identification Test consumption score (AUDIT-C) intercept (6 months following the baseline, FU-06). The model-free neural reward prediction error in the ventromedial prefrontal cortex (vmPFC) predicted an increase/less decrease of AUDIT-C over the 2.5 years. The model-based behavioral score was negatively associated with the slope of the gram/occasion variable.

detailed task instructions (42). Therefore, when participants misconceive the task, there might also be a mismatch between the strategies that participants used and the strategy captured by the model. Taken together, larger sample sizes, an improved version of the paradigm (43), and improved parameter estimates (44) may potentially resolve such discrepancies.

Limitations

Although MB and MF control were found to predict risky drinking during the 3-year follow-up, we do not have any information about whether the participants with risky drinking trajectories would develop AUD in a later phase of life. This would require a longer follow-up period, as direct evidence is needed. Additionally, we assumed that the missing data are at random, but we could not test whether other factors contributed to participants dropping out of the study. Given that the missing rates are 30%–40% at almost every time point, computational methods had to be applied to preserve the data. Nevertheless, we did not reach the current conclusions without the assumptions about the missing data. Also, the AUDIT was first assessed 6 months after the baseline. We thus could not infer the association between the two-step predictors and general risky alcohol consumption at baseline, but rather only 6 months later. Lastly, this study included only male participants, and therefore the results cannot be generalized to non-male populations.

Conclusions

By assessing two modes of instrumental learning (i.e., MB and MF learning) and recording the drinking behavior of a large cohort of young men over a period of 3 years, we were able to identify predictors of risky alcohol use. Our data reveal that a higher MB behavioral score predicts a decrease in binge drinking, while a higher MF RPE neural signal predicts a higher AUDIT consumption score that further increases over time. Our findings may also suggest that the AUDIT consumption score and binge drinking trajectories may develop differently during young adulthood and involve different mechanisms. Dysbalanced control might ultimately also predispose to the later development of AUD, but the duration of the follow-up and the limited sample size of this study do not allow drawing conclusions yet. We propose that future studies could further examine these links by carefully assessing different aspects of alcohol consumption in larger cohorts and over longer periods of time. To better comprehend the link between the unbalanced MB and MF control and (pathological) alcohol use, another direction for future studies is to investigate the consequences of drinking, i.e., whether alcohol consumption further changes the MB and MF control. Lastly, the current study also opened a new door for future studies to develop interventions to target these proposed mechanisms in preventing risky alcohol use.

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ARTICLE INFORMATION

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