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RESEARCH ARTICLE

Effects of alcohol intake on time-based event expectations

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Abstract Previous evidence suggests that alcohol affects various forms of temporal cognition. However, there are presently no studies investigating whether and how alcohol affects on time-based event expectations. Here, we investigated the effects of alcohol on time-based event expectations. Seventeen healthy volunteers, aged between 19 and 36 years, participated. We employed a variable foreperiod paradigm with temporally predictable events, mimicking a computer game. Error rate and reaction time were analyzed in placebo (0 g/kg), low dose (0.2 g/kg) and high dose (0.6 g/kg) conditions. We found that alcohol intake did not eliminate, but substantially reduced, the formation of time-based expectancy. This effect was stronger for high doses, than for low doses, of alcohol. As a result of our studies, we have evidence that alcohol intake impairs time-based event expectations. The mechanism by which the level of alcohol impairs time-based event expectations needs to be clarified by future research.

Keywords Alcohol · Timing · Associative learning · Time-based expectancy · Attentional control

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Introduction

Alcohol effects on temporal cognition

The effects of alcohol intake are well documented for the majority of our perceptual, motor and cognitive functions. For example, motor functions and general cognitive capacity decline after alcohol intake (Brumback et al. 2007; Guillot et al. 2010); alcohol strongly affects attention, target stabilization, visual short-term memory and temporal processing (Abroms and Fillmore 2004; Abroms et al. 2006; Ivanec et al. 2009; Khan and Timney 2007; Kunchulia et al. 2012; Patel et al. 2010; Saults et al. 2007; Wegner and Fahle 1999).

Previous studies have demonstrated that the perception of time is also affected by alcohol intake (Ogden et al. 2011; Terry et al. 2009; Tinklenberg et al. 1976). Ogden et al. (2011), for example, have shown that a high dose of alcohol increased the speed of internal pacemakers in temporal processing, leading to over-estimations of duration in prospective timing tasks. Interestingly, these authors also found some improvements in timing ability after alcohol consumption in a temporal generalization task, but only when a short duration range was employed (Ogden et al. 2011). In addition, Terry et al. (2009) found that alcohol affects time perception and production in different directions, depending on dosage. A low dose of alcohol improved temporal discrimination in a time perception task. In contrast, a higher dose of alcohol caused bimanual tapping task performance to deteriorate, as evidenced by increasing timer variability, whereas motor variability was unaffected. However, there was no effect of alcohol on coordination timing in a grip and lift task (Terry et al. 2009). Taken together, these results suggest that the effects

of alcohol on timing ability essentially depend on the dosage of alcohol, on the duration ranges applied and on the task itself.

The number of studies suggests that impacts of psychoactive drugs on timing would be useful for better understanding theories of timing (Terry et al. 2009). For example, in accordance with their findings that alcohol had no effects on the retrospective timing, Ogden et al. (2011) followed that retrospective judgments were based on other internal timing systems than prospective timing.

Time-based expectancy

Effects of alcohol on various types of temporal cognition have been intensely investigated in the previous literature. These studies have in common that they investigate the ability to estimate the duration of a certain interval. This ability is usually referred to as timing ability. A fundamentally different, but also very important, aspect of human temporal cognition is time-based expectancy. In time-based expectancy, one does not expect a temporal interval as such, but one expects an event, *based* on the duration of an interval. Time-based expectancies are essential for human behavior and have an impact on many types of interaction environments, such as verbal communication and human–machine interaction (Thomaschke and Haering 2014).

The paradigm typically used to study time-based event expectancy is a certain variant of the foreperiod paradigm (Niemi and Näätänen 1981)—namely, the time–event correlation paradigm (Wagener and Hoffmann 2010, see Thomaschke and Dreisbach 2015, for a detailed overview). In a foreperiod paradigm, target stimuli are preceded by a preparatory interval—the foreperiod—which can vary in duration, either from trial to trial or between blocks. A common finding is that response time (RT) decreases with foreperiod duration when foreperiod varies randomly between trials. This effect is referred to as the foreperiod effect (Los and van den Heuvel 2001; Los et al. 2001; Steinborn et al. 2008, 2009). In the time–event correlation paradigm, two target stimuli and two foreperiods appear equally often overall, but one of the targets is paired with the short foreperiod and the other target with the long foreperiod, in 80 % of the trials. The formation of a time-based event expectation leads to faster responses to frequent foreperiod–target combinations, relative to infrequent ones (Thomaschke et al. 2011).

Much of the detail of the mechanisms underlying time-based expectancy is still to be determined. For example, the question of whether the expectancy is based on absolute or relative representations of time is still under debate. In a recent study, we found evidence that participants applied relative, instead of absolute, time representations in time-based expectancy (Thomaschke et al. 2015). After learning time-based event expectations in an initial experimental phase, we applied a new duration range with foreperiods that were either considerably shortened or lengthened. Participants responded, after the longer (or shorter, respectively) foreperiod, faster to the event that had previously been frequent at the formerly relatively longer (or shorter, respectively) foreperiod, suggesting that the participants formed their time-based event expectancy according to relative duration (Thomaschke et al. 2015). This finding suggests that time-based expectancy relies on fundamentally different mechanisms than general timing ability. General timing ability (see above) has repeatedly been shown to rely on absolute time representations (Creelman 1962; Treisman 1963; Gibbon 1977). Therefore, it is important to determine whether alcohol impacts on time-based expectancy as well.

Aim of the present study

We aim at determining whether alcohol impacts on time-based expectancy. Previous literature might suggest that time-based expectancy is not as susceptible the effect of alcohol as general timing ability is, because time-based expectancy probably relies on relative—not on absolute—representations of time. These studies indicate that the glutamatergic *N*-methyl-D-aspartate (NMDA) receptor antagonist ketamine impaired accuracy on a perceptual timing task, while it had no significant effect on RT when temporal pre-cues could be used to predict in advance when the target would appear during performance of a cued RT task (Coull et al. 2011). Alcohol has been shown to decrease the NMDA-related glutamatergic excitability (Hoffman et al. 1989). This means if time-based expectancy would employ absolute representations, the formation of time-based event expectancies would be impaired by alcohol intake and the degree of impairment would depend on dosage. To this end, we tested how different dosages of alcohol impact on the formation of time-based expectancy. Furthermore, in the same experiment, we control whether time-based expectancy really relies on relative time representations.

Methods

Participants

The participants were 18 right-handed healthy volunteers, aged 19–36 years (mean age 23.67 years, SD 3.95 years, six female). The participants were social drinkers, and they consumed at least 1 unit of alcohol weekly. The participants did not show any signs of potential problems with drinking, as tested by the Alcohol Use Disorders Identification Test (AUDIT; Babor et al. 2001). Participants were asked to

abstain from alcohol for 24 h and from any other psychotropic drugs for 48 h, prior to the testing session. None of the participants reported neurological disorders, pregnancy, allergy to alcohol or prescription to any medication for any mental or physical illness. The research was approved by the Georgian National Bioethics Committee.

Overview

The experimental design was within subjects. Participation took place across three days. On each day, a different dosage of alcohol (high/low/placebo) was administered before the experimental procedure. The procedure was identical on each day. It consisted of a learning phase and a test phase. In the learning phase, participants formed time-based expectancies (i.e., they learned the associations between foreperiods and events). In the test phase, they transferred these expectancies to new foreperiod regimes. We tested whether alcohol affected the formation of the transfer of time-based expectancy.

Alcohol administration

Different dosages (high/low/placebo) of alcohol were administered single-blind, in three sessions separated by at least 24 h. Participants were informed that their drink might or might not contain alcohol and they were oblivious about the exact contents of the administered beverage. The session order was counterbalanced across participants. For female participants, alcohol was administered at 0.2 g/kg (low dose) and 0.6 g/kg (high dose). To adjust for gender, men received 10 % more alcohol than women (see Guillot et al. 2010). The administered beverage consisted of grapefruit juice mixed with vodka. Participants in the placebo condition were given the same amount of beverage as the alcohol group, consisting of only the grapefruit juice with 0.25 ml of vodka that floated on the drink surface and around the rim of the glass, to mask olfactory cues. The mean total volume of the drink was 230 ml (Kunchulia et al. 2012). The beverage was administrated before each experimental session. One male participant completed only one experimental session and was, consequently, excluded from the analysis.

The breath alcohol concentration (BAC) was determined from breath samples measured by an alcohol tester (COSMOS CA-2001) and was measured before beverage consumption, at the beginning of each session and again before the fourth and fifth blocks. In order to minimize dose by peak confounds, the experimental session started 15 min after beginning of drinking in the low alcohol condition and 30 min after beginning of drinking in the high alcohol condition. In the placebo condition, the session started 22.5 min after beginning of drinking (Guillot et al. 2010).

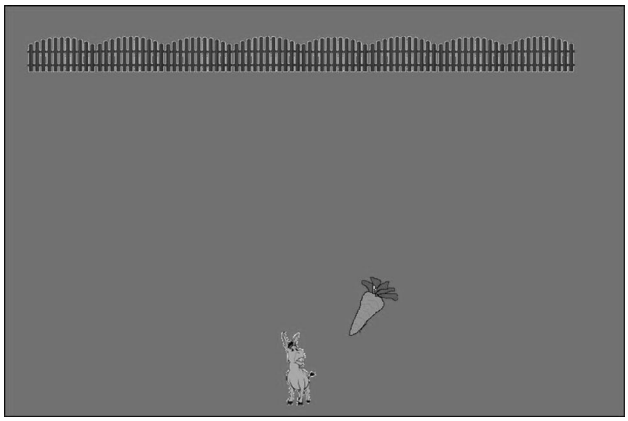

Fig. 1 Schematic illustration of the layout of the choice response tasks. The donkey starts chasing the carrot at the *bottom* of the screen and "captures" it on the fence

Apparatus

We used E-Prime2 for running the experiment and for collecting data (Schneider et al. 2002). Data were collected on a Windows PC with LCD display (screen resolution 1280 × 800 pixels). Responses were collected using a standard optical mouse.

Procedure

Throughout the entire experimental procedure, the participants performed a binary choice response task, mimicking a basic computer game.

The task was to chase a carrot with a donkey character, which moved repeatedly from the bottom to the top of the screen in a zigzag left-to-right course until it could finally be caught at a fence in the upper border of the screen (see Fig. 1). One experimental block consisted of 25 carrot chases, each chase being composed of six jumping steps. When the carrot jumped to the upper left of the donkey, participants had to press the left mouse button in order to make the donkey follow the carrot leftward (pressing the right mouse button moved the donkey to the right). After the mouse click, the donkey immediately jumped on the carrot. After a short or long response–stimulus interval (i.e., from mouse click to carrot movement), the carrot jumped away again. This response–stimulus interval represented the foreperiod in this task. The carrot's movement was either diagonally upward left or diagonally upward right. Depending on the experimental phase, the duration of this foreperiod predicted the carrot's next movement direction (see below). When the participants pressed the wrong key or pressed the key before the carrot had jumped, an error message was displayed, an aversive tone was played over the headphones, and the game was paused for 3 s (see,

Kunchulia and Thomaschke 2014, Szameitat et al. 2009; Thomaschke et al. 2015).

Throughout the experiment, three different foreperiods were used: a short (200 ms), a medium (800 ms) and a long one (1400 ms). In the learning phase, lasting over four blocks, only the long and the medium foreperiods were used. They randomly varied from trial to trial. In this phase, the foreperiod duration predicted the carrot's next movement direction and the next required response, with $p = 0.8$. For half of the participants, medium foreperiods predicted left and long foreperiods predicted right, and for the other half, this relation was inverted. We expected that participants form time-based expectancies in this phase. This means we expected them to respond faster to frequent combinations of foreperiod and direction than to infrequent combinations.

In a subsequent test phase (fifth block), the medium foreperiod appeared again, but the long one was replaced by the short foreperiod. Furthermore, the carrot's next movement direction was not predictable, $p = 0.5$, based on the foreperiod duration. Thus, it was not possible for participants to form new time-based expectancies in the test phase. But we expected that expectancies formerly acquired in the learning phase will transfer to the test phase. If time-based expectancy was formed according to *absolute* time, participants should respond in the test phase at the medium foreperiod faster with the response that was associated with the medium foreperiod in the learning phase. If, on the contrary, time-based expectancy was formed according to *relative* time, participants should respond in the test phase at the medium foreperiod faster with the response that was associated with the *long* foreperiod in the learning phase, because in the test phase the medium foreperiod becomes the *relatively longer* one.

Data analyses

Data from the test phase and the last block of the learning phase were each analyzed with a repeated-measures analysis of variance (ANOVA). The factors were condition (low dose, high dose and placebo), relative foreperiod duration (short vs. long) and frequency (frequent vs. infrequent foreperiod–event combination).

Error trials (1.52 %) and trials with response times (RT) deviating from the condition mean by more than three standard deviations (1.81 %) were excluded from the RT analysis (Bush et al. 1993). In addition, the trials following errors and the initial trial in each block were excluded from the RT analysis and the error analysis. This screening procedure is the same as in our previous studies using this paradigm (see, e.g., Thomaschke et al. 2015).

Results

Alcohol concentration

For all participants, the breath alcohol level was zero before alcohol consumption and remained zero after the placebo drink. The low dose of alcohol produced a mean BAC of $M = 0.184$ µg/ml (SD 0.04 µg/ml) before the first block, $M = 0.18$ µg/ml (SD 0.05 µg/ml) before fourth and $M = 0.173$ µg/ml (SD 0.043 µg/ml) before the fifth block; after the high dose, they were $M = 0.58$ µg/ml (SD 0.134 µg/ml), $M = 0.621$ µg/ml(SD 0.148 µg/ml) and $M = 0.62$ µg/ml (SD 0.146 µg/ml), respectively.

Behavioral data

Formation of time-based expectancy (learning phase)

In the learning phase, a repeated-measures analysis of variance (ANOVA) on the within-subjects factors condition (placebo, low dose, high dose), relative foreperiod duration (short vs. long) and frequency (frequent vs. infrequent foreperiod–event combination) showed significant main effects on foreperiod and frequency: The participants responded slower to short, than to long, foreperiods, $F(1,16) = 18.5$, $p = 0.001$, and faster to frequent combinations than to infrequent combinations, $F(1,16) = 25$, $p < 0.001$ (see Fig. 2, left bars in each panel). The latter main effect means that overall time-based expectancy was formed in the learning phase. There was a marginal tendency toward two interactions between condition and foreperiod, $F(2,15) = 2.88$, $p = 0.087$, and between condition and expectancy, $F(2,15) = 2.977$, $p = 0.081$.

However, no other interaction was significant.

In order to specify these tendencies, we conducted separate analyses of variance (ANOVA) for all treatment conditions. There were main effects of foreperiod for the low, $F(1,17) = 4.95$, $p = 0.04$, and for the high alcohol condition, $F(1,17) = 25.47$, $p < 0.001$, but not for the placebo condition, $F(1,17) = 2.27$, $p = 0.15$. The main effect of expectancy was not significant in the high alcohol condition, marginally significant in the low alcohol condition, $F(1,17) = 2.926$, $p = 0.1$, and significant only in the placebo condition, $F(1,17) = 45.176$, $p < 0.001$. This means that time-based expectancy was formed significantly only in the placebo condition.

The interaction between foreperiod and expectancy was not significant in all treatment conditions. Although the interactions between foreperiod and expectancy were not significant, we analyzed the expectancy effects separately for the medium and the long foreperiod in each

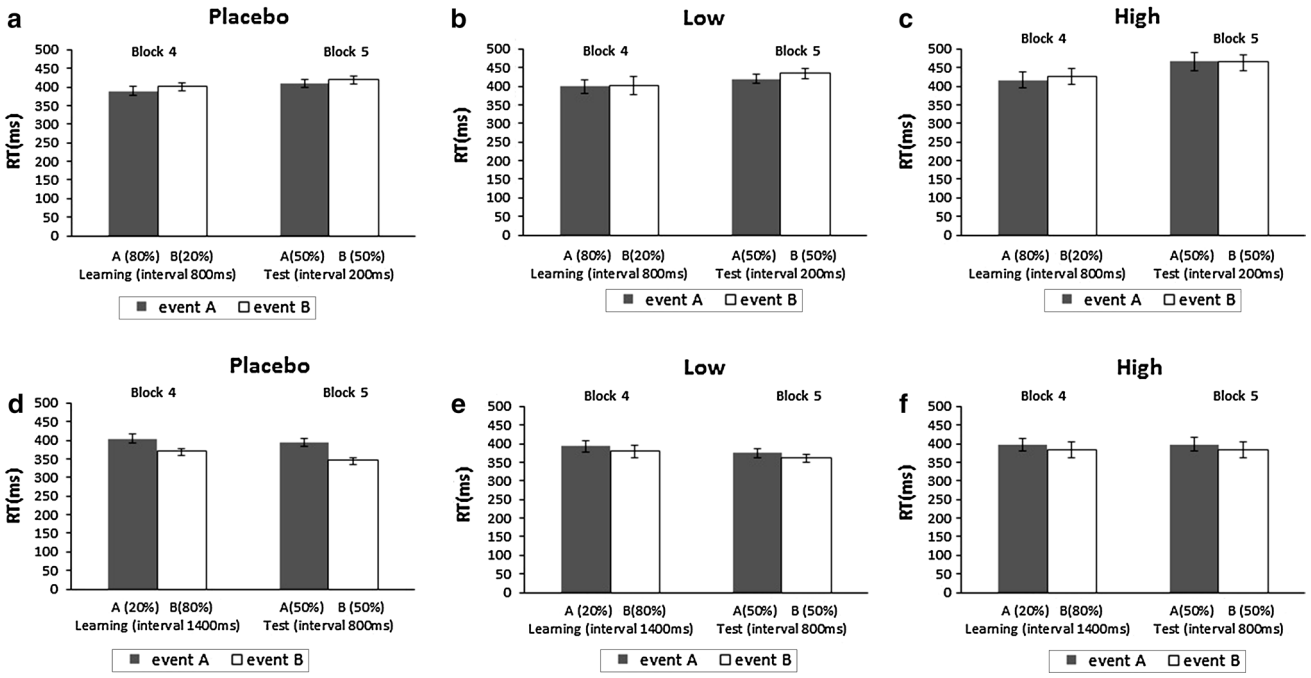

Fig. 2 Mean response times (RTs) for all foreperiods and conditions. *Error bars* represent the standard error of the mean. The *upper panels*, **a**, **b** and **c**, show the mean RTs at the medium foreperiod in the learning phase (*block 4*) and at the short foreperiod in the test phase (*block 5*). Events A and B are the left/right carrot movements. Event A represents the movement that was predicted by the medium foreperiod in the learning phase. We have plotted the medium learning foreperiod and the short test foreperiod together in the *upper panels*, because *relative* time-based expectancy would transfer from the medium learning to the short test foreperiod, as we hypothesized. The *lower panels*, **d**, **e** and **f**, show the mean RTs at the long foreperiod in the learning phase (*block 4*) and at the medium foreperiod in the test phase (*block 5*). Event A represents the movement that was predicted by the long foreperiod in the learning phase. When time-based expectancy is *relative*, it would transfer from the long learning to the medium test foreperiod

treatment condition, because, in some previous studies with the time–event correlation paradigm, the effect was more pronounced at the longer interval (e.g., Thomaschke and Haering 2014; Thomaschke et al. 2011, 2015).

There was a tendency toward an expectancy effect at the longer interval.

In the placebo condition, a numeric advantage for frequent combinations, over infrequent ones, was not significant for the medium foreperiod $t(17) = 0.7$, $p = 0.491$, but was significant for the long foreperiod, $t(17) = 2.84$, $p = 0.011$. In the low alcohol group, there was a marginally significant tendency for the long foreperiod, $t(17) = 1.798$, $p = 0.09$, but not for the medium foreperiod $t(17) = 0.027$, $p = 0.979$. There were no main effects of expectancy after high alcohol administration. These patterns of results, for the placebo and low alcohol condition, also match findings from previous studies with this design (Thomaschke et al. 2015). However, this pattern should be interpreted with caution, because the interaction was not significant.

In an analogous ANOVA for error rates, no main effect or interaction yielded significance.

Transfer of time-based expectancy (test phase)

In the test phase, we conducted a repeated-measures ANOVA with the factors foreperiod (short vs. medium), frequency (frequent vs. infrequent foreperiod–event combination) and condition (placebo, low dose, high dose). Note that frequency of foreperiod–event combination refers to whether the current combination *was* frequent in the learning phase. In the test phase itself, each combination is equally frequent (see "Methods" section; Fig. 2). Note further that the factor frequency is coded according to *relative* time representation (i.e., "frequent" are, e.g., the combination of 800 ms with the event that had been frequent at 1400 ms previously and the combination of 200 ms with the target that had been frequent with 800 previously). Based on previous studies (e.g., Thomaschke et al. 2015), we assume that the learned expectancy transfers from learning to test, according to *relative* duration (see "Introduction").

In the test phase, we observed a main effect for foreperiod, $F(1,16) = 154.49$, $p < 0.001$. The participants responded more quickly after medium, than after shorter, foreperiods. A main effect for expectancy, $F(1,16) = 33.47$, $p < 0.001$, was significant. Responses were faster for

expected (according to *relative* duration) than unexpected combinations. This means participants overall transferred relative time-based expectancy from learning to test. There was also a main effect for condition, $F(2,32) = 4.65$, $p = 0.017$ (see Fig. 2).

However, there were no reliable interactions between condition and foreperiod or expectancy, as well as no interaction between foreperiod and expectancy. However, because there were differences between the formations of time-based expectancy in the learning phase, we conducted separate t tests for expectancy at each interval and each condition. As in the practice phase, we found that there was a tendency toward an expectancy effect at the relatively longer interval (here the medium interval).

The t test showed significant effects of expectancy for all treatment conditions for the medium foreperiod: alcohol high ($t(17) = 2.3$, $p = 0.034$), alcohol low ($t(17) = 2.29$, $p = 0.035$) and placebo ($t(17) = 2{,}423$, $p = 0.027$), but not for the shorter foreperiod. This means that participants transferred time-based expectancy from the longer learning foreperiod to the medium test foreperiod, irrespective of treatment condition.

In an analogous ANOVA for error rates, no main effect or interaction yielded significance, except the interaction between condition and foreperiod, $F(2,32) = 5.468$, $p = 0.009$. Separate ANOVAs for each condition showed a marginal tendency toward a reversed variable foreperiod effect (i.e., fewer errors after short, than after long, foreperiods, see "Introduction") in the placebo condition, $F(1,17) = 3.786$, $p = 0.068$, and a significant reversed foreperiod effect in the high alcohol group, $F(1,17) = 8.227$, $p = 0.011$, but not in the low alcohol group $F(1,17) = 2.581$, $p = 0.127$. There were no significant main effect of expectancy and no significant interaction. This means that alcohol might have interfered with general time expectancy (effect for foreperiod), but this did not interact with time-based expectancy (no interaction with frequency).

Discussion

This study was concerned with determining the possible effects of alcohol on time-based expectancy. In the first part of the experiment (learning phase), we trained human participants to associate two choice responses to different foreperiods. We found that time-based expectancy was formed and that this time-based event expectancy was more pronounced with longer intervals. These results are in accordance with our previous studies on time-based expectancy (e.g., Thomaschke et al. 2015). Note, however, that the modulation of the expectancy effect by foreperiod duration was, in this study, not supported by a significant interaction.

However, after alcohol administration, both effects were no longer statistically significant, and the reduction was greater for a high dose, than for a low dose, of alcohol, suggesting that alcohol impaired the formation of time-based expectancy. Dose-dependent effects of alcohol on various cognitive functions are well known from other studies (Dry et al. 2012). Interestingly, after alcohol administration, the participants showed a stronger variable foreperiod effect (i.e., responding faster to longer intervals, see "Introduction"), suggesting that participants were still able to estimate the interval durations after alcohol intake. Time-based expectancy (i.e., expecting an event based on its time of occurrence), on the other hand, deteriorated after alcohol consumption. In sum, this means that general timing ability was not affected by alcohol, but time-based expectancy was.

In the second part of the experiment (test phase), we substituted the long by the short foreperiod and eliminated the response–foreperiod correlation. In short, the formerly long interval was replaced by a new one, which was shorter than the formerly short interval and both events appeared equally often after both intervals, so that no new time-based event expectancy was induced. Evaluation of RTs after the formerly short and now long foreperiods allowed us to dissociate between absolute and relative models of time-based event expectancy. The idea was that when RTs were shorter for the event that was frequently paired with that foreperiod in the learning phase, the absolute model would be confirmed. The relative model would, on the contrary, predict that RTs would be shorter to the event which was frequently paired with the (now suspended) long foreperiod in the learning phase, because the medium foreperiod becomes, in the test phase, the *relatively* longer foreperiod.

We found that the participants in all treatment conditions transferred their expectancy to a new pair of foreperiods according to relative, not to absolute, foreperiod duration, and that the effect was more pronounced in long, than in short, intervals. These results confirm our previous study, with the same paradigm without alcohol administration. However, there was no significant modulation of this effect by alcohol administration. This means that the formation of time-based expectancy relies on relative timing, irrespective of any influence by alcohol.

In summary, we found that alcohol intake did not change the representational mode in the formation of time-based expectancy (test phase). With and without alcohol, time-based expectancy relies on relative, not on absolute, timing. But alcohol substantially impairs the formation of time-based expectancy (learning phase).

This extends our knowledge of the impact of alcohol on timing. Previous studies in the area have investigated alcohol effects on timing functions which are known to rely on absolute, not relative, timing (Creelman 1962; Treisman

1963; Gibbon 1977, see "Introduction"). Here, we demonstrate that relative timing, as employed in time-based expectancy, can also be impaired by alcohol intake.

However, our study is not directly informative about how alcohol impairs processing of relative time in time-based event expectancy. One possible mechanisms would be related to the effect of alcohol in impairing attention (do Canto-Pereira et al. 2007; Post et al. 1996; Roehrs et al. 1994; Rohrbaugh et al. 1987; Schulte et al. 2001). Alcohol might distract attention away from time (Ogden and Montgomery 2012), which reduces the formation of time-based expectancy. However, further studies are necessary to test the effect of alcohol on time-based event expectancy in further detail.

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