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Acute ethanol administration and reinforcer magnitude reduction both reduce responding and increase response latency in a Go/No-Go task

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Abstract

Background—Ethanol administration decreases behavioral inhibition in human subjects, assessed using cued Go/No-Go tasks, in which an unreliable cue suggests whether participants will be required to respond or not when a signal occurs. Few studies have examined ethanol's effects on behavioral inhibition in animals, and those that have done so, have used Go/No-Go tasks in which no warning cue was provided.

Methods—Two cohorts of male Long-Evans rats were trained and tested on two different Go/No-Go procedures with differing ratios of Go to No-Go trials (25–75 and 50–50). Using a within subjects design, each rat was administered 0.0, 0.63, 0.95, and 1.27 g/kg of ethanol (i.p.) on three separate occasions according to an incomplete Latin Square. An additional experiment examined the effects of reducing the amount of sucrose given for correct responses to either the Go or the No-Go signal in the absence of ethanol administration.

Results—Acute intraperitoneal ethanol administration dose-dependently decreased responding during the No-Go signal (False Alarms), the Go signal (Hits), and responding prior to the occurrence of either signal (Precue Response Rate). These effects were more pronounced in rats with the 50–50 ratio. Reducing the amount of sucrose presented generally led to a decrease in responding, although this effect was also moderated by the Go to No-Go ratio employed and the contingency relationship (reduced sucrose for correct Go trial responding or for correct No-Go trial response withholding).

Conclusions—Acute ethanol administration does not decrease behavioral inhibition in rats in this task. Rather ethanol appears to dose-dependently decrease behavior in general, possibly by reducing the efficacy of the sucrose reinforcer, as both ethanol administration and sucrose reduction for Go trials yielded similar patterns of behavioral responding in this task in rats.

Keywords

Ethanol; Behavioral Inhibition; Impulsivity; Go/No-Go; Motivation

Introduction

Studies in humans have shown that ethanol administration decreases behavioral inhibition using a variety of tasks, for example, in the Stop task (de Wit et al., 2000; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997), which measures an individual's ability to inhibit an already-initiated response. Ethanol administration also decreases behavioral inhibition in

the Go/No-Go task, which measures an individual's ability to respond during one particular signal and refrain from responding during a different signal (Assaad et al., 2006; Easdon et al., 2005; Finn et al., 1999; Loeber & Duka, 2009; Marczinski & Fillmore, 2003 but also see Ortner et al., 2003; Reynolds et al., 2006; Rose & Duka, 2008). Presumably, the decreased inhibition following ethanol contributes to increased risk-taking in individuals under the influence of alcohol, such as increased violence and unprotected sex (Bushman & Cooper, 1990; Erickson & Trocki, 1992; Fendrich et al., 1995).

Despite the numerous studies conducted in the human literature, noticeably fewer studies have examined the effects of ethanol administration on behavioral inhibition in nonhuman animals. Several studies suggest that ethanol administration decreases behavioral inhibition, similar to results seen in most studies of humans. For example Feola et al., (2000) found that ethanol increased the lead-time required for rats to successfully withhold an ongoing response in the rat-version of the Stop task. Another study found that ethanol administration decreased efficiency (number of correct responses/total responses) in the Signaled Nose Poke task in mice (Olmstead et al., 2009), which measures the ability of the individual to refrain from responding before a cue is presented. Similarly, using the 5-Choice Serial Reaction Time task, Oliver et al., (2009) found that ethanol administration increased the number of premature responses prior to a cue in mice, though Bizarro et al., (2003) found no effect of ethanol administration in rats using the same task. The only study of acutely administered ethanol on the Go/No-Go task in rats also found no effect (Hellemans et al., 2005). However, the Go/No-Go task used in the Hellemans et al. study was markedly different from those used in human studies and in the current study, in that it segregated Go and No-Go trials into different blocks and reinforced only correct Go trials (i.e., the No-Go trials were effectively extinction trials).

To examine the effects of ethanol on a task more similar to those used in humans, ethanol was administered to rats trained on a Go/No-Go task with interspersed Go and No-Go trials in which both trial types had reinforcement contingencies. Based on the human Go/No-Go literature, we hypothesized that ethanol would dose-dependently decrease behavioral inhibition, operationally defined as an increase in False Alarms. We were also interested in the effect of motivation in this task. Ethanol has been shown to effect motivation in different ways depending on the paradigm used. For example, ethanol enhances sexual motivation in rats (Ferraro & Keifer, 2004), while it also depresses rats' motivation for food-reinforced operant responding (Petry, 1998). Data from these two studies suggest that, at moderate doses, this motivational effect may be independent of any general locomotor effects of ethanol. To understand the effects of motivation on behavior in the Go/No-Go task, we performed a second experiment in which we reduced reinforcer magnitude for either Go or No-Go trials to determine if the effects resembled those following ethanol administration.

Materials and Methods

Subjects

Male Long-Evans rats ($n = 32$) were obtained from Charles Rivers Laboratories (Hollister, CA) for these experiments. Rats were housed two per cage under a 12:12 h light:dark cycle in a temperature controlled vivarium in the Department of Comparative Medicine at Oregon Health & Science University, an AAALAC-approved facility. All procedures were approved by the appropriate Institutional Animal Care and Use Committee and adhered to NIH Guidelines. Rat free-feeding weights were obtained over one week prior to the beginning of the study. To facilitate training and maintain responding in behavioral tasks, 3 days prior to the start of training, animals were food restricted to approximately 90% of their free-feeding body weights. Animals were maintained at this weight with standard laboratory chow following each test session. Four rats were removed from the experiment because their Go/

No-Go task performance was not stable over 5 consecutive days after 90 sessions of testing (determined by visual inspection of Hits, False Alarms, and Precue Rate), making it unreasonable to examine drug effects.

Apparatus

For Go/No-Go testing, we used eight identical (Med Associates Inc., St. Albans, VT, USA) modular rat test chambers (25 x 32 x 26 cm) housed individually within melamine sound-attenuating cabinets (40 x 64 x 42 cm). A fan provided constant ventilation and low-level background noise. The front, top, and back panels of the chamber were made of clear acrylic, and the front panel could be opened to allow access to the chamber. Both the left and right panels were made of stainless steel. A houselight (5 lux) and response clicker were mounted in the center-top of the left panel. Immediately below the houselight a tone generator was mounted (4500 Hz, 68 dB). Three nonretractable levers were mounted on the opposing panel directly below lights (4 lux) and above recessed nose pokes. It should be noted that the levers and center nose poke recess and light were used for other studies, but had no function in this study. The floor of the chamber consisted of 19 metallic rods spaced 1 cm apart. Below the floor was a stainless steel removable tray filled with bedding. Computer-controlled pumps were used to deliver 75 μ l of sucrose (10% w/v) to liquid cups located in the recesses of the outer nose poke recesses. A computer program written in MED-PC (Med Associates Inc., St. Albans, VT, USA) controlled the output devices and recorded responses on the input devices.

Drugs

Ethanol (200 proof; 100%) was diluted with 0.9% saline to obtain a solution containing 20% w/v ethanol. On test days, ethanol was administered intraperitoneally (i.p.) immediately before animals began the test session. Each animal was administered the doses 0.0 g/kg, 0.63 g/kg, 0.95 g/kg, and 1.27 g/kg three times each (incomplete Latin square).

Test sessions for all experiments involving ethanol took place on Tuesdays and Fridays, with no-injection sessions on Mondays, Wednesdays and Thursdays. No-injection sessions on the day immediately prior to a test session were used as the measure of baseline behavior for that test session. Animals were not tested on weekends, but were maintained on their food-restricted diet.

Go/No-Go Task

The Go/No-Go task was modeled after those used previously (Helms et al., 2008; McDonald et al., 1998; Wilhelm et al., 2007). For the initial training phase, rats had to nose poke in the appropriate hole (counterbalanced left or right between subjects) during a 30-s Go cue to receive sucrose. After rats had successfully completed 50 or more trials within 1 hour for 2 consecutive sessions, the cue duration was reduced from 30 s to 10 s. When rats successfully completed 50 or more trials within 1 hour for 2 consecutive sessions at 10 s, the No-Go cue was introduced on future sessions (Go/No-Go task implemented).

Each session of the Go/No-Go task ended after 100 trials were completed or 1 hour had passed. For each trial of the Go/No-Go task (Fig. 1), there was a precue period of varying duration (9–24s), signaled by the houselight turning on. Responses during this period were recorded but not reinforced. Precue responses can be used as a measure of inhibition in gauging how long an animal is willing to withhold responding before the cue appears (this is frequently used as a measure of inhibition in other tasks such as the 5 Choice Serial Reaction Time Task). The variable timing is used so that the animal cannot predict when the cue will begin. Preliminary data in our laboratory showed that animals responded very little during a darkened precue period, so we used a houselight to distinguish the precue period

from the darkened inter-trial interval (ITI) and to potentially elicit more responding. Following the precue period a 3-s cue occurred signaling a Go or No-Go trial. Different cues were used to distinguish Go trials from No-Go trials. The two cues were a 4 lux light above the appropriate nose poke recess and a 68-dB 4.5 kHz tone. The light was the Go cue and the tone was the No-Go cue for half of the rats, and vice versa for the other half. When the Go cue was displayed, the first response in the appropriate nose poke recess ended the cue and started the reward consumption period (3 s), which was signaled by a “click” and resulted in 75 μ l of 10% sucrose solution being delivered to the liquid cup in the nose poke recess. When the No-Go cue occurred, if a nose poke was recorded then the cue period was immediately terminated and sucrose was not delivered. However, if no nose poke was recorded during the cue period, the reward was delivered to the same cup as for the Go cue reward and a 3-s reward period began. After either trial type, a 5-s ITI occurred during which the house light was off. Responses during this period were recorded but had no programmed consequences. Go and No-Go trials were randomly ordered.

There were three measures of primary interest: Hits, False Alarms, and Precue Response Rate. Hits are the number of times a rat nose pokes during the cue period of a Go trial in a particular session. False alarms are the number of No-Go trials on which the rat nose pokes during the No-Go cue period (they are reinforced for waiting until *after* the cue period is over). Precue response rate is the total number of responses made during all of the precue periods divided by the total duration of all of the precue periods.

Experiment 1: Effects of ethanol

Studies indicate that ethanol can increase low basal rates of responding while decreasing high basal rates of responding (Leander et al., 1976; Barrett & Stanley, 1980). Thus, it may be that ethanol decreases behavioral inhibition only when there is a relatively low overall response rate. Therefore, in experiment 1, one group of rats (experiment 1a; $n=15$) were exposed to a task with 25 Go trials to 75 No-Go trials and a second group of rats (experiment 1b; $n=13$) that were exposed to a task with 50 Go trials and 50 No-Go trials (higher expected response rate). After rats achieved stable performance (determined by visual inspection of Hits, False Alarms, and Precue Rate over the preceding 5 days) and had acquired sufficient discrimination of the Go and No-go cues, they began the ethanol dosing schedule. Sufficient discrimination was defined as having a d-prime value of at least 1, where d-prime equals the z-score transformation of the proportion of Go trials on responding occurred (Hits) obtained minus the z-score transformation of the proportion of No-Go trials on which responding occurred (False Alarms) for each session (average d-prime for all rats at start of injection phase: 1.65 ± 0.08). Because d-prime is also sensitive to response rate, it also ensured that our animals had moderate levels of responding.

Experiment 2: Reinforcement manipulations

Ethanol's effects on responding may be due to reducing the reinforcing efficacy of the reward. Therefore, we performed a series of manipulations to determine the behavioral effects of reducing the amount of sucrose delivered after the successful completion of a Go or No-Go trial, which should reduce the reinforcing efficacy of the reward. At least 10 sessions after rats had completed ethanol dosing in the Go/No-Go task in experiments 1a and 1b, we manipulated the amount of sucrose delivered in the Go/No-Go task to examine reward motivation. The task protocol was basically unchanged; however, for 7 rats from experiment 1a correct Go trials were reinforced with only 1/8th the original amount of sucrose (10 μ l), whereas correct No-Go trials were still reinforced with the original amount. For the remaining 8 rats from experiment 1a correct No-Go trials were reinforced with only 1/8th the original amount of sucrose, whereas correct Go trials were still reinforced with the original amount (experiment 2a). Rats from experiment 1b ($n = 12$) underwent both

manipulations (experiment 2b), separated by a 5 day recovery period and counterbalanced between subjects as to the order of administration. We predicted that animals with sucrose-reduced Go trials would decrease their number of Hits, and that animals with sucrose-reduced No-Go trials would increase their number of False Alarms.

Data Analysis

Data from each experiment were analyzed using ANOVAs (SPSS Inc, Version 16). Ethanol Go/No-Go data were analyzed with a 4 X 2 X 3 X 2 ANOVA, with ethanol Dose (0, 0.63, 0.95, 1.27 g/kg), Injection (injection day, preceding day [baseline]), Occasion (1st, 2nd, 3rd ethanol exposure at each dose) and Stimulus (Go=light and No-Go=tone, Go=tone and No-Go=light) as the factors, respectively. Stimulus was a between-subjects factor, other factors were within-subject. There were some interactions involving the effects of stimulus arrangement and occasion, but they were not replicated between the two experiments, and will not be discussed.

Reinforcement magnitude manipulation data were analyzed with t-tests, comparing the average of the 10 days before the sucrose manipulation and the average of the 10 days during the sucrose manipulation.

Violations of sphericity were adjusted with Huynh-Feldt corrections, and corrected degrees of freedom are shown where appropriate. Bonferroni-corrected post hoc t-tests and planned comparisons were used to compare individual means. Significance was assessed as $p < .05$.

For a few animals, ethanol administration or reduction of sucrose lowered responding to the point where an accurate latency to respond to the Go or No-Go cue could not be determined. In these cases, those animals were removed from the analysis of Go or No-Go cue response latency (exp 1a: $n = 4$; exp 1b: $n = 2$; exp 2b: $n = 2$).

Results

Training

We first examined the number of sessions to complete training and for performance to stabilize prior to ethanol administration for rats with the Light as the Go cue and the rats with the Tone as the Go cue to see if stimulus arrangement had an effect on learning the task. In both experiments there were no differences in number of sessions between rats with either arrangement of stimuli pairings to complete training (exp 1a: Light (mean $\pm$ SEM): 7.63 ± 1.81 , Tone: 6.75 ± 1.21 ; $t(13) = 0.17$, $p = 0.866$ and exp 1b: Light: 10.75 ± 1.83 , Tone: 10.75 ± 1.87 ; $t(14) = 0.00$, $p = 1.000$) or for performance to stabilize (exp 1a: Light: 50.43 ± 5.02 , Tone: 42.75 ± 1.39 ; $t(13) = 1.57$, $p = 0.141$ and exp 1b: Light: 53.33 ± 5.90 , Tone: 41.86 ± 3.93 ; $t(11) = 1.66$, $p = 0.125$).

Ethanol effects

We next examined the effects of ethanol administration (i.p.) on inhibition in the Go/No-Go task. We anticipated that ethanol administration would increase either False Alarms, Precue Response rate, or both. However, ethanol administration decreased these two measures in both experiments while behavior on pre-injection, baseline sessions remained stable (Dose x Injection: Fig. 2): False Alarms (Exp 1a: $F(3, 39) = 3.27$, $p = 0.031$, although the effects of ethanol on False Alarms were not significant at any one dose when compared to saline; Exp 1b: $F(2.27, 22.68) = 11.46$, $p < 0.001$), and Precue Response Rate (Exp 1a: $F(2.70, 27.27) = 12.46$, $p < 0.001$; Exp 1b: ($F(3, 30) = 6.48$, $p = 0.002$)). However, these decreases were accompanied by significant decreases in Hits (Exp 1a: $F(3, 39) = 20.89$, $p < 0.001$; Exp 1b: $F(3, 30) = 19.45$, $p < 0.001$), ITI responses (Exp 1b only: $F(3, 30) = 12.10$, $p < 0.001$) and an

increase in the latency to respond to the Go cue (Exp 1a: $F(2.41, 21.73) = 10.77$, $p < 0.001$; Exp 1b: $F(3, 24) = 8.01$, $p = 0.002$), suggesting the increase in behavioral inhibition measures in fact was due to a decrease in overall activity (see Figs. 2, 3).

We explored the source of the Dose x Injection effects in more depth using Bonferroni-corrected post hoc t-tests. For experiment 1a, the 1.27 g/kg ethanol administration resulted in lower Precue Response Rates and Hits compared to saline and 1.27 g/kg and 0.95 g/kg resulted in a longer latency to respond to the Go cue compared to saline. These effects were more pronounced for rats in experiment 1b. Compared to saline, all doses of administration resulted in lower False Alarms, while both 1.27 g/kg and 0.95 g/kg ethanol resulted in lower Precue Response Rates, Hits, ITI responses and a longer Go cue latency (see Figs. 2, 3). This supports our hypothesis that ethanol would have stronger suppressant effects on animals with a higher response rate, because animals in experiment 1b (50-50 Go: No-Go trials) tended to respond more quickly (0.38 ± 0.08 resp/s vs. 0.25 ± 0.02 resp/s for animals with 25-75 Go: No-Go trials; $t(25) = 1.74$, $p = 0.094$).

Reinforcement Manipulations

To examine whether the reductions in responding on Go and No-Go trials could be explained as a reduction in the efficacy of the reinforcer during the ethanol treatment, and if so whether similar effects were produced when reinforcer efficacy for Go and No-Go trials were manipulated separately, we examined the effects of reducing the amount of the reinforcer on Go trials and on No-Go trials.

Selectively reducing the amount of sucrose delivered for successfully responding to Go trials while maintaining the amount for successful inhibition during No-Go trials significantly decreased Hits for rats in both experimental groups (exp 2a: $t(6) = 4.54$, $p = 0.004$; exp 2b: $t(11) = 10.04$, $p < 0.001$, see Fig. 4) and increased latency to respond to the Go cues for rats in both experiments (exp 2a: $t(6) = 6.72$, $p < .001$; exp 2b: $t(9) = 6.74$, $p < .001$, see Fig. 4). The reduced motivation to respond also generalized to No-Go trials. Latency to respond on No-Go trials increased in both experiments (exp 2a: $t(6) = 4.81$, $p = .003$; exp 2b: $t(11) = 3.87$, $p = .003$, see Fig. 4). Number of False Alarms also decreased but only significantly for rats in experiment 2a ($t(11) = 3.93$, $p = 0.002$, see Fig. 4). Conversely, selectively reducing the amount of sucrose delivered for successful inhibition during No-Go trials while maintaining the amount for responding during the Go signal significantly increased False Alarms ($t(7) = 3.32$, $p = 0.013$), and decreased Hits ($t(7) = 3.03$, $p = 0.019$) (Fig. 5) in experiment 2a, while it decreased latency to respond to the No-Go cue in experiment 2b ($t(11) = 2.38$, $p = 0.036$; see Fig. 5). Therefore, it appears that reducing motivation selectively associated with earning the reward from correct responding on Go or No-Go trials affects behavior on both types of trial, although the No-Go manipulation seemed to have limited effects on rats in experiment 2b.

Discussion

Ethanol administration did not increase False Alarms or Precue Response Rate, indicating that ethanol administration did not decrease rats' behavioral inhibition. Rather, ethanol administration dose-dependently *decreased* False Alarms and Precue Response Rate. While this initially might suggest that ethanol administration increased behavioral inhibition, the fact that ethanol administration also decreased Hits and ITI responses suggests that this effect is probably due to a general reduction in task-related behavior. The actual nature and cause of this reduction is intriguing. However, before exploring that issue we discuss possible reasons for the differences between our results and the human studies that report ethanol-induced decreases in behavioral inhibition. Although we designed our task to more closely approximate tasks used with human subjects, none of the tasks used in the human

studies are identical to our own, and few are identical to each other. In particular, none of the tasks used in human studies delivered a primary reinforcer upon successful completion of each trial. Previous research has suggested that differences between human and non-human data in some impulsive and risky choice tasks may be in part because animals receive primary reinforcers and humans receive secondary reinforcers. Indeed, if humans are given primary reinforcers, their behavior more closely resembles animal behavior (Jimura et al., 2009; Lagorio & Hackenberg, 2010) while if animals are given secondary reinforcers, their behavior more closely resembles human behavior (pigeons, Lagorio & Hackenberg, 2010). Thus, future research using the Go/No-Go task may need to either use secondary reinforcers for animals or primary reinforcers for humans in order to see comparable effects between the two. It should also be noted that our study is not the first to find that ethanol administration does not decrease behavioral inhibition in the Go/No-Go task (Ortner et al., 2003); Reynolds et al., 2006; Rose & Duka, 2008). Furthermore, the one other study in rats that has examined the effect of ethanol administration on the Go/No-Go task also did not find a decrease in behavioral inhibition (Hellemans et al., 2005), although this version of the task was markedly different from our own and others seen in the human literature.

There are also several differences between our study and others that have examined the effect of ethanol administration on behavioral inhibition in rodents. For example, the 5-choice serial reaction time task employed by Bizarro et al. (2003) and Oliver et al. (2009) used responses during an ITI as the measure of behavioral inhibition. Interestingly, Oliver et al. (2009) used a timeout period as punishment for responding during the ITI and found ethanol administration to decrease behavioral inhibition, while Bizarro et al. (2003) had no such timeout for inappropriate responding and found no effects. Therefore, it is possible that including a punishment contingency in the task would have elicited an ethanol administration effect on behavioral inhibition. The Stop task used by Feola et al. (2000) also differs from our task. Importantly, the Stop cue is initiated very shortly after the Go cue within the same trial, whereas in our task the Go cue and No-Go cue are never initiated within the same trial. Furthermore, decreased behavioral inhibition in the Stop task is seen as a slower reaction time to the Stop cue due to a decrease in successful Stop responses. Such results are comparable to the decreases in responding we see. Finally, these three tasks (5-choice serial reaction time task, Stop, and Go/No-Go tasks) appear to depend on different neurological and pharmacological mechanisms (Eagle et al., 2008; Eagle & Baunez, 2010), and it would not necessarily be surprising for ethanol to affect one task and not the other. Indeed, such task-dependent effects have been seen with other drugs: in rodents, d-amphetamine has been shown to decrease behavioral inhibition in the 5-choice serial reaction time task (increase in premature responses; Cole & Robbins, 1987; Loos et al., 2010), increase behavioral inhibition in animals with low basal behavioral inhibition in the Stop task (decrease in stop signal reaction time; Feola et al., 2000) and decrease general responding in the Go/No-Go task without affecting behavioral inhibition at all (no effects specific to False Alarms or Precue Responding; Loos et al., 2010).

Although ethanol did not affect behavioral inhibition, it did reduce overall levels of responding. This finding is consistent with several previous studies (Barrett & Stanley, 1980; Laties & Weiss, 1962; Leander et al., 1976; Sidman, 1955). One possible explanation for this is that ethanol simply reduced locomotion. This idea was not examined in the current study. However, although ethanol does decrease general locomotor activity in Long-Evans rats at 1.6 g/kg, at doses of 1.0 g/kg and less it does not (Duncan et al., 2000; Scott et al., 1994). Therefore, a simple reduction in basal locomotion does not fully explain the reduction, especially considering the effects seen at the lower doses in experiment 1b animals. However, it is also possible that ethanol is reducing motivation to respond in the task (such an effect might also explain ethanol's ability to decrease general locomotion).

Indeed, a study by Petry (1998) suggests that ethanol suppresses response rate by acting on reinforcement efficacy, and not motor ability, at a dose of 0.9 g/kg i.p. in Wistar rats.

To further test this hypothesis we examined the effect of sucrose reductions for either the Go or the No-Go component of the task to determine if changes in reinforcement motivation would cause reductions in behavior observed with ethanol administration. In both experimental groups, animals with sucrose-reduced Go trials made fewer Hits and increased their latency to the Go and No-Go cues, suggesting that reducing sucrose for Go trials can mimic some of the suppressant effects of ethanol (Fig. 4). However, only animals in experiment 2b decreased False Alarms when we reduced sucrose after Go trials. This may be because animals in experiment 2a had a relatively low level of False Alarms at baseline and that a floor effect was occurring. Interestingly, a different effect was seen when reducing the sucrose after successful inhibition of No-Go trials. Although it had little effect on animals in experiment 2b, animals in experiment 2a increased False Alarms and decreased Hits (Fig. 5). The increase in False Alarms is not unexpected because animals had less motivation to inhibit their response. Lack of a similar finding in experiment 2b may be because those animals already had low inhibition at baseline. At any rate, the effects of reducing the magnitude of reward following the Go cue, but not the No-Go cue, appear to be similar to the effects of ethanol in this task. This may be because ethanol reduces the reinforcing efficacy of sucrose in general, and that the behavioral effects of ethanol on the reinforcing efficacy of the Go cue mask the behavioral effects of ethanol on the reinforcing efficacy of the No-Go cue (since the behavioral effects of reducing the reinforcing efficacy of Go trials are more pronounced, see Figs. 4, 5).

Interestingly, ethanol had more pronounced effects on responding in animals in experiment 1b than those in experiment 1a. Furthermore, these effects occurred at lower doses in experiment 1b than in experiment 1a (see Figs. 2, 3). It is difficult to determine the exact cause of this effect. For example, what we are seeing may be due to different basal rates of activity, motivation, or stimulus control of the Go and No-Go cues. Future studies will be needed to determine which, if any, of these explanations is responsible for the effects seen in these two groups of animals.

In conclusion, we have several findings to report. First, ethanol administration (i.p.) did not alter behavioral inhibition in this task. However, it did lead to a general decrease in responding. The ethanol-induced reduction in responding seen may be due to either a motor deficit or a decrease in motivation. Although the former may be the more intuitive possibility, we show evidence suggesting that latter is perhaps more likely. Future work should seek to disentangle these two potential different causes of ethanol administration's suppressant effects.

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Both authors designed the studies together. TMM conducted experimental sessions, processed data, conducted data analyses and was primarily responsible for manuscript writing. SHM advised on data analyses and assisted in manuscript writing.

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Figure 1.

The nose-poke contingencies of a single trial in the Go/No-Go procedure (Figure adapted from Gubner et al., 2010). Note that this pair of sample trials uses the Light as the Go cue and the Tone as the No-Go cue. Half of the rats received the opposite stimulus pairings.

Figure 2.

Effects of ethanol on False Alarms, Precue Response Rate, and Hits in the Go/No-Go task for experiment 1a (a, b, c) and experiment 1b (d, e, f). The Ethanol dose-response curve shows the value of each measure after administration of each dose of ethanol, while the Baseline dose-response curve shows the value of each measure for the day prior to injection. Asterisks indicate significant differences from saline (0.0 g/kg ethanol), * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

Figure 3.

Effects of ethanol on Inter-Trial Interval (ITI) responses, No-Go Latency, and Go Latency in the Go/No-Go task for experiment 1a (a, b, c) and experiment 1b (d, e, f). The Ethanol dose-response curve shows the value of each measure after administration of each dose of ethanol, while the Baseline dose-response curve shows the value of each measure for the day prior to injection. Asterisks indicate significant differences from saline (0.0 g/kg ethanol), * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

Figure 4.

The effects of reducing sucrose for Go trials on Hits, Go Latency, False Alarms, and No-Go Latency for experiment 2a (a, b, c, d) and experiment 2b (e, f, g, h). Baseline levels of responding are on the left in each set of bars, and levels of responding after sucrose reduction are on the right. Asterisks indicate differences from baseline, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

Figure 5.

The effects of reducing sucrose for No-Go trials on False Alarms, No-go Latency, Hits, and Go Latency for experiment 2a (a, b, c, d) and experiment 2b (e, f, g, h). Baseline levels of responding are on the left in each set of bars, and levels of responding after sucrose reduction are on the right. Asterisks indicate differences from baseline, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.