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ORIGINAL INVESTIGATION

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Working memory, executive processes and the effects of alcohol on Go/No-Go learning: testing a model of behavioral regulation and impulsivity

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Abstract *Rationale:* Impulsivity is associated with increased risk for alcoholism. Alcohol also may increase impulsive behavior, although little is known about the processes underlying this effect. *Objectives:* This study tested a model proposing that the executive processes of working memory (WM) and conditional associative learning (CAL) modulate behavioral inhibition. Subjects had either a positive (FHP) or a negative (FHN) family history of alcoholism. Hypotheses were that alcohol would increase Go/No-Go impulsive responding but only in subjects with low working memory capacity (low-WM), low-CAL ability, or FHP for alcoholism. The model also predicted that WM and CAL modulate inhibitory responses to contingency reversal on a Go/No-Go task. *Methods:* A Go/No-Go learning task with a midway contingency reversal was administered to 71 FHP and 78 FHN subjects when sober and after drinking one of two moderate doses of alcohol. WM (digits backward) and CAL (conditional spatial association task) were also assessed when sober. *Results:* Alcohol resulted in more false alarms but only in low-WM subjects. Both WM and CAL modulated learning to inhibit behavior after contingency reversal, suggesting separate modulation mechanisms for WM and CAL. Subjects with low-capacity WM and subjects with low-capacity CAL ability had more difficulty learning response inhibition after contingency reversal. FHPs and FHNs did not differ in their response to alcohol. *Conclusions:* The results support our model of the modulatory role of WM and CAL in the ongoing regulation of behavioral inhibitory systems. The results also suggest that individuals with low capacity WM are more susceptible to alcohol's effect of increasing impulsive behavior, suggesting that alcohol reduces the capacity of working memory to modulate response inhibition.

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Introduction

It is well documented that impulsive, under-controlled behavior predicts alcohol and drug abuse (e.g., Cloninger et al. 1988; Luengo et al. 1994) and reflects a major portion of the vulnerability to substance abuse in the offspring of alcoholics (Sher et al. 1991). Considerable evidence suggests that alcohol promotes impulsive behavior as well (Steele and Southwick 1985). Less is known about the processes by which impulsivity promotes, or is promoted by, substance use/abuse. Factors serving to impede our understanding of the impulsivity-drug abuse relationship are the confusion about how to conceptualize and measure impulsivity, and the reliance on single mechanism models of impulsivity (White et al. 1992; Luengo et al. 1994).

The literature reveals considerable diversity in theory, definition, and approach to the measurement of impulsivity (cf. Luengo et al. 1994), with little agreement about how to conceptualize the construct (Parker and Bagby 1997). There are numerous complaints that many measures of impulsivity do not correlate very highly with each other (Barratt and Patton 1983; White et al. 1992; Parker and Bagby 1997). Different schemes to characterize the multidimensional nature of impulsivity have been proposed (Gerbing et al. 1987; White et al. 1992), but have had little impact on research and theory on mechanisms underlying impulsivity. Nonetheless, recent studies have identified a range of important potential mechanisms underlying impulsive behavior. For instance, deficient inhibition of prepotent behavior, assessed using "stop-signal" tasks, has been associated with attention-deficit hyperactivity disorder (ADHD) and trait impulsivity (Schachar et al. 1993; Logan et al. 1997) and is evident after moderate doses of alcohol (Mulvihill et al. 1997). Impulsivity in drug abusers has been linked to in-

creased discounting of delayed large rewards in favor of immediate but smaller rewards (Kirby et al. 1999). Deficits in executive cognitive processes have been associated with impulsive, aggressive responses (Lau et al. 1995). Considerable research also suggests that increased reward responsiveness or reduced responsiveness to punishment are associated with impulsive behavior (Newman 1987; Lykken 1995). Although these studies have contributed to our knowledge of the mechanisms of behavioral regulation and impulsivity, their major limitation is that they present only single-mechanism models of impulsivity. With the exception of Mulvihill et al. (1997), they rely on bivariate correlations between the mechanism (e.g., stop-signal performance) and some manifestation of impulsivity (e.g., a self report scale).

In this paper we test a cognitive-modulation model of impulsivity by examining the influence of executive processes on the effects of alcohol on behavioral inhibition in a Go/No-Go task with contingency reversal. We conceptualize impulsivity more broadly as dysregulated behavior. This term better conveys the complex interplay of factors underlying the breakdown in behavioral regulation that the construct of impulsivity implies, such as poorly planned, unreflective, reckless, abrupt, undercontrolled, or inappropriate behavior that leads to negative outcomes (Eysenck and Eysenck 1978; Barratt and Patton 1983). We use a Go/No-Go learning task with contingency reversal, because this task involves the interplay between behavioral inhibition and activation, learning, and response perseveration, which captures a degree of the complexity of behavioral regulation including the involvement of executive processes. Our model holds that low correlations are likely among some of the processes underlying behavioral regulation for two reasons. First, low correlations are likely because a number of different mechanisms are involved in the regulation of behavior, such as foresight, attention, or differential responses to threat or reward. Second, correlations of zero also are likely because at least some of these processes probably operate independently of each other and may involve separate neural substrates (cf. Petrides et al. 1993). We expect stronger correlations between inter-related processes when the system (person) is challenged significantly.

Our model focuses on the interplay between behavioral inhibition and the inter-related executive processes of working memory and conditional associative learning. Executive cognitive processes are those processes that are thought to monitor, direct, and organize (regulate) behavior to effectively achieve desired goals while minimizing negative outcomes (Barkley 1997; Kimberg et al. 1997). Executive functions are thought to include a range of higher-order processes such as associative learning, memory, problem solving, and planning (Giancola 1995; Zelazo and Frye 1998). Working memory and conditional associative learning are thought to play important roles in the self-regulation of behavior (Petrides et al. 1993; Lau et al. 1995) and are likely to contribute to the reflectivity/planning functions underlying behavioral inhibition (Barratt and Patton 1983). Although our model distinguishes between working memory and other executive processes (such as conditional associative learning), others consider executive processes as a subset of a broader working memory system (Baddeley 1986; Kimberg et al. 1997).

We propose that working memory is involved in the ongoing, moment to moment, regulation of behavior and serves to modulate activity in behavioral inhibition systems, such as the system described by Gray (1987a). Rather than simply serving as the comparator, as in Gray's (1987a) model, we refer to Baddeley's (1986) concept of the "central-executive" aspect of working memory involved in allocating "attention for processing and manipulating information in a short-term memory store" (Stout et al. 1995, p. 1221). In our model a second executive process, conditional associative learning, subserves behavioral regulation via the reflective/planning, decision process which is thought to be deficient when impulsive behavior is manifested (Barratt and Patton 1983). Conditional associative learning involves 1) bringing into working memory a set of previously learned stimulus-response associations, 2) then selecting an appropriate response out of that set of possible responses in order to 3) adjust or program behavior to adapt to unexpected, or challenging circumstances. Our model proposes a threshold function for the modulating effect of working memory and conditional associative learning processes on behavioral inhibition, such that the modulating roles of working memory and conditional associative processes are more easily disrupted by alcohol, or unexpected challenging circumstances in individuals with low operating capacity in these systems. Consistent with this hypothesis are data indicating that subjects with low scores on measures of conditional associative task learning show heightened aggression under high provocation conditions (Giancola and Zeichner 1994; Lau et al. 1995). In addition, some work suggests that increased sensitivity to alcohol in the offspring of alcoholics is associated with executive deficits (Peterson et al. 1992). Although some studies report deficits in executive function in the offspring of alcoholics (Tarter et al. 1989; Peterson et al. 1992), a number of studies do not find this association (Schuckit et al. 1987; Bates and Pandina 1992).

This experiment tested the hypotheses that: 1) alcohol will disinhibit behavior, resulting in more false alarms in a Go/No-Go learning task, especially in persons at risk for alcoholism, 2) alcohol will cause greater disruptions in response inhibition (i.e., more false alarms) in persons with either low capacity central executive working memory or low conditional associative learning ability, and 3) persons low in working memory or conditional associative learning capacity will develop inhibitory responses to a contingency reversal less effectively, resulting in more false alarms, than persons high in executive cognitive function. Given earlier studies suggesting that subjects with a positive family history of alcoholism (FHP) have deficits in self-regulation,

we hypothesized that alcohol will disinhibit behavior more in FHP compared with family history negative (FHN) subjects.

Materials and methods

Subjects

FHP subjects were 34 men and 37 women with a mean age of 23.1±2.9 years and 15.2±1.9 years of education. FHN subjects were 35 men and 43 women with a mean age of 22.2±1.8 years and 15.8±1.4 years of education. All subjects were non-alcoholic and Caucasian. Subjects were recruited using newspaper advertisements asking for inquiries from persons interested in psychological research, or alcohol research, or the sons and daughters of heavy drinking fathers (cf. Finn et al. 1997). FHP subjects had alcoholism in two generations including their father. FHN subjects had no evidence of alcoholism in any first- or second-degree relatives. FHP/FHN status was ascertained with the Family History Assessment Module (FHAM: Rice et al. 1995), an interview using DSM-III-R criteria (Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, revised; American Psychiatric Association 1987). Non-alcoholic status (no alcohol abuse or dependence) was ascertained with the Psychiatric Diagnostic Interview (PDI: Othmer et al. 1981). FHP subjects drank an average of 3.2±2.3 drinks on 2.8±1.8 occasions per week. FHN subjects drank an average of 2.5±1.7 drinks on 2.4±1.6 occasions per week. Subjects were excluded if there was evidence of a personal or parental history of psychosis. Subjects were paid $7 per hour for their time in addition to any money won on the Go/No-Go task.

Apparatus

Alcohol

Subjects consumed one of two doses of alcohol after having fasted from food for 3 h: a dose targeting a breath-alcohol level of 0.07% (19 FHP men, 22 FHP women, 18 FHN men, 25 FHN women) or a dose targeting 0.09% (15 FHP men, 15 FHP women, 17 FHN men, 18 FHN women). These two dose levels were used to maximize the likelihood of detecting FHP: FHN differences in response to alcohol (cf. Stewart et al. 1992; Cohen et al. 1993 for dose-response studies of FHP and FHN subjects). Subjects drank either vodka or rum mixed with decaffeinated coca cola, orange juice or pineapple-orange juice mix. The 0.07% dose was 1.75 ml vodka or rum per kg body weight. The 0.09% dose was 2.25 ml (vodka or rum) per kg body weight. The alcohol was combined with mixer in a ratio of 1:3 parts by volume and divided into two drinks of equal volume. Breath alcohol was measured with an AlcoSensor III (Intoximeters, Inc.) breathalyzer. Breath alcohol levels at testing were 0.069±0.006 (FHP=0.069±0.006; FHN=0.068±0.004) and 0.087±0.007 (FHP=0.087±0.009; FHN=0.087±0.006) for each respective dosage.

Go/No-Go learning task

The task, adapted from Newman and Kosson (1986), involved the serial presentation on a computer screen (750 ms duration) of eight different two-digit numerical stimuli (four Go and four No/Go), displayed white on black background (1.5 cm×1.5 cm in size), organized into 20 blocks of eight stimuli, and pseudo-randomly presented with no more than three consecutive trials with either a Go or No-Go stimulus. Subjects were told that the task involved learning when to Go (bar press) or not to Go (withhold response) and that responses after some numbers would result in winning money ($0.17 per trial) but responses after others would lose money (loss of $0.17 per response). The response window was 750 ms and the inter-trial-interval (ITI) was 2.5 s. Reward contingencies (green background with +$0.17 in white) or punishment contingencies (red background with –$0.17 in white) were presented on the computer screen for 2 s immediately after a response (within the 2.5 s ITI). The task differed from Newman and Kosson's (1986) in that the contingencies were reversed after the end of the tenth block. Responses made after numbers that resulted in a reward during the first ten blocks (i.e., Go stimuli) resulted in the loss of money in the last ten blocks (became No-Go stimuli). Likewise, responses to the numbers designated as No-Go in the first ten blocks changed to Go stimuli in the last ten blocks and resulted in winning money.

There were two separate versions of the task with two different sets of stimuli, since subjects repeated the task in the alcohol and no-alcohol sessions. Specific numbers were chosen following Newman's (1987) suggestion for balancing even/odd and above/below 50. In version 1 (order 1) the Go stimuli in the first ten blocks were "08", "63", "74" and "25"; the No-Go were "58", "19", "14" and "79." In version 2 (order 1), the Go stimuli in the first ten blocks were "86", "17", "61" and "42"; the No/Go were "38", "11", "97" and "62." Order 2 for each version simply exchanged the designation of Go for No-Go. The order of presentation for versions 1 and 2 and orders 1 and 2 was completely counterbalanced across groups and alcohol conditions.

Cognitive assessment

Central executive working memory was assessed with the "digits backward" condition of the Digit Span subtest of the Wechsler Adult Intelligence Scale, Revised (WAIS-R: Wechsler 1981). In the digits backward condition, subjects must repeat increasingly longer strings of numbers in reverse order. The central executive aspect of working memory directs attentional resources for manipulating objects held in the short-term memory store (Baddeley 1986). Digits backward taps this central executive aspect of working memory (Stout et al. 1995). The sample mean on digits backward was 8.42±2.5 (FHN=8.2±2.7; FHP=8.6±2.4, no significant differences).

The Conditional Association Task (CAT: Petrides 1985) was used to measure the executive cognitive ability involving the selection of appropriate responses based upon specific learned stimulus-response associations that are brought into working memory to solve a specific problem. The CAT involves the trial and error learning of the arbitrary associations between each of six blank cards and each of six different randomly placed small lamps (on small box in front of the subject). A trial involves lighting a lamp and having the subject touch a card, one at a time, until she/he touches the correct card. Success at the task involves learning and then keeping in mind each of the six different matches and the effective utilization of this information to correctly select the appropriate response to meet a criterion of 18 correct consecutive trials (matches). Total trials and errors (square root transformed) were taken as a measure of executive function capacity (higher scores reflect less capacity). The sample mean for the raw score was 89.0±51.7 (FHP=94.0±48.4; FHN=84.4±54.1, no significant differences).

Procedure

The first testing session included an informed consent and the psychodiagnostic interviews. Subjects meeting the inclusion criteria came for two sessions, a no-alcohol and an alcohol session (session order counterbalanced across subjects). Women were required to test negative on a pregnancy test administered at the university health center prior to the alcohol session. In the no alcohol session, subjects were administered the Go/No-Go task, digit span, and the CAT along with other measures of cognitive function. In the alcohol session, after having the procedure explained, subjects were weighed and then consumed the alcohol (see above) in a 15-min period. Breath alcohol (BAL) was monitored every 5 min and then every minute as BAL approached the target peak level. Sub-

jects were then administered the Go/No-Go task. Digit Span was also administered in the alcohol session after the Go/No-Go task; however, due to a procedural error, digit span was included in this session only part way through the study (n=116 subjects).

Results

The effects of alcohol on Go/No-Go learning

False alarm rates

Our first hypothesis that alcohol would increase false alarms was tested using an ANOVA with Family History (FHP/FHN) and Dose (0.07/0.09%) as the between-group factors, and Beverage (no-alcohol/alcohol) and Task phase (pre-reversal/post-reversal) as the within-group factors. The ANOVA revealed that alcohol increased false alarm rates [Main effect: $F(1,145)=13.7$, $P<0.0005$]. There were no significant effects involving family history or dose. The only other significant effect was a main effect of Phase [$F(1,145)=62.5$, $P<0.0001$]. Table 1 lists the means and standard deviations for false alarm (and hit rates) for each phase and beverage condition. A preliminary analysis indicated no significant sex differences in response to alcohol.

A post-hoc analysis was conducted to explore whether alcohol had a specific effect on false alarms after contingency reversal (phase 2) when controlling for its effect on false alarms in phase 1. ANCOVA (Family history by Beverage) on false alarms in phase 2 was conducted while covarying out a measure of alcohol's effect false alarms in phase 1 (false alarms in alcohol minus false alarms in no-alcohol during phase 1). The ANCOVA revealed that alcohol still had the effect of increasing false alarms after reversal [$F(1,146)=10.1$, $P<0.005$].

Table 1 Mean false alarm and hit rates for both phases and beveraage conditions

Measure	No-alcohol	Alcohol
False alarm rates		
Phase 1	0.34±0.15	0.39±0.18
Phase 2	0.25±0.17	0.33±0.22
Hit rates		
Phase 1	0.78±.015	0.82±0.12
Phase 2	0.77±0.16	0.80±0.12

There was also a significant alcohol by covariate interaction [$F(1,146)=48.8$, $P<0.0001$], indicating that there was a significant alcohol main effect only for those with alcohol-induced increases in false alarms in phase 1.

Hit rates

ANOVA (same model as above) was also used to investigate the effects of alcohol on hit rates. Alcohol significantly increased hit rates [Main effect: $F(1,145)=9.7$, $P<0.005$]. Table 1 displays the mean hit rates for phases 1 and 2 in both beverage sessions. There was also a Main effect of family history [$F(1,145)=4.03$, $P<0.05$]. FHPs had modestly higher hit rates (mean=0.81±0.08) than FHNs (mean=0.78±0.12). There were no significant dose-related effects. A preliminary analysis indicated no significant sex differences in response to alcohol.

Working memory, conditional associative learning, and effects of alcohol on Go/No-Go learning

A general linear models regression equation (SAS GLM procedure: SAS Institute Inc. 1996) was used to test the hypothesis that alcohol would cause greater disruptions in response inhibition in persons with either low capacity working memory or low conditional associative learning ability. Predictor variables were the main effects and interactions of CAT, digits backward, and beverage (treated as a within-group factor). CAT scores and digits backward (working memory: WM) were centered on their respective means (raw score–mean) for the interaction terms. Table 2 lists the correlations between the cognitive measures and false alarm and hit rates in both task phases and beverage conditions.

The GLM analysis of false alarm rates revealed significant main effects of CAT scores [$F(1,145)=27.3$, $P<0.0001$], digits backward [$F(1,145)=23.0$, $P<0.0001$], and beverage [$F(1,145)=15.6$, $P<0.0001$] and significant of beverage by digits backward interaction [$F(1,145=9.15$, $P<0.005$]. For simple main effects testing, we divided subjects into those with high-and those with low-capacity working memory (WM) using a median split of the digits backward score. In support of our hypothesis, alcohol increased false alarm rates in low-WM [Main effect: $F(1,76)=21.0$, $P<0.0001$] but not

Table 2 Working memory and executive function while sober and Go/No-Go performance in alcohol and no-alcohol conditions

	False alarm rates				Hit rates			
	No-alcohol		Alcohol		No-alcohol		Alcohol	
	Phase 1	Phase 2	Phase 1	Phase 2	Phase 1	Phase 2	Phase 1	Phase 2
Digits backward	–0.15	–0.16[a]	–0.32[c]	–0.37[c]	0.15	0.12	0.12	0.14
CAT	0.16[a]	0.30[b]	0.31[c]	0.35[c]	0.01	–0.05	–0.04	–0.18[a]

CAT, conditional association test (square root of total trials and errors)
[a] $P<0.05$, [b] $P<0.001$; [c] $P<0.0001$

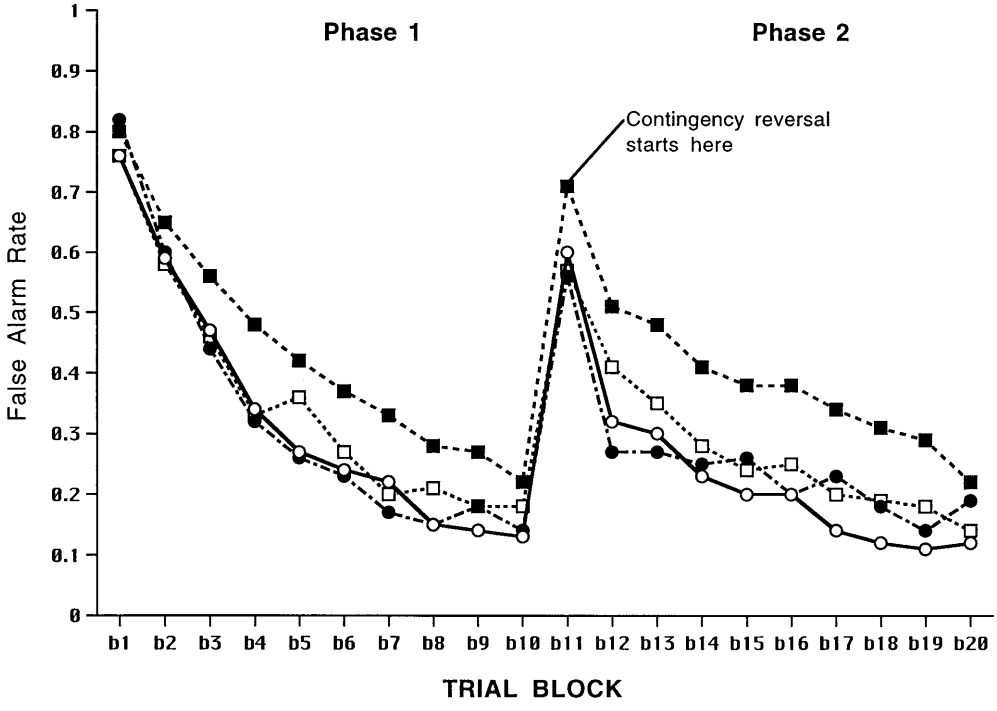

Fig. 1 The effects of alcohol on false alarm rates during phase 1 (blocks 1–10) and phase 2 (blocks 11–20 after contingency reversal) in subjects with high and low working memory capacity. ○ High working memory: no alcohol, □ low working memory: no alcohol, ● high working memory: alcohol, ■ low working memory: alcohol

high-WM subjects [$F(1,69)=0.4$, NS]. False alarm rates were also significantly higher in low-WM subjects than high-WM subjects in the alcohol session [Main effect: $F(1,147)=19.9$, $P<0.0001$]. Figure 1 displays the false alarm rates for high- and low-WM groups by block of eight stimuli during phase 1 (pre-reversal) and phase 2 (post-reversal) of both beverage conditions. The data are displayed by block to illustrate the learning curves. The beverage by CAT interaction was not significant [$F(1,145)=3.4$, $P<0.07$].

The GLM analysis of hit rates revealed significant main effects of digits backward [$F(1,145)=4.69$, $P<0.05$] and beverage [$F(1,145)=9.27$, $P<0.005$]. There were no significant interactions between beverage and either digits backward or CAT scores [$F(1,145)=0.22$ and 0.82, respectively]. There were no other significant effects.

Alcohol effects on working memory

We conducted a post-hoc analysis with the subset of subjects who were administered the digits backward measure in both alcohol and no-alcohol sessions to investigate whether: 1) alcohol had a direct effect on working memory, and 2) whether the effect of alcohol on working memory varied as a function of baseline WM capacity (i.e., high- versus low-capacity baseline working memory). Subjects were divided into high- and low-WM groups (median split) and the data were analyzed using an ANOVA (Beverage by WM). The results revealed a significant main effect of beverage [$F(1,114)=11.0$, $P<0.01$] and a significant interaction of baseline working memory capacity by beverage [$F(1,112)=22.0$, $P<0.0001$]. Simple effects analyses revealed that alcohol significantly reduced working memory capacity but only in high-WM subjects [$F(1,52)=25.8$, $P<0.0001$: Digits backward at baseline=10.7±1.5; after alcohol=9.1±2.3] and not in low-WM subjects (baseline=6.48±1.0; after alcohol: 6.76±2.18).

Working memory, conditional associative learning, and the effects of contingency reversal

A GLM regression equation was used to test our last hypothesis that subjects with low conditional associative learning ability or low working memory capacity will develop inhibitory responses after contingency reversal less effectively (i.e., have higher false alarm rates). The independent variables were the CAT, digits backward (WM), and contingency reversal (phase 1 hit rates/phase 2 false alarm rates were treated as a within factor). The GLM also included the interaction terms for the independent variables. Phase 1 false alarm rates served as a covariate because false alarms during phase 1 are associated with the dependent measures (phase 1 hits and phase 2 false alarms).

The GLM analysis revealed significant main effects of Phase [$F(1,144)=234.6$, $P<0.0001$] and Digits backwards [$F(1,144)=5.9$, $P<0.05$]. GLM also revealed significant a CAT by Phase interaction [$F(1,144)=4.9$, $P<0.05$] and a significant Digits backward by Phase interaction [$F(1,144)=7.5$, $P<0.01$]. Simple effects analysis of the interaction supported our hypothesis. Subjects were divided into low- and high-CAT groups using a median split of CAT scores. Low-CAT subjects had higher false alarm rates during phase 2 than high-CAT subjects [Main effect: $F(1,147)=9.97$, $P<0.01$, see Fig. 2]. Low-

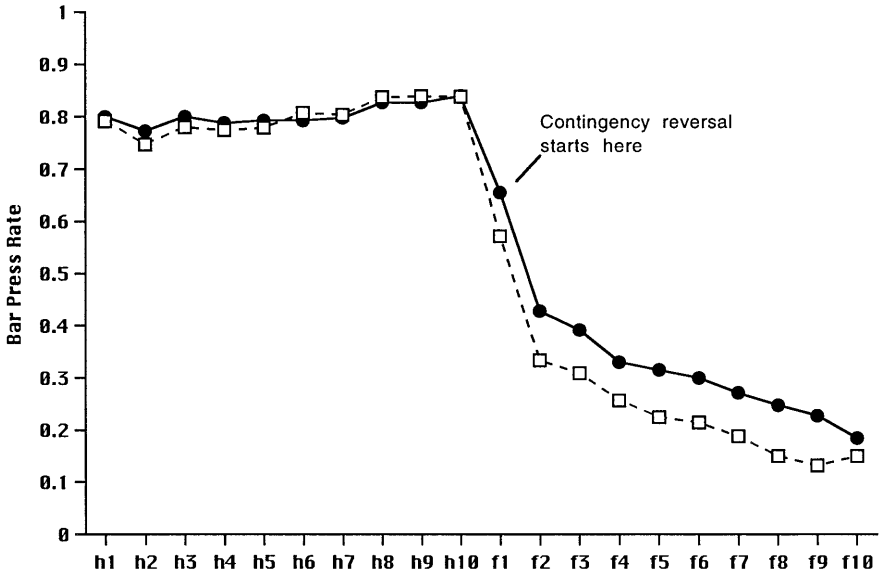

Fig. 2 The effects of contingency reversal on response inhibition in subjects with high and low scores on the conditional association task (*CAT*). H1–H10=hit rates to Go stimuli in the first ten blocks. F1–F10=false alarm rates to No-Go stimuli in the ten blocks after contingency reversal. High CAT reflects better performance on the test (lower scores). Low cat reflects poorer performance (high scores) □ High CAT group, ● low CAT group

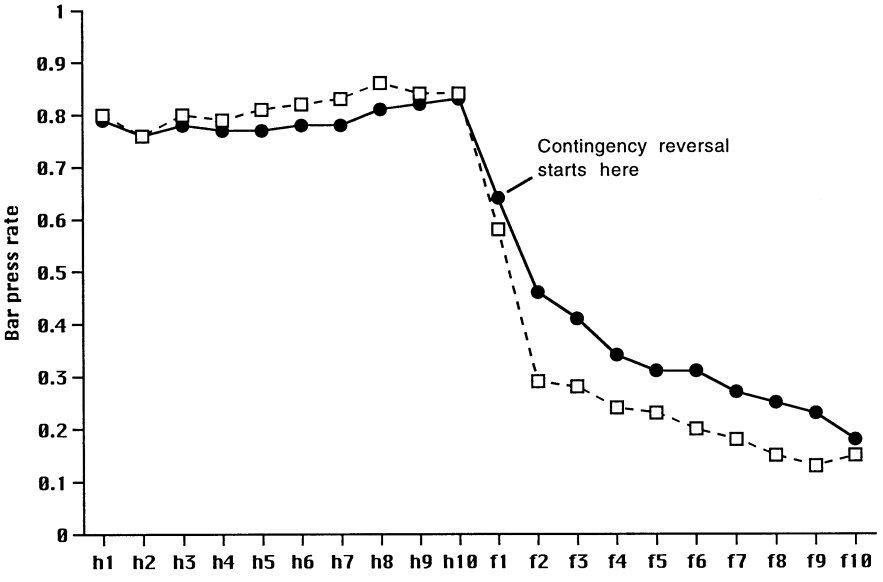

Fig. 3 The effects of contingency reversal on response inhibition in subjects with high and low working memory capacity. H1–H10=hit rates to Go stimuli in the first ten blocks. F1–F10=false alarm rates to No-Go stimuli in the ten blocks after contingency reversal. □ High working memory, ● low working memory

WM subjects also had higher false alarms in phase 2 compared with high WM subjects [$F(1,147)=14.2$, $P<0.001$, see Fig. 3].

Discussion

The purpose of this study was to test our cognitive-modulation model of behavioral inhibition by examining the effects of alcohol on response inhibition on a Go/No-Go learning task. The results supported our model of the modulatory roles of working memory and conditional associative learning on behavioral inhibition. The data also suggested an individual-differences mechanism by which alcohol increases impulsive behavior. Only subjects with low capacity in the central executive aspect of working memory (as assessed with digits backward) showed alcohol-induced increases in false alarm rates. However, inconsistent with the results of studies reporting risk-related differences in EEG and autonomic responses to alcohol (e.g., Stewart et al. 1992; Cohen et al. 1993), we found no differences between FHP and FHN subjects in the effects of alcohol on Go/No-Go learning. Finally, lower scores on our measures of central executive working memory and conditional associative learning were also associated with greater difficulty learning to inhibit responses after contingency reversal. We urge some caution in interpreting these results because the use of only single measures of working memory and conditional associative learning limits the generalizability of these findings.

Our model proposed that behavioral inhibition systems are modulated by central executive working memory and conditional associative learning, both of which we

conceptualize as executive cognitive processes. We postulated that alcohol would disrupt the modulation of behavioral inhibition in subjects with either low capacity in central executive working memory processes or low conditional associative learning ability. The model also proposed that subjects with low functional capacities in central executive working memory or conditional associative learning ability would show greater difficulty learning to inhibit behavior after a reversal of response contingencies. The results partially supported our first set of hypotheses. Alcohol appeared to disinhibit behavior (i.e., led to higher false alarms), but only in subjects with lower scores at baseline on the digits backward test. Conditional associative learning or familial alcoholism risk did not modulate responses to alcohol. These results suggest that low capacity central executive working memory (at least as assessed by digits backward) modulates alcohol's disinhibiting effect. Our results supported our second hypothesis. Low baseline scores on both digits backward and the CAT were both associated with higher false alarm rates after contingency reversal, suggesting that both of these measures tap processes which are critical for the adaptation of behavior to unexpected circumstances.

Although our cognitive-modulation model of behavioral inhibition predicts the effects of alcohol and contingency reversal on behavioral inhibition on a Go/No-Go learning task, it is by no means exhaustive. For instance, it is likely that other factors influence Go/No-Go learning, such as response to reward or punishment (Newman 1987; Lykken 1995). In addition, our model does not discuss other mechanisms underlying behavioral regulation, such as error detection (Scheffers et al. 1996), conditionability (Finn et al. 1994), aversive emotional processes (Patrick 1994; Lykken 1995), individual differences in activity in behavioral inhibition or behavioral activation (Gray 1987b), or temperamental traits such as harm avoidance or novelty seeking (Masse and Tremblay 1997). Finally, our measures (digits backward and CAT) are rather simple and imperfect measures of complex, dynamic processes. This is perhaps especially true of the CAT which we used to assess a very specific and complex, multilevel, multistage executive cognitive process.

Although we cannot determine from our data the mechanisms by which alcohol increased false alarm rates in subjects with low working memory capacity, the results suggest that it was not simply by reducing overall working memory capacity. Alcohol did not reduce working memory capacity in those subjects who showed significant increases in false alarms (i.e., low-WM subjects). We did not assess alcohol effects on CAT performance, although it seems likely that performance on that test would be compromised by alcohol given its complexity. We speculate that alcohol probably: 1). decreases activity in behavioral inhibition systems, 2) compromises, or slows, complex associative executive functions to some degree, and 3) interferes with the interfaces between behavioral inhibition (limbic) systems and working memory and executive function (Finn 1999). Research into the precise mechanisms by which alcohol might lead to impulsive behavior in persons with low capacity working memory is an important, and potentially fruitful, direction for this kind of research.

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References

American Psychiatric Association (1987) Diagnostic and statistical manual of mental disorders, 3rd edn revised. APA, Washington, D.C.

Baddeley AD (1986) Working memory. Oxford University Press, Oxford

Barkley RA (1997) Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. Psychol Bull 121:65–94

Barratt ES, Patton JH (1983) Impulsivity: cognitive, behavioral, and psychophysiological correlates. In: Zuckerman M (ed) Biological bases of sensation seeking, impulsivity, and anxiety. Erlbaum, Hillsdale, pp 77–116

Bates ME, Pandina RJ (1992) Familial alcoholism and premorbid cognitive deficit: a failure to replicate subtype differences. J Stud Alcohol 53:320–327

Cloninger CR, Sigvardsson S, Bohman M. (1988) Childhood personality predicts alcohol abuse in young adults. Alcohol Clin Exp Res 12:494–505

Cohen HL, Porjesz B, Begleiter H (1993) The effects of ethanol on EEG activity in males at risk for alcoholism. Electroencephalogr Clin Neurophysiol 86:368–376

Cooper ML (1992) Alcohol and increased behavioral risk for AIDS. Alcohol Health Res World 16:64–72

Eysenck SBG, Eysenck HJ (1978) Impulsiveness and venturesomeness: their position in a dimensional system of personality description. Psychol Rep 43:1247–1255

Finn PR (1999) Studies of the acute effects of alcohol on cognition and impulsive-disinhibitory behavior. Res Monogr (in press)

Finn PR, Kessler DN, Hussong AM (1994) Risk for alcoholism and classical conditioning to signals for punishment: evidence for a weak behavioral inhibition system. J Abnorm Psychol 103:293–301

Finn PR, Sharkansky EJ, Viken R, West TL, Sandy J, Bufferd GM (1997) Heterogeneity in the families of sons of alcoholics: the impact of familial vulnerability type on offspring characteristics. J Abnorm Psychol 106:26–36

Gerbing DW, Ahadi SA, Patton JH (1987) Toward a conceptualization of impulsivity: components across the behavioral and self-report domains. Multivar Behav Res 22:357–379

Giancola PR (1995) Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. Aggress Behav 21:431–450

Giancola PR, Zeichner A (1994) Neuropsychological performance on tests of frontal-lobe functioning and aggressive behavior. J Abnorm Psychol 103:832–855

Gray JA (1987a) The psychology of fear and stress, 2nd edn. Cambridge University Press, New York

Gray JA (1987b) Perspectives on anxiety and impulsivity: a commentary. J Res Person 21:493–509

Kimberg DY, D'Esposito M, Farah MJ (1997) Cognitive functions in the prefrontal cortex – working memory and executive control. Curr Dir Psychol Sci 6:185–192

Kirby KN, Petry NM, Bickel W K (1999). Heroin addicts have higher discount rates for delayed rewards than non-drug using controls. J Exp Psychol Gen 128:78–87

Lau MA, Pihl RO, Peterson JB (1995) Provocation, acute alcohol intoxication, cognitive performance, and aggression. J Abnorm Psychol 104:150–155

Logan GD, Schachar RJ, Tannock R (1997) Impulsivity and inhibitory control. Psychol Sci 8:60–64
Luengo MA, Carrillo-de-la-Pena MT, Otero JM, Romero E (1994) A short-term longitudinal study of impulsivity and antisocial behavior. J Person Soc Psychol 66:542–548
Lyyken DT (1995) The antisocial personalities. Erlbaum, Hillsdale
Masse LC, Tremblay RE (1997) Behavior of boys in kindergarten and the onset of substance abuse during adolescence. Arch Gen Psychiatry 54:62–68
Mulvihill LE, Skilling TA, Vogel-Sprott M (1997) Alcohol and the ability to inhibit behavior in men and women. J Stud Alcohol 58:600–605
Newman JP (1987) Reaction to punishment in extraverts and psychopaths: implications for the impulsive behavior of disinhibited individuals. J Res Person 21:464–480
Newman JP, Kosson DS (1986) Passive avoidance learning in psychopathic and nonpsychopathic offenders. J Abnorm Psychol 95:252–256
Othmer E, Penick EC, Powell BJ (1981) Psychiatric diagnostic interview. Western Psychological Services, Los Angeles
Parker JDA, Bagby RM (1997) Impulsivity in adults: a critical review of measurement approaches. In: Webster CD, Jackson MA (eds) Impulsivity: theory, assessment, and treatment. Guilford, New York, pp 142–157
Patrick CJ (1994) Emotion and psychopathy: startling new insights. Psychophysiology 31:319–330
Peterson JB, Finn PR, Pihl RO (1992) Cognitive dysfunction and the inherited predisposition to alcoholism. J Stud Alcohol 53:154–160
Petrides M (1985) Deficits on conditional associative-learning tasks after frontal- and temporal-lobe lesions in man. Neuropsychology 20:601–614
Petrides M, Alivisatos B, Evans A, Meyer E (1993) Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. Proc Natl Acad Sci USA 90:873–877
Rice JP, Reich T, Bucholz KK, Neuman RJ, Fishman R, Rochberg N, Hesselbrock VM, Nurnberger JI, Schuckit MA, Begleiter H (1995) Comparison of direct interview and family history of diagnoses of alcohol dependence. Alcohol Clin Exp Res 19:1018–1023
SAS Institute (1996) SAS software release 6.12. SAS, Cary, N.C.
Schachar RJ, Tannock R, Logan GD (1993) Inhibitory control, impulsiveness, and attention deficit disorder. Clin Psychol Rev 13:721–739
Scheffers WJ, Coles MGH, Bernstein P, Gehring WJ, Donchin E (1996) Event-related brain potentials and error-related processing: an analysis of incorrect responses to go and no-go stimuli. Psychophysiology 33:42–53
Schuckit MA, Butters N, Lyn L, Irwin M (1987) Neuropsychologic deficits and the risk for alcoholism. Neuropsychopharmacology 1:45–53
Sher K J, Walitzer KS, Wood PK, Brent EE (1991) Characteristics of children of alcoholics: putative risk factors, substance use and abuse and psychopathology. J Abnorm Psychol 100:427–448
Steele CM, Southwick L (1985) Alcohol and social behavior I: the psychology of drunken excess. J Person Soc Psychol 48:18–34
Stewart SH, Finn PR, Pihl RO (1992) The effects of alcohol on the cardiovascular stress response in men at high risk for alcoholism: a dose response study. J Stud Alcohol 53:499–506
Stout JC, Salmon DP, Butters N, Taylor M, Peavy G, Heindel WC, Delis DC, Ryan L, Atkinson JH, Chandler JL, Grant (1995) Decline in working memory associate with HIV infection. Psychol Med 25:1221–1232
Tarter RE, Jacob T, Bremer DL (1989) Specific cognitive impairments in sons of early onset alcoholics. Alcohol Clin Exp Res 13:786–789
Wechsler D (1981) Manual for the Wechsler Adult Intelligence Scale, revised. Psychological Corporation, New York
White JL, Moffitt TE, Caspi A, Bartusch DJ, Needles DJ, Stouthamer-Loeber M (1994) Measuring impulsivity and examining its relationship to delinquency. J Abnorm Psychol 103:192–205
Zelazo PD, Frye D (1998) Cogntive complexity and control: II. the development of executive function in childhood. Curr Dir Psychol Sci 7:121–126