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# Implicit Activation of Alcohol Concepts by Negative Affective Cues Distinguishes Between Problem Drinkers With High and Low Psychiatric Distress

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This study tested the hypothesis that negative affective (NEG) cues activate alcohol concepts more strongly in problem drinkers with high rather than low psychiatric distress (PD) and that a parallel difference applies to activation of NEG concepts by alcohol cues. A lexical decision task assessed memory activation in 36 problem drinkers with high vs. low scores on the Symptom Checklist–90. High PD drinkers displayed activation in each condition; low PD drinkers displayed inhibition. Activation of alcohol concepts by NEG cues correlated with intensity of PD, bias to drink in NEG states, and confidence to avoid drinking in these states. Results corroborate in vivo cue reactivity studies and indicate a functional difference between problem drinkers with high and low PD.

In the general North American population, more than half of all individuals with an alcohol use disorder also meet formal diagnostic criteria for a nonsubstance use psychiatric disorder (Regier et al., 1984). Similar comorbidity rates have been observed in treatment-seeking problem drinkers (Ross, Glaser, & Germanson, 1988). With the exception of major depression, comorbidity is generally associated with poorer treatment outcomes in problem drinkers, including heavier posttreatment drinking (Kranzler, Del Boca, & Rounsaville, 1996; Rounsaville, Dolinksy, Babor, & Meyer, 1987) and higher rates of rehospitalization (Slater & Linn, 1982). Thus, comorbidity is a widespread problem that undermines the treatment of problem drinkers.

Although certain psychiatric diagnoses are more likely to co-occur with problem drinking, the detrimental effects of comorbidity on treatment outcome appear to be nonspecific (Kranzler et al., 1996). That is, more diagnoses coincide with heavier posttreatment drinking which, in turn, coincides with greater psychiatric severity. The nonspecific effect of comorbidity suggests that psychiatric distress (PD) per se may be a critical determinant of treatment outcome. Accordingly, high psychiatric severity on the Addiction Severity Index has been found to predict poorer treatment outcome in problem drinkers (McLellan, Luborsky, Woody, O'Brien, & Druley, 1983), and a similar relation was observed when PD was measured with the Symptom Checklist-90 (SCL-90; Slater & Linn, 1982). Of the numerous correlates of substance abuse, the extent of pretreatment PD has been identified as the single best predictor of treatment outcome in substance abusers (McLellan, Childress, Ehrman, & O'Brien, 1986).[1]

One way PD may undermine treatment outcome is by promoting relapse. It is well established that negative affect is the most common antecedent of relapse in problem drinkers (e.g., Litman, Stapleton, Oppenheim, Peleg, & Jackson, 1983; Marlatt & Gordon, 1985; Vuchinich & Tucker, 1996). Furthermore, negative affect is more likely to precede a major relapse than a minor one (Hodgins, el-Guebaly, & Armstrong, 1995). Self-report indexes have shown that the intensity of negative affect is significantly higher in psychiatric outpatients with an alcohol use disorder than in those with no alcohol use disorder (Zack, Toneatto, & Streiner, 1998). This may reflect the summation or interaction of PD and situational negative affect in problem drinkers (McCusker & Brown, 1991). The resulting increase in the actual or perceived intensity or frequency of negative affect may increase the risk of relapse in high PD drinkers. In accord with this suggestion, alcoholics with a negative temperament, as measured by the General Temperament Survey (Clark & Watson, 1989), were significantly more likely to abuse alcohol in negative affective states than were alcoholics with a positive temperament (Cannon, Leeka, Patterson, & Baker, 1990; Cannon et al., 1992). The correspondence between PD and drinking in negative affective states, coupled with the strong association between negative affect and relapse, suggests that differential sensitivity to negative affect may predispose problem drinkers with high PD to relapse when in negative affective states. Accordingly, problem drinkers whose PD symptoms increase after detoxification are more likely to relapse (Brown, Irwin, & Schuckit, 1991; Svanum & McAdoo, 1989), and self-medication of these symptoms is commonly cited by high PD drinkers as the reason for

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[1] In the present article, the term *PD* is used when referring to dispositional psychiatric distress, and the term *negative affect* is used when referring to situational distress.

their return to alcohol use (e.g., La Bounty, Hatsukami, Morgan, & Nelson, 1992).

Although negative affect often precedes relapse, research testing a craving model of relapse has found that positive affect can also induce strong cravings in cigarette smokers (Tiffany & Drobes, 1990). Similarly, research testing the effects of mood induction on cue reactivity found that a substantial minority of problem drinkers (24%) reported positive mood states as their strongest drinking trigger (Rubonis et al., 1994). These investigators suggested that this subgroup of drinkers may respond differently to negative mood cues than do most problem drinkers. In addition, the bias to drink in positive mood states appears to be associated with positive temperament (Cannon et al., 1990, 1992). These findings indicate that positive affect may be an important antecedent of alcohol use in certain individuals and that positive affective cues are more likely to trigger drinking in problem drinkers whose usual affective state is positive.

The probability of relapse depends, in part, on a drinker's confidence in his or her ability to successfully abstain (Rollnick & Heather, 1982). The Situational Confidence Questionnaire (Annis & Graham, 1988) was developed to "assess Bandura's concept of self-efficacy in relation to changes in drinking behaviour" (Solomon & Annis, 1990, p. 661). Simply put, self-efficacy reflects the belief that one can successfully execute a particular behavior in a particular situation. With respect to alcohol use, self-efficacy reflects the belief that one can abstain from drinking in a particular affective or environmental state. Theoretical accounts have long implicated self-efficacy beliefs in relapse to alcohol use by problem drinkers (e.g., Marlatt & Gordon, 1980). Cue exposure studies have verified these accounts (Cooney, Gillespie, Baker, & Kaplan, 1987; Rubonis et al., 1994), with exposure to alcohol cues decreasing self-efficacy to abstain from alcohol in problem drinkers and negative mood induction amplifying this effect.

The cue exposure findings are consistent with classical conditioning models of craving to drink (Ludwig & Wikler, 1974), and research has found that conditioned cues for drinking are often interoceptive (e.g., negative affect) rather than environmental (Ludwig, 1986). The reliable and frequent pairing of negative affect and heavy drinking in problem drinkers with high PD (Bibb & Chambless, 1986; Chambless, Cherney, Caputo, & Rheinstein, 1987; Smail, Stockwell, Canter, & Hodson, 1984) would provide an opportunity for conditioned associations to develop between negative affect and alcohol use.

Research showing a concomitant change in autonomic reactivity and self-reported levels of self-efficacy in response to alcohol cues suggests that cognitive factors (e.g., beliefs and expectations) mediate between conditioned responses and alcohol use (Laberg, 1990; Niaura et al., 1988). Social learning theory recognizes the contribution of both conditioning and cognitive processes to alcohol use and argues that these processes may be connected through memory (Bradizza, Stasiewicz, & Maisto, 1994; Stacy, 1995). Specifically, conditioned associations are believed to be encoded in a memory network (Kehoe, 1992; Rescorla, 1992) that also contains affective and behavioral information (Baker, Morse, & Sherman, 1987). As a result, both affective stimuli themselves and thoughts about these stimuli may act as conditioned stimuli or responses (Bradizza et al., 1994; Rubonis et al., 1994).

The importance of memory processes as determinants of drinking has been widely verified in social drinkers (e.g., Rather & Goldman, 1994; Stacy, 1995) and in problem drinkers (Baker et al., 1987). Much of this work has focused on the effects of expectancies—learned prospective information about a forthcoming event evoked by the occurrence of a historical antecedent of that event (Bolles, 1972). The research shows that expectancies about the effects of alcohol are encoded in memory (Goldman, Brown, Christiansen, & Smith, 1991; Stacy, Leigh, & Weingardt, 1994), are clustered in predictable ways in individuals with different drinking habits (Rather & Goldman, 1994), and can be evaluated using both explicit (conscious, deliberate; Earlywine, 1995) and implicit (unconscious, involuntary; Stacy, 1997) memory measures.

Recent research has used implicit memory tasks to investigate the effects of affective stimuli on alcohol-related cognition and drinking behavior in social drinkers. Weingardt, Stacy, and Leigh (1996) found that brief exposure to phrases denoting positive affective outcomes of drinking (e.g., "He felt relaxed when he was _____") resulted in faster naming of alcohol-related target words (e.g., *bombed*) than did neutral cues (e.g., "They said they were_____"). The occurrence of a faster naming response or lexical (word–nonword) decision to a target stimulus owing to the prior presentation of a conceptually related verbal cue—semantic priming—is believed to reflect the activation of the target concept in memory (e.g., McNamara, 1992). Activation, in turn, denotes a process whereby exposure to a stimulus confers energy to its representation and related representations in memory, making them more available for further cognitive processing (Collins & Loftus, 1975). Thus, compared with a neutral word like *pencil*, exposure to the word *doctor* reduces lexical decision response time to the letter string *n-u-r-s-e* by activating medically oriented concepts in memory. Faster lexical decision time to a target word preceded by a related as opposed to an unrelated word is a standard operational definition of *activation* in the cognitive literature.

Spreading activation theorists contend that activation occurs involuntarily and without conscious awareness (Collins & Loftus, 1975; Neely, 1977; Posner & Snyder, 1975). Yet, such activation can influence conscious decisions (Fazio & Williams, 1986) as well as complex behavior (McClelland & Rumelhart, 1985). In accord with the established effects of memory activation, Roehrich and Goldman (1995) found that social drinkers who were exposed to words denoting positive affective outcomes of drinking (e.g., *confident*) in a modified Stroop task (Mathews & MacLeod, 1985) drank more placebo beer in a subsequent, ostensibly unrelated taste test than did drinkers exposed to neutral words. The implicit nature of this effect was verified by the fact that no participant detected the relation between the Stroop prime task and the taste test. Weingardt et al.'s (1996) findings show that verbal positive affective correlates of drinking can activate alcohol-related cognitions in social drinkers without their awareness. Roehrich and Goldman's (1995) results show that such activation can have a direct causal effect on drinking behavior.

On average, social drinkers drink more often in positive than in negative affective states (Cunningham, Sobell, Sobell, Gavin, & Annis, 1995). This tendency may foster conditioned associations between positive affect and alcohol use, and these associations may in turn be represented in memory. Activation of alcohol concepts (Weingardt et al., 1996) and drinking behavior (Roehrich & Goldman, 1995) by positive affect words in social drinkers may partly derive from these associations. Unlike social drinkers, problem drinkers tend to drink more often in negative than in positive affective states, a bias that increases with levels of alcohol dependence (Cunningham et al., 1995). This drinking pattern may foster associations between negative affect and alcohol use in problem drinkers, associations that may also be encoded in memory. In this case, negative affective verbal cues should activate memory-based alcohol concepts in problem drinkers just as positive affective verbal cues activated alcohol concepts in social drinkers.

Cue exposure research with dependent drinkers shows that negative mood induction can increase desire to drink even when physical alcohol cues (e.g., smell of alcohol) are absent (Litt, Cooney, Kadden, & Gaupp, 1990). This research supports the notion that negative affective cues may activate alcohol-related concepts in problem drinkers. Litt et al. also noted high variability in the degree of drinking desire evoked by the mood induction, suggesting that some factor or factors apart from dependence contributed to the effects of negative mood cues. McCusker and Brown (1991) found that dependent drinkers with high levels of Neuroticism and Introversion on the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1963) displayed greater anxiety when exposed to physical alcohol cues than did drinkers low in Neurotic–Introversion. The degree of anxiety evoked by alcohol cues in turn predicted the desire to drink. Neurotic–Introvert profiles on the EPI coincide with trait anxiety as well as trait depression (Gershuny & Sher, 1998). This correspondence indicates a rather general relation between Neurotic–Introversion and mood disturbance and suggests that PD may have moderated the anxiogenic effects of alcohol cues in McCusker and Brown's study. Their observed relation between increased anxiety and desire to drink further suggests that PD may have contributed to the variable effects of negative mood cues on desire to drink in Litt et al.'s study.

Litt et al. (1990) acknowledged that demand characteristics may have influenced the effects of their mood induction procedure, in that "subjects may have expected to be more reactive in the negative mood induction conditions and therefore may have reported greater desire for alcohol" (p. 145). The present study was designed to test the effects of negative affective cues and alcohol-related cues on memory activation in problem drinkers, while controlling for potential biases such as demand characteristics or self-perceptions. To this end, an implicit paradigm (Hill & Paynter, 1992) was used to assess memory activation in problem drinkers with high and low levels of PD.

If PD moderates the effects of negative affective cues, drinkers with high PD should display more activation of alcohol concepts by negative affective words than should drinkers with low PD. If PD also moderates the effects of alcohol cues, drinkers with high PD should display greater activation of negative affective concepts by alcohol words than should drinkers with low PD. If classical conditioning contributes to memory activation, a historical association between negative mood (the conditioned stimulus; CS) and alcohol (the unconditioned stimulus; UCS) should correlate with activation of alcohol concepts by negative affective cues. Thus, those with a stronger bias to drink in negative affective states should display greater activation of alcohol concepts by negative affective cues. If self-efficacy is also related to memory activation, greater activation of alcohol concepts by negative affective cues should predict less confidence to avoid drinking in negative affective states.

A lexical decision task measured implicit activation of alcohol-related and negative affective concepts in memory. An affective drinking history questionnaire measured the frequency of alcohol use in negative and positive affective states. The Situational Confidence Questionnaire measured self-efficacy to avoid drinking in several affective and environmental states.

## Method

### *Participants*

Twenty-nine male and 7 female problem drinkers (35 Caucasian, 1 Native Canadian), ages 21 to 54 years, were recruited from an outpatient population at the Addiction Research Foundation division of the Centre for Addiction and Mental Health (Toronto, Ontario, Canada). Prospective participants completed the SCL-90 (Derogatis, 1975) at intake or at an initial assessment before treatment. The SCL-90 is a self-report rating scale that measures PD on nine dimensions. The Obsessive–Compulsive (OC), Anxiety (ANX), and Phobic Anxiety (PHOB) subscales of the SCL-90 tap symptoms corresponding to *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*) criteria for obsessive–compulsive disorder, generalized anxiety, and agoraphobia, respectively (American Psychiatric Association, 1994; Derogatis, 1983). The Interpersonal Sensitivity (INT) subscale taps symptoms corresponding to *DSM–IV* criteria for social phobia. The Global Severity Index (GSI), based on the mean rating for all 90 items, measures overall PD.

Previous research with a large sample of outpatients ($N$ = 740) at the Foundation established norms on the SCL-90 for comorbid substance abusers (Zack et al., 1998). These norms provided empirical criteria for classifying participants as high PD or low PD. Those who scored above the 84th percentile ($\mu + \sigma$) for this population on at least one of the four anxiety-related subscales were classified as high PD (Group HPD, $n$ = 18). Those who scored below the mean on all four subscales and who reported no history of psychiatric illness were classified as low PD (Group LPD, $n$ = 18). The 84th percentile corresponded to scores of 2.55 (OC), 2.46 (INT), 2.55 (ANX), and 1.92 (PHOB). Predictably, these cutoff scores exceeded the 84th percentile in noncomorbid alcoholic inpatients: 1.92 (OC), 1.72 (INT), 1.73 (ANX), and 1.08 (PHOB; Mercier et al., 1992), but were lower than the 84th percentile for alcoholic inpatients with panic disorder: 2.90 (OC), 2.76 (INT), 2.96 (ANX), 2.16 (PHOB; Norton, Block, & Malan, 1991). The means for the subscales in our previous sample were 1.67 (OC), 1.55 (INT), 1.64 (ANX), and 0.97 (PHOB). Whereas depressive symptoms can decline rapidly with initial abstinence (Brown et al., 1995), anxiety symptoms often do not (Schuckit & Hesselbrock, 1994). The anxiety-related subscales of the SCL-90 thus provided a relatively reliable index of PD in this population.

All participants in Group HPD met *DSM–IV* criteria for alcohol dependence. In Group LPD, 17 participants met criteria for alcohol dependence, and 1 participant met criteria for alcohol abuse. The group difference in the frequency of each diagnosis was not significant, $\chi^2(1, N = 36) = 1.03, p > .20$. No participant had consumed alcohol or a benzodiazepine within 12 hr of the test. Two participants in Group HPD and 1 in Group LPD were taking an antidepressant medication; the group difference in number of medicated participants was not significant, $\chi^2(3, N = 36) = 1.03, p > .70$.[2] In addition, no participant in Group LPD scored above the mean for psychiatric outpatients on the Depression subscale of the SCL-90 ($M$ = 1.79; Derogatis & Cleary, 1977); the mean (*SD*) for the group was 1.00 (0.13). Thus, Group LPD's performance did not reflect a protective effect of depression in the absence of other PD (see Kranzler et al., 1996).

A number of additional psychiatric scales were administered after the lexical decision task. These included the Alcohol Dependence Scale (ADS; Skinner & Allen, 1982), the Anxiety Sensitivity Index—Revised (ASI; Peterson & Reiss, 1992), the State–Trait Anxiety Inventory (STAI; Speilberger, Gorsuch, & Lushene, 1970), the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), and the Situational Confidence Questionnaire (SCQ; Annis & Graham, 1988). Drinking habits for the preceding 90 days were assessed by the Timeline Followback (TLFB; Sobell & Sobell, 1992). Vocabulary was assessed by the Vocabulary subtest of the Wechsler Adult Intelligence Scale—Revised (WAIS–R; Wechsler, 1981).

A 2 (group) × 6 (scale) multivariate analysis of variance (MANOVA) assessing scores on the ADS, BDI, ASI, GSI, STAI–trait, and STAI–state obtained a significant multivariate effect of group, $F(6, 29) = 7.99, p < .001$. The univariate group difference was significant on each scale except the STAI–state (Bonferroni $p > .15$).[3] Therefore, Groups HPD and LPD differed in terms of the consequences of alcohol use they experienced, depression, anxiety sensitivity, overall PD, and dispositional anxiety. A discriminant function analysis of the supplemental scales showed that the variable with the highest canonical correlation was GSI ($R = .85$). This indicated that, of the six additional psychiatric scales, the variable that best distinguished between the groups was overall PD.

A $t$ test comparing the groups' scores on the Vocabulary subtest of the WAIS–R revealed no significant difference ($p > .50$). Therefore, word knowledge differences could not account for group differences in RT to the various word stimuli. The mean (*SD*) Vocabulary score for the entire sample, 40.3 (11.3), did not differ from the mean score for alcohol-dependent participants in the previous study that detected within-category activation of alcohol-related stimuli, 42.1 (6.3), $p > .50$ (Hill & Paynter, 1992). Therefore, the vocabulary of the present sample was sufficient to yield activation of alcohol-related words.

A 2 (group) × 5 (drinking measure) MANOVA compared the average duration (years) of drinking problems, drinks consumed per week, drinks per drinking day, number of heavy drinking days (>4 standard drinks), and number of abstinent days per week in Groups HPD and LPD. The multivariate difference between the groups was nonsignificant ($p > .80$), indicating that their overall drinking behavior was similar. Moreover, none of the univariate group differences were significant (Bonferroni $ps > .90$). The mean (*SD*) duration of drinking problems for the entire sample was 13.0 (8.3) years, and the overall mean weekly consumption was 56.4 (34.6) standard drinks. Thus, alcohol problems were generally long-standing, and the level of alcohol consumption was heavy (Wilkinson & LeBreton, 1986) but also quite variable. The lack of significant differences on all drinking measures ensured that group differences in performance on the lexical decision task could not be attributed to differences in the degree or duration of problem drinking.

All participants had normal or corrected-to-normal vision. A handheld J4-X A.L.E.R.T. breathalyzer (Alcohol Countermeasures Systems, Mississauga, Ontario, Canada) confirmed that participants' blood alcohol level was zero prior to testing. All participants provided informed consent prior to the study and received an honorarium of $10 upon completion.

### *Apparatus*

A 386 IBM-compatible PC administered the task and recorded the responses. Participants sat facing the screen at a distance of approximately 60 cm. Stimuli were white uppercase letter strings, 1 cm in height, that were presented against a black background. Prime stimuli appeared in the center of the screen; target stimuli appeared 1 cm below the center. Lexical decisions to target stimuli were made by pressing the "/" key for word responses or the "z" key for nonword responses. Colored stickers ensured that these two keys were clearly identified. The labels WORD and NONWORD were affixed to the lower right and left corners of the computer monitor, respectively, to control for confusion or memory lapse.

An affective drinking history questionnaire (ADHQ) assessed the frequency of drinking in negative and positive affective states. Participants rated how often they drank alcohol (beer, wine, or liquor) when they were in each of 70 affective states or situations. Frequency ratings (from *never* to *always*) were made on a 7-point Likert-type scale. The negative affective items were the 60 original words from the negative affective condition on the lexical decision task (see below for derivation procedure). The positive affective words were derived from items on the Positive Emotional States and Pleasant Times With Others subscales of the Inventory of Drinking Situations (Annis, Graham, & Davis, 1987). Factor analyses have shown that these two theoretical scales form one coherent empirical scale when assessed in problem drinkers (Cannon et al., 1990). The items reflected a variety of positive affective states (e.g., upbeat, relaxed, playful) and situations (party, dancing) associated with alcohol use. A reliability analysis determined that coefficient alpha was .97 for the 60 negative affective items and .88 for the 10 positive affective items. Therefore, the negative and positive affective scales were internally consistent.

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[2] New-generation antidepressants (i.e., selective serotonin reuptake inhibitors, SSRIs; venlafaxine) do not cause any disturbances of memory (Danion, 1993). Similarly, these drugs have negligible effects on choice reaction time (RT) and psychomotor speed (Hindmarch, 1995). Although SSRIs may enhance simple RT, they do not affect RT as a function of stimulus–response compatibility (Hasbroucq, Rihet, Blin, & Possamai, 1997). Furthermore, they do not alter electrophysiological responses such as P300 latency, which reflects speed of neural information processing, or P300 amplitude, which reflects the increased response to novel or salient stimuli (Semlitsch, Anderer, Saletu, Binder, & Decker, 1993). These findings indicated that performance on the lexical decision task would not be biased by SSRIs. Therefore, participants receiving an SSRI were not excluded from this study. SSRIs are used for a variety of reasons other than depression. For example, because of their low abuse liability and lack of synergistic effects with alcohol (Lader, 1996), SSRIs may be preferable to benzodiazepines for the treatment of insomnia that often accompanies initial abstinence in problem drinkers (Linnoila, 1989). The 1 participant in Group LPD who was taking an SSRI reported sleep disturbance on the SCL-90. However, because his scores on all other psychiatric indices (including the anxiety-related subscales and GSI of the SCL-90) met criteria for Group LPD, and he reported no current or previous history of psychiatric illness, he was assigned to this group. To ensure that inclusion of participants taking antidepressants did not bias the results of the lexical decision task, we reanalyzed RT scores with these participants excluded. The findings from the nonmedicated participants ($n = 33$) were entirely consistent with those of the sample as a whole, except that the rank correlation between affective drinking history and activation of alcohol targets by negative affective primes (NEG-ALC activation) was somewhat attenuated ($\tau = .19, p = .06$).

[3] The mean (*SD*) scores for Group HPD were 29.2 (10.4) on the ADS, 44.9 (11.7) on the ASI, 15.1 (6.7) on the BDI, 2.32 (0.79) on the GSI, 59.3 (13.7) on the STAI–trait, and 50.7 (16.7) on the STAI–state. The corresponding scores for Group LPD were 18.9 (7.5) on the ADS, 29.8 (11.7) on the ASI, 6.3 (5.5) on the BDI, 0.93 (0.48) on the GSI, 40.4 (11.3) on the STAI–trait, and 40.1 (10.5) on the STAI–state.

## Stimuli and Design

Stimuli were English words or nonwords.[4] Nonwords (NON) were derived by randomly selecting one fourth of the word stimuli, matching each word with a word of equal length and frequency of occurrence in print, then randomly replacing one vowel and one consonant in the matched word. This yielded a set of pronounceable facsimiles of real words (e.g., *mopical, slore*).

Five types of word stimuli were used: negative affective (NEG; e.g., *worry, hopeless*), alcohol-related (ALC; e.g., *beer, smashed*), neutral (NEU; e.g., *chimney, plank*), and two sets of neutral categorized words (CAT; e.g., *rabbit, shirt*). NEG words were derived from the symptom statements on the SCL-90. Responses on the SCL-90 from previous high PD substance abusers at the Foundation ($N = 240$) were used (Zack et al., 1998). The mean ratings for each of the 90 symptoms were computed for the previous sample and then ranked. The 60 symptoms with the highest mean ratings were then used as NEG stimuli. To obtain additional stimuli, we determined synonyms for each of the symptom words using Roget's Thesaurus (Lewis, 1978). ALC words were derived from previous studies that tested cognitive biases in alcohol-dependent participants (Hill & Paynter, 1992; Stetter, Ackermann, Bizer, Straube, & Mann, 1995). Supplementary ALC stimuli were derived from Roget's Thesaurus based on face validity (e.g., *cocktail*).

Prime stimuli for the baseline conditions (NEU–target) were the names of parts of a building derived from published word frequency norms (Battig & Montague, 1969). A control condition (CAT) tested semantic activation using neutral, categorized words from two explicit categories (four-legged mammals, articles of clothing) derived from the same norms.

The stimulus conditions tested on the task are outlined in Table 1. There were eight word–word conditions and four word–nonword conditions. Five of the word–word conditions were test conditions that contained putatively related primes and targets. Each of these five test conditions was evaluated against one of three baseline conditions that contained the same type of target preceded by an unrelated, neutral prime.

As indicated in Table 1, several conditions were included to validate the task or to explore additional cognitive relations. The NEG–ALC and ALC–NEG conditions tested the hypotheses. The ALC–ALC condition verified that the sample displayed within-category activation of alcohol concepts, in accordance with previous results from problem drinkers (Hill & Paynter, 1992). The CAT–CAT condition assessed RT when the prime and target categories were matched (CAT–$CAT_m$) or unmatched (CAT–$CAT_u$). Faster RT on matched trials would ensure that the task was capable of detecting activation due to a conceptual prime–target relation. The NEG–NEG condition tested within-category activation of negative affective cues. Previous research had detected activation of the threatening meanings of ambiguous words in anxious individuals with no alcohol problem (Richards & French, 1992), implying that a coherent network of negative affective concepts may exist in high PD individuals, similar to the alcohol network that exists in problem drinkers (Hill & Paynter, 1992).

There were equal numbers of word–word and word–nonword trials for each type of prime. Thus, in each condition, half of the targets were words and half were nonwords. Word–word and word–nonword trials were randomly interspersed, so that on any particular trial word and nonword targets were equally probable. The 12 prime–target conditions were also randomly distributed over trials, so that it was not possible to anticipate a particular stimulus or to detect a reliable association between prime and target stimuli. Stimuli were combined randomly into 18 different lists, so that items and conditions were unconfounded. Each list was seen by only 1 participant in Group HPD and 1 participant in Group LPD.

In light of previous research that detected differences in activation depending on how often a word appeared in print (Scarborough, Cortese, & Scarborough, 1977), the mean frequency of occurrence in print of each category of words was compared using a list of word frequency norms (Kucera & Francis, 1967). A one-way analysis of variance (ANOVA) of the mean frequency scores for each of the word types detected no significant difference, $F(4, 475) = 1.52$, $p > .10$. A one-way ANOVA also compared the mean word length in each of the word categories. This ANOVA obtained a significant result, $F(4, 475) = 13.69$, $p < .001$. Post hoc analyses using Tukey's honestly significant difference indicated that the mean number of letters in the NEG words ($M = 7.76$) exceeded the mean numbers of letters in the ALC ($M = 6.61$), NEU ($M = 6.53$), CAT-mammal ($M = 5.82$), and CAT-clothing ($M = 6.93$) conditions ($p < .05$), and that CAT-clothing words were longer than CAT-mammal words ($p < .05$). Previous research found that long words attenuate activation compared with short words (Balota & Chumbley, 1984). Thus, in the present study, activation effects may be more conservative on trials containing the longer NEG and CAT-clothing stimuli than they might otherwise be. However, there is no reason to expect that word length should differentially affect high PD and low PD participants.

Table 1
*Stimulus Conditions for Word and Nonword Trials in the Semantic Priming Task*

| Word–word trials[a] | | | | Word–nonword trials[b] | |
|---|---|---|---|---|---|
| Test | | Baseline | | Control | |
| Prime | Target | Prime | Target | Prime | Target |
| NEG | ALC | NEU | ALC | NEG | NON |
| ALC | NEG | NEU | NEG | ALC | NON |
| ALC | ALC | NEU | ALC | NEU | NON |
| NEG | NEG | NEU | NEG | CAT | NON |
| CAT | CAT-m | CAT | CAT-u | | |

*Note.* NEG = negative affective; ALC = alcohol-related; NEU = neutral; NON = nonword; CAT-m = neutral, categorical, matched; CAT-u = neutral, categorical, unmatched.
[a] Twenty trials per condition. [b] Forty trials per condition.

## Procedure

Testing occurred between intake and treatment, with participants advised to abstain from alcohol at this time. Upon reporting to the laboratory, participants were advised of the procedure and that the study had been approved by an Ethics Committee. They then signed a consent form and provided a breath sample. Participants with a positive blood alcohol level or who reported consuming alcohol or other psychotropic drugs within the previous 12 hr were excluded.

Each participant performed the task individually in a quiet, well-lit room. Verbal instructions were provided by the experimenter and were repeated on the screen prior to the task. Following the instructions, the participant performed 335 lexical decision RT trials. The first 15 trials were practice trials containing stimulus pairs from each prime–target condition. The remaining 320 trials were experimental trials. These trials were administered in five blocks of 60 and one block of 20, with a 1-min break between blocks. The sequence of events was identical on all trials: fixation stimulus (++++; 250 ms), blank screen (250 ms), prime (750 ms), target (until response), blank screen (1,000 ms). These stimulus parameters have been found to yield activation of the threatening interpretation of homographs in anxious participants (Richards & French, 1992) and of alcohol words in alcohol-dependent participants (Hill & Paynter, 1992).

Participants were instructed to read each prime silently and to decide as quickly and carefully as possible if the subsequent target was a word or a

[4] The complete set of stimuli is available from Martin Zack upon request.

nonword. To reduce confusion around colloquial ALC words (e.g., *booze*), we told participants that slang words "like *bash* or *busted*" also counted as real words. To reduce performance anxiety, we told participants not to worry if they made a mistake and to keep on working quickly and carefully on the remaining trials. Because of the large number of trials, and because the prime stimuli did not require an overt response, it was possible that participants would begin to focus selectively on the target stimuli as the trials continued. To help ensure continued attention to the primes, we gave participants a pop quiz immediately after they performed their practice trials. They were shown a list of eight words and were instructed to select the ones that occurred as a first word (i.e., the prime) on the practice trials and to indicate their degree of confidence (0–10) in each decision. The quiz raised the possibility of a subsequent recognition test following the experimental trials, and thus provided an incentive to continue attending to the prime stimuli. To reduce apprehension about a posttest, the experimenter assured participants after the quiz that "you will do fine provided you pay full attention to both the first and second letter string on every trial." The task took approximately 25 min to complete.

Immediately after the task, the experimenter administered the TLFB and Vocabulary subtest of the WAIS–R. Participants then received the questionnaire battery containing the additional psychiatric scales. After completing the battery, participants were asked if they had any "theories" about the purpose of the study, as well as how much attention they paid to the first and second stimulus on each decision trial. Although most participants indicated seeing several alcohol-related words, no participant detected a relation between the primes and targets. Participants consistently reported attending equally to the first and second stimulus on every trial. After these inquiries, participants were debriefed and paid. They agreed not to discuss the study with other potential participants.

## Results

### Participant Characteristics

An independent-samples $t$ test found no difference in the mean age of Groups HPD and LPD ($p > .90$). The mean (*SD*) age of the sample was 37.3 (7.7) years. A chi-square test of independence confirmed that the gender composition of the groups also did not differ (male = 81%, female = 19%; $p > .20$).

To verify the effectiveness of the assignment procedure, we used a 2 (group) × 4 (subscale) MANOVA to compare the scores of the two groups on the OC, INT, ANX, and PHOB subscales of the SCL-90. The analysis obtained a significant multivariate effect of group, $F(4, 31) = 14.78$, $p < .001$, confirming that the assignment procedure was effective in creating groups with significantly different levels of PD. The univariate group difference on each subscale was also significant (Bonferroni *ps* < .001). The mean (*SD*) scores of Group HPD were 2.74 (0.61), 2.64 (1.01), 2.37 (0.93), and 1.77 (1.22) for the OC, INT, ANX, and PHOB subscales, respectively. These scores corresponded to percentile ranks of 85 to 92 ($M = 89.8$) for psychiatric outpatients (Derogatis & Cleary, 1977), indicating rather extreme levels of PD. The mean (*SD*) scores of Group LPD were 1.11 (0.65), 0.98 (0.71), 0.81 (0.75), and 0.48 (0.41) for the OC, INT, ANX, and PHOB subscales, respectively. These scores corresponded to percentile ranks of 23 to 37 ($M = 31.8$) for psychiatric outpatients, reflecting low to moderate levels of PD.

The mean correlation between the four anxiety-related subscales of the SCL-90 and each additional psychiatric scale was substantial: .66 (ADS); .78 (ASI); .76 (BDI); .92 (GSI); .61 (STAI–state); .70 (STAI–trait); overall mean $r = .74$. Thus, on average, each subscale shared 55% of its variance with the additional psychiatric scales. These results indicate poor discriminant validity of the scales in this sample and suggest that, to a large degree, each scale tapped a common underlying construct, namely, PD. Consistent with this suggestion, inclusion of each additional scale as a covariate in the variance analyses (see below) reduced the between-groups difference ($\eta^2$) in NEG–ALC activation an average of 32%, and reduced the between-groups difference in ALC–NEG activation an average of 21%.

Table 2
*Mean and Standard Deviation Lexical Decision Response Time (in Milliseconds) on Test and Baseline Trials in Problem Drinkers With High Psychiatric Distress (HPD) or Low Psychiatric Distress (LPD)*

| Group | Condition | | | |
|---|---|---|---|---|
| | Test: NEG–ALC | Baseline: NEU–ALC | Test: ALC–NEG | Baseline: NEU–NEG |
| Group HPD | | | | |
| *M* | 911 | 962 | 918 | 956 |
| *SD* | 234 | 281 | 252 | 262 |
| Group LPD | | | | |
| *M* | 938 | 911 | 1011 | 936 |
| *SD* | 261 | 278 | 341 | 281 |

*Note.* $n = 18$ per group. NEG = negative affective; ALC = alcohol-related; NEU = neutral.

### Task Performance

In line with conventional procedure in semantic priming studies (Fischler & Goodman, 1978), outlier responses ([RT < 100 ms] or [RT > 3,000 ms]) were analyzed separately from other responses. Analyses of the untrimmed RT distributions were generally congruent with the analyses of the trimmed distributions.[5] Nonwords are semantically meaningless, so that only trials containing word targets were relevant for the assessment of semantic activation. Therefore, data from the word–word and word–nonword trials were analyzed separately. The mean lexical decision RT to word targets preceded by unrelated, neutral primes measured baseline performance. Faster mean RT on test trials (e.g., NEG–ALC) than on baseline trials (e.g., NEU–ALC) indicated activation.

Table 2 reports the mean (*SD*) RT for Groups HPD and LPD in the critical test and baseline conditions. The table shows that in

[5] Inclusion of outliers resulted in a large increase in the variability of the RT distributions. Therefore, nonparametric tests were used to assess the mean effects of group and prime. To perform these nonparametric analyses, a unitary measure of activation (mean difference from baseline RT) was required. To obtain a unitary measure of activation while controlling for shared variance between test and baseline scores (that may reduce the reliability of the difference), we used the regression residual (Webster & Bereiter, 1963). In each prime–target condition, the RT difference score was regressed onto the baseline score, and the residual of each analysis was retained to assess activation. Wilcoxon signed-ranks tests on the residualized difference scores were entirely consistent with the analyses of the trimmed RT distributions, with the exception of the ALC–NEG condition, which failed to demonstrate any effects of group or prime when outliers were included.

Group HPD the mean RT for each test condition was faster than its corresponding baseline condition. In contrast, in Group LPD the mean RT for each test condition was slower than its corresponding baseline condition.

A preliminary 2 (group) × 2 (target) ANOVA assessing RTs on the baseline trials (NEU–ALC, NEU–NEG) obtained no significant effects ($ps > .40$). Therefore, group differences in baseline RT did not account for any group differences in change from baseline RT (i.e., activation).

A 2 (group) × 2 (prime) ANOVA comparing the RT to ALC targets preceded by NEG versus NEU primes in Groups HPD and LPD obtained a significant Group × Prime interaction, $F(1, 34) = 4.22$, $p < .05$, and no other significant effects ($ps > .50$). Mean RT was significantly faster on test (NEG–ALC) than on baseline (NEU–ALC) trials in Group HPD, $t(34) = 3.85$, $p < .001$, and tended to be slower on test than on baseline trials in Group LPD, although the difference was marginal, $t(34) = -1.96$, $p = .061$. These results indicated that negative affective primes activated alcohol concepts in Group HPD and tended to slow RT or inhibit access to alcohol concepts in Group LPD (see Posner & Snyder, 1975).

A 2 (group) × 2 (prime) ANOVA comparing RT to NEG targets preceded by ALC versus NEU primes in the two groups also obtained a significant Group × Prime interaction, $F(1, 34) = 6.38$, $p < .05$, and no other significant effects ($ps > .40$). Mean RT was significantly faster on test (ALC–NEG) than on baseline (NEU–NEG) trials in Group HPD, $t(34) = 2.38$, $p < .05$, and significantly slower on test than on baseline trials in Group LPD, $t(34) = -4.76$, $p < .001$. These results indicate that alcohol primes activated negative affective concepts in Group HPD but inhibited access to negative affective concepts in Group LPD, although the nonsignificant effects in this condition when outliers were included qualifies this conclusion somewhat (see Footnote 5).

### *Correlates of NEG–ALC Activation*

To ensure that outliers (scores > 2 standard deviations from the sample mean) did not distort the correlational results, we used stem-leaf plots to examine the distribution of scores for each correlated measure. Outliers were excluded from the analyses.

*Overall PD.* Previous research suggested that PD may explain variability in the degree to which negative mood cues evoke alcohol-related thoughts (Litt et al., 1990; McCusker & Brown, 1991). In the present study, the mean difference from baseline RT (NEU–ALC − NEG–ALC) measured activation of alcohol concepts by negative affective cues. The residualized difference, obtained by regressing the difference score onto its corresponding baseline score (Webster & Bereiter, 1963), controlled for shared variance in RT on test trials and baseline trials that could confound the correlation between the difference score and other variables (Cohen & Cohen, 1983). Although residuals facilitate interpretation, they primarily retain ordinal information about cases and are therefore rather conservative in terms of statistical power (Heise, 1975). The GSI of the SCL-90 measured overall PD (Derogatis, 1975). The Pearson correlation between the GSI and the residualized NEG–ALC difference score was significant ($r = .29$, $p < .05$, one-tailed); inspection of the scatterplot verified that this result did not derive from dichotomous sets of scores in the two groups. Thus, higher overall PD correlated with greater activation of alcohol concepts by negative affective cues.

*Affective drinking history.* Classical conditioning theory suggested that a bias to drink in negative affective states may coincide with increased activation of alcohol-related concepts (the UCS) by negative affective cues (the CS; Bradizza et al., 1994; Rescorla, 1992). The ratio of the mean negative affective item rating divided by the mean positive affective item rating on the ADHQ measured the relative frequency of drinking in negative affective and positive affective states. To remove shared variance between the numerator and denominator of this ratio, we regressed the ratio onto its denominator, which yielded an unconfounded unitary measure of bias to drink in negative affective states.

The residualized mean (*SD*) ratio scores were 0.24 (0.45) in Group HPD and −0.24 (0.31) in Group LPD. The mean difference between the groups was significant, $t(34) = 3.72$, $p < .005$, indicating that the groups differed in their bias to drink in negative affective versus positive affective states. Tests comparing the residualized mean ratio to the null ($\mu = 0$) were also significant in Group HPD, $t(17) = 2.27$, $p < .05$, and in Group LPD, $t(17) = -3.26$, $p < .01$. Thus, participants in Group HPD drank significantly more often in negative than in positive affective states, whereas participants in Group LPD drank significantly more often in positive than in negative affective states.

Although the Pearson correlation between the residualized ADHQ ratio and NEG–ALC difference scores was marginal, the rank correlation between these variables was significant ($\tau = .20$, $p < .05$, one-tailed). Inspection of the scatterplot verified that this result did not derive from dichotomous sets of scores in the two groups. The scatterplot also showed that a stronger bias to drink in negative affective states coincided with greater NEG–ALC activation, whereas a stronger bias to drink in positive affective states coincided with negative difference scores, or inhibition of RT on NEG–ALC trials relative to NEU–ALC trials. A comparison of NEG–ALC activation in groups formed by a median split of ADHQ ratio scores obtained no significant effects ($ps > .08$), verifying that differences in NEG–ALC activation as a function of PD did not simply reflect group differences in affective drinking history.

When variables related to a criterion correlate with each other, one variable may mediate the relation between the other variable and the criterion. In the present study, the correlation between the GSI and residualized ADHQ ratio score was significant ($r = .77$, $p < .001$), suggesting that affective drinking history may have mediated the relation between PD and NEG–ALC activation. A series of regression analyses tested this possibility (see Baron & Kenny, 1986). Regression of the residualized NEG–ALC difference score onto the ADHQ ratio score and the GSI yielded nonsignificant regression coefficients for both predictors ($ps > .10$). The coefficient for the simple regression of NEG–ALC difference onto GSI ($b = 35.94$) was comparable with the coefficient for GSI in the multiple regression ($b = 34.01$), and the decline in $R^2$ from the multiple to the simple regression when ADHQ ratio was excluded was small: 0.2%. These findings indicate that affective drinking history did not mediate the relation between PD and NEG–ALC activation and that PD was the better predictor of NEG–ALC activation.

*Confidence to abstain from drinking.* Social learning theory suggests that self-efficacy expectations are specific to the situation in which the relevant response is required (Bandura, 1977). Therefore, if memory activation predicts risk of relapse, a specific association should exist between activation of alcohol concepts by negative affective cues and confidence to avoid drinking in negative affective states. A preliminary 2 (group) × 8 (subscale) MANOVA of SCQ subscale scores assessed confidence (0%–100%) to abstain from drinking in eight types of situations. The analysis yielded no significant multivariate or univariate effects (Bonferroni *ps* > .20). Thus, mean confidence to abstain did not vary between groups or across situations.

To measure relative confidence to avoid drinking in negative versus positive affective states, we divided the mean SCQ rating from the Unpleasant Emotions subscale by the mean rating from the Pleasant Emotions subscale and regressed the ratio onto the denominator (the Pleasant Emotions subscale). The mean (*SD*) SCQ ratio scores were 0.78 (0.68) in Group HPD and 1.30 (1.45) in Group LPD; the corresponding residualized scores were −0.15 (0.64) and 0.15 (1.28), respectively. Low scores on the SCQ reflect less confidence to avoid drinking. Thus, Group HPD reported relatively less confidence to abstain in negative than in positive affective states, whereas Group LPD reported the reverse bias. However, the group difference in the mean residualized ratio was nonsignificant ($p > .30$). Thus, confidence to abstain in negative versus positive affective states did not differ reliably in the two groups.

The Pearson correlation between the residualized SCQ ratio and NEG–ALC difference scores was significant ($r = -.28$, $p = .05$, one-tailed). Inspection of the scatterplot verified that this result did not derive from dichotomous sets of scores in the two groups. Participants who reported relatively less confidence to avoid drinking in negative affective states displayed more activation on NEG–ALC trials, whereas those who reported relatively less confidence to avoid drinking in positive affective states showed more inhibition.

*Alcohol dependence.* Group differences in ADS scores suggested that differential alcohol dependence may have accounted for group differences in NEG–ALC activation. However, neither the Pearson correlation nor the rank correlation between the ADS and the residualized NEG–ALC difference score was significant, *ps* > .07, one-tailed (two-tailed *ps* > .14). Thus, alcohol dependence did not reliably predict NEG–ALC activation. This result is consistent with previous research that found no relation between desire to drink after negative mood induction and level of alcohol dependence in problem drinkers (Litt et al., 1990). In addition, this result verifies that ADS did not mediate the relation between NEG–ALC difference scores and measures of PD (e.g., GSI, SCL-90 subscales), ensuring that group differences in NEG–ALC activation as a function of PD do not simply reflect differences in dependence.

### *Correlates of ALC–NEG Activation*

Previous research suggested several possible correlates of ALC–NEG activation, including overall PD and a personality dimension "akin to trait anxiety" (McCusker & Brown, 1991, p. 911). These investigators also observed greater anxiety following alcohol cue exposure in dependent than in nondependent drinkers, although other research did not (Cooney et al., 1987). The GSI, STAI–trait, and ADS measured overall PD, trait anxiety, and alcohol dependence, respectively. The difference from baseline RT (NEU–NEG − ALC-NEG) measured ALC–NEG activation. The difference scores were residualized, and outliers identified by stem-leaf plots were excluded. None of the Pearson or rank correlations between the residualized ALC–NEG difference score and the other variables were significant (pairwise *ps* > .20).

### *Additional Prime–Target Conditions*

Three additional prime–target conditions were included to confirm the adequacy of the procedure and to explore further the effects of negative affective cues. Table 3 reports the mean (*SD*) RT scores for the two groups in these three test conditions and in their corresponding baseline conditions. Mean RT was faster in each of the three test conditions relative to baseline, and this relation occurred in both groups.

In line with previous research that demonstrated ALC–ALC activation in dependent drinkers (Hill & Paynter, 1992), a 2 (group) × 2 (prime) ANOVA comparing RT to ALC targets preceded by ALC or NEU primes obtained a significant main effect of prime, $F(1, 34) = 6.62$, $p < .05$, and no main effect of group or interaction (*ps* > .20). Thus, the two groups displayed equivalent activation of alcohol concepts by alcohol cues, verify-

Table 3
*Mean and Standard Deviation Lexical Decision Response Time (in Milliseconds) on Additional Test and Baseline Trials in Problem Drinkers With High Psychiatric Distress (HPD) or Low Psychiatric Distress (LPD)*

| | Condition | | | | | |
|---|---|---|---|---|---|---|
| Group | Test: ALC–ALC | Baseline: NEU–ALC | Test: NEG–NEG | Baseline: NEU–NEG | Test: CAT–CAT-m | Baseline: CAT–CAT-u |
| Group HPD | | | | | | |
| *M* | 897 | 962 | 940 | 956 | 817 | 865 |
| *SD* | 257 | 281 | 281 | 262 | 212 | 216 |
| Group LPD | | | | | | |
| *M* | 888 | 911 | 933 | 936 | 845 | 873 |
| *SD* | 290 | 278 | 292 | 281 | 232 | 274 |

*Note.* *n* = 18 per group. NEG = negative affective; ALC = alcohol-related; NEU = neutral; CAT-m = neutral, categorical, matched; CAT-u = neutral, categorical, unmatched.

ing that the task could detect activation of the alcohol network in both groups.

Research with anxious nonproblem drinkers (Richards & French, 1992) observed activation of the threatening meaning as opposed to the neutral meaning of homographic targets (e.g., *tumor-growth* faster than *plant-growth*). This result implied that negative affective primes might activate negative affective targets in the present study. However, a 2 (group) × 2 (prime) ANOVA comparing RT to NEG targets preceded by NEG or NEU primes obtained no significant effects ($ps > .60$). Therefore, negative affective cues did not reliably activate negative affective concepts in either group.

Considerable research shows that prime words that are semantically related to their targets lead to greater activation than do primes that are unrelated to their targets (Neely, 1991). Accordingly, a 2 (group) × 2 (prime) ANOVA comparing RT to neutral, categorized targets preceded by primes from the same category (CAT-matched) or a different category (CAT-unmatched) obtained a significant main effect of prime, $F(1, 34) = 4.86, p < .05$, and no main effect of group or interaction ($ps > .50$). These findings were consistent with previous research and showed that the task could detect activation because of semantic relatedness. They further ensured that group differences in activation in the other conditions did not derive from differences in the groups' ability to detect a conceptual relation between prime and target stimuli.

A 2 (group) × 4 (prime) ANOVA assessed RT to nonword targets preceded by NEG, ALC, NEU, and CAT word primes. The analysis obtained no significant effects ($ps > .40$). Thus, there were no differences in RT as a function of group or prime when targets were semantically meaningless. This ensured that faster RT to word targets following specific types of primes did not derive from a general facilitatory effect of the prime (e.g., due to vigilance) irrespective of the target, but rather reflected a specific relation between the prime and the target.

ANOVAs comparing the frequency of errors (i.e., reporting a word when the target was a nonword or vice versa) and the frequency of outliers ([RT < 100 ms] or [RT > 3,000 ms]) in each group and condition found no effects involving group.[6] Therefore, the ANOVAs of RT scores were not biased by differences in the number of correct responses that comprised the mean RT score in each group. In the sample as a whole, there were no differences in RT in any condition due to gender ($ps > .06$). In Group HPD, order of onset of drinking problems and PD was unrelated to RT in any condition ($ps > .60$).

## Discussion

The correlation between drinking in negative affective states and increasing alcohol dependence (Cunningham et al., 1995) suggested that negative affective cues may be functionally related to alcohol use in problem drinkers, whereas positive cues may be related to alcohol use in social drinkers. Activation of alcohol concepts by positive affective cues in social drinkers (Weingardt et al., 1996) led to the prediction that negative affective cues would activate alcohol concepts in problem drinkers. The variable effects of negative mood induction on desire to drink in dependent drinkers (Litt et al., 1990) suggested that some factors apart from dependence moderated the effects of negative affective cues on alcohol-related thoughts. The correspondence between drinking in negative affective states and negative temperament in problem drinkers (Cannon et al., 1990, 1992) implied that PD could be one such factor. The correspondence between Neurotic–Introversion and increased anxiety following alcohol cue exposure in problem drinkers (McCusker & Brown, 1991) further implied that PD might moderate the effects of alcohol-related cues on negative affective thoughts. Together, these findings led to the prediction that negative affective cues would activate alcohol concepts to a greater degree in problem drinkers with high PD than in problem drinkers with low PD, and that alcohol cues would likewise activate negative affective concepts to a greater degree in high PD than in low PD problem drinkers.

Consistent with these hypotheses, Group HPD made faster lexical decisions to alcohol target words preceded by negative affective rather than neutral prime words, and the degree of activation exceeded that of Group LPD. The low PD drinkers not only displayed significantly less activation than the high PD drinkers but also tended to display inhibition of alcohol targets by negative affective primes. A parallel pattern of results was observed in the ALC–NEG condition, indicating that alcohol cues activated negative affective concepts to a greater degree in high PD than in low PD drinkers.

Implicit activation of alcohol concepts by negative affective cues in Group HPD is consistent with previous research that tested the effects of negative mood induction on desire to drink in problem drinkers (Litt et al., 1990). Activation of negative affective concepts by alcohol cues in Group HPD is also consistent with previous cue exposure research (McCusker & Brown, 1991). Because the present study used an implicit procedure that controls for self-perceptions and experimental demand, the present results support the validity of the in vivo cue exposure studies. The lack of correlation between ADS scores and NEG–ALC or ALC–NEG

[6] A 2 (group) × 8 (condition) ANOVA compared the proportion of errors on word–word trials in Groups HPD and LPD in the eight prime–target conditions. The analysis obtained a significant main effect of condition, $F(7, 238) = 6.39, p < .001$, but no group effect or interaction ($ps > .07$). The highest proportion of errors occurred on NEU–ALC and NEG–ALC trials ($M = .08$), the smallest proportion of errors occurred on the CAT trials ($M = .03$), and an intermediate proportion of errors occurred in the other conditions ($M = .05$). Thus, on average, participants responded incorrectly on 0.6 to 1.6 of the 20 trials in each condition. A 2 (group) × 4 (prime) ANOVA comparing the proportion of errors for each group and type of prime on word–nonword trials obtained no significant effects ($ps > .20$). The mean proportion of errors for the entire sample on nonword trials was .06.

A 2 (group) × 8 (condition) ANOVA assessed the proportion of outliers ([RT < 100 ms] or [RT > 3000 ms]) on word–word trials. The analysis obtained a significant main effect of condition, $F(7, 238) = 2.14, p < .05$, but no group effect or interaction ($ps > .70$). Inspection of the RT scores indicated that outlier trials typically were exceedingly long rather than exceedingly short. Post hoc analyses determined that there were significantly fewer outliers on CAT–CAT-unmatched trials ($M = .01$) than on CAT–CAT-matched trials ($M = .02$), $p < .05$, and significantly fewer outliers on CAT–CAT-matched, CAT–CAT-unmatched, and NEG–NEG trials ($M = .02$) than in the other conditions ($M = .03$), $p < .05$. A 2 (group) × 4 (prime) ANOVA comparing the proportion of outliers in the two groups on the nonword target trials obtained no significant effects ($ps > .08$). The mean proportion of outliers on these trials was .05.

difference scores ensured that group differences in alcohol dependence did not mediate the group difference in activation in either condition.

Although NEG–ALC and ALC–NEG activation scores each distinguished between the groups, only NEG–ALC scores correlated with GSI. The lack of correlation between ALC–NEG and STAI–trait scores further shows that this condition did not reliably tap trait anxiety. In addition, the decline in between-groups effect size for NEG–ALC activation when the supplemental psychiatric scales were entered as covariates was 50% greater than the decline in ALC–NEG activation produced by these covariates. These findings indicate that NEG–ALC activation better distinguishes between problem drinkers as a function of PD.

The data from the ADHQ showed that high PD participants tended to drink more often in negative than in positive affective states, whereas low PD participants tended to drink more often in positive than in negative affective states. The correlational data further showed that a bias to drink in negative affective states predicted greater NEG–ALC activation, whereas a bias to drink in positive affective states predicted more inhibition. The differential activating effects of negative affective cues on alcohol-related concepts in the two groups are consistent with previous suggestions that negative mood cues may have different effects on problem drinkers who report positive mood as their primary drinking "trigger" (Rubonis et al., 1994).

The correlation between the tendency to drink in negative affective states and NEG–ALC activation suggests that conditioned associations between negative affect and alcohol may contribute to cue-induced activation of memory. This is consistent with neural network theories of drug use which propose that "the threshold of activation of the network is reduced, and the network becomes more articulated and extensive, as a function of associative and nonassociative learning produced by iterative drug exposures" (Baker et al., 1987, p. 304). However, the regression analyses showed that drinking history did not mediate the effects of PD on NEG–ALC activation. Therefore, classical conditioning alone did not account for the differential effects of negative affective cues on alcohol cognitions in high PD and low PD drinkers.

The data from the SCQ showed that the groups did not differ significantly in their relative confidence to avoid drinking in negative as opposed to positive affective states, although the mean difference between the groups was sizable. The correlational data from the SCQ indicated that greater activation on NEG–ALC trials predicted lower confidence to abstain in negative affective states, whereas greater NEG–ALC inhibition predicted lower confidence to abstain in positive affective states. In light of the correspondence among low self-efficacy, situational negative affect, and relapse to alcohol use (Marlatt & Gordon, 1980), the present findings suggest that implicit activation of alcohol concepts by negative affective cues may explain some of the variance in problem drinkers' risk of relapse in negative affective states. However, the causal effects of implicit activation remain unknown.

The occurrence of NEG–ALC and ALC–NEG activation in Group HPD suggests that negative affective and alcohol-related concepts are associated in memory in problem drinkers with high PD. The lack of activation in these conditions in Group LPD suggests that negative affective and alcohol-related concepts are not reliably associated in memory in problem drinkers with low PD. The significant inhibition of RT on ALC–NEG trials in Group LPD further suggests that alcohol cues activated concepts incompatible with negative affect in these individuals. This suggestion is consistent with research that found inhibition of RT to targets preceded by conceptually incompatible primes in healthy volunteers (Dagenbach, Carr, & Barnhardt, 1990). It is also consistent with Baker et al.'s (1987) network theory of drug use that posited "two distinct, incompatible (mutually inhibitory) urge networks" (p. 304), one that subserves urges evoked by positive affect and one that subserves urges evoked by negative affect.

Although an associative explanation for implicit activation is intuitively appealing, the precise mechanism of this phenomenon remains controversial. Some investigators have argued that faster lexical decision RT reflects "spreading activation" among concepts connected to the prime concept by means of a semantic memory network (e.g., Collins & Loftus, 1975; McNamara, 1992). Others have proposed that faster lexical decision RT derives from the inherent similarity between the particular stimulus complex (i.e., a compound cue comprising a specific prime–target pair) and the corresponding memory structure (Ratcliff & McKoon, 1988). A third possibility is that faster lexical decision RT derives from concurrent forward-acting and backward-acting processes (Koriat, 1981; Neely, 1991). In forward-acting processes, activation flows from the prime to the target; in backward-acting processes, activation flows from the target to the prime. Forward-acting processes reduce RT by rendering a target concept more accessible before the target stimulus has been presented, signifying a priori associations between prime and target concepts (Koriat, 1981). In contrast, backward-acting processes can only occur once the target stimulus has been presented, possibly reflecting post hoc associations between prime and target stimuli (Koriat, 1981).

The relative contribution of forward-acting and backward-acting processes to lexical decisions depends on several variables (e.g., ratio of word–nonword target trials, interval between the prime and target stimuli). Neely, Keefe, and Ross (1989) argued that "to account for the complete array of activation phenomena, one must appeal to the operation of at least three experimentally isolable mechanisms: automatic spreading activation, expectancies, and postlexical semantic matching" (p. 1018). Because the last of these processes can only occur when the proportion of word and nonword target trials differs (Neely, 1991), which was not the case in the present study, this rules out a semantic matching explanation for our results. The first two mechanisms both entail forward-acting processes, so that facilitation of RT owing to either mechanism implies an a priori association between prime and target concepts. Inhibition of RT further suggests the activation by the prime cue of concepts that are incompatible with the target stimuli, and which interfere with its appraisal (Becker, 1980; Dagenbach et al., 1990). The inhibition of RT in Group LPD thus provides indirect support for a forward-acting process in these conditions. Together, the evidence, although not indisputable (Ratcliff & McKoon, 1988), supports the conclusion that NEG–ALC and ALC–NEG activation each resulted from a preexisting association between negative affective and alcohol-related concepts in memory.

Of the two forward-acting processes just mentioned, expectancies are more likely to operate when the time between the onset of the prime and the onset of the target exceeds 200 ms (de Groot, 1984; Neely, 1977). In the present study, this interval was 750 ms.

allowing more than enough time for a coherent expectancy to develop. Evidence of a classically conditioned relation between negative affect and alcohol use would also imply the operation of expectancies (Bolles, 1972). The correlation between NEG–ALC difference scores and ADHQ ratio scores suggests that classical conditioning may have contributed to NEG–ALC activation, and this in turn supports an expectancy explanation. An expectancy account of NEG–ALC activation is consistent with the wealth of evidence demonstrating the role of expectancies in alcohol-related cognition in social drinkers (e.g., Rather & Goldman, 1994; Stacy, 1997). Implicit activation of drug and alcohol-related concepts has also been shown to predict use of these substances in adolescents (Stacy, Ames, Sussman, & Dent, 1996).

ALC–NEG activation and inhibition may also reflect the operation of expectancies. Heightened expectations of alcohol reinforcement have been reported by heterogeneous problem drinkers following in vivo cue exposure (Cooney, Baker, Pomerleau, & Josephy, 1984; Cooney et al., 1987). These findings are consistent with the notion that alcohol cues activated positive affective rather than negative affective concepts in Group LPD, leading to ALC–NEG inhibition in this group. Other research has found that an acute dose of alcohol can sometimes lead to increased anxiety, and that chronic heavy drinking can induce anxiety symptoms in vulnerable individuals when they are sober (Freed, 1978; George, Nutt, Dwyer, & Linnoila, 1990). These findings suggest that alcohol cues may have recruited expectations of increased anxiety in Group HPD, leading to ALC–NEG activation in this group.

The lack of correlation between ALC–NEG activation and measures of PD or alcohol dependence indicates that some other factors contributed to the group difference in this condition. The investigators who observed differences in anxiety following alcohol cues as a function of Neurotic–Introversion noted that this personality profile has been associated with *conditionability* (McCusker & Brown, 1991)—an increased susceptibility to negative affect, to cues predicting punishment, and to autonomic arousal in response to punishment (Eysenck & Eysenck, 1985; Gray, 1981; Zinbarg & Revelle, 1989). Drinkers high in conditionability may perceive the negative affective consequences of alcohol to be highly salient and may therefore acquire more distinct expectancies about these consequences. Such expectancies, in turn, may augment activation of negative affective concepts and attenuate activation of positive affective concepts by alcohol cues in highly conditionable drinkers; no such effects would be expected in drinkers low in conditionability. Direct assessment of conditionability with the EPI and of alcohol outcome expectancies would enable an empirical test of the relation between these variables and ALC–NEG activation.

The occurrence of ALC–ALC activation in each of our groups is consistent with previous research showing that within-category activation of alcohol concepts distinguished between dependent and nondependent drinkers (Hill & Paynter, 1992). The lack of other effects in this condition ensures that group differences in NEG–ALC activation do not reflect differences in the ability to activate the alcohol network. The occurrence of CAT–CAT activation in each group ensured that the absence of activation in other conditions was not due to inadequacies in the procedure or to functional deficits in the sample. The lack of other effects in this condition ensured that group differences in NEG–ALC and ALC–NEG activation did not derive from group differences in the ability to perceive a conceptual relation between lexical stimuli.

The results of this study provide support for the value of implicit paradigms in the assessment of problem drinkers. However, the study also has limitations. The lack of NEG–NEG activation was not consistent with previous research that observed activation of the threatening meanings of ambiguous words in anxious individuals (Richards & French, 1992). That study found that primes associated with the threatening meaning of a homographic target yielded greater activation. In contrast, the present study tested a general relation between heterogeneous negative affective concepts. This more general prime–target relation may not have been discernible in the present sample. However, the apparent lack of previous research showing such within-category activation means that the lack of NEG–NEG activation in the present study does not contravene existing evidence, and therefore does not compromise the validity of our other findings. Future research on implicit activation in problem drinkers should include a comparison group of participants with high PD but no alcohol problem to determine the generality of the present findings. Inclusion of a healthy control group would further confirm that activation of specific concepts by negative affective cues does not occur in the absence of psychopathology.

The present study shows that implicit cognition is a useful way to assess memory associations between affective and alcohol-related concepts while controlling for potential biases due to self-perceptions or experimental demand. Whether implicit cognition has a causal effect on drinking behavior is a matter for future investigation. However, the research that demonstrated increased consumption of placebo beer in social drinkers exposed to positive affective words in a Stroop task supports an inference of causality (Roehrich & Goldman, 1995). That result extends the evidence from other branches of psychology showing that implicit activation can bias decision making as well as psychomotor behavior in healthy volunteers (Fazio & Williams, 1986; McClelland & Rumelhart, 1985). Together, the findings suggest that activation of memory-based, alcohol-related concepts not only may reflect but also may promote drinking behavior.

Although the activation effects measured in this study were of very brief duration, other research has shown that activation can persist over long periods of time and across situations (e.g., Kolers, 1975). Such residual activation may help to explain why cues for alcohol need not be physically present to evoke cravings and drinking behavior (Mathew, Claghorn, & Largen, 1979). Residual activation may be particularly important with respect to relapse: A problem drinker may successfully abstain from drinking while in a high-risk situation (e.g., negative affect) but remain at heightened risk of drinking after the episode because of continued activation of alcohol concepts in memory. This cognitive "sleeper effect" may be part of the process outlined by Sanchez-Craig and her colleagues (1987; cited in Miller & Brown, 1991, p. 28), who suggested that relapse is likely to occur "once the effortful memory system (employed deliberately to effect short-term changes in behaviour) is relaxed, and behaviour is allowed to be governed by more automatic processes." Future research could apply the Stroop prime/taste-test procedure (Roehrich & Goldman, 1995) to examine the causal effects of implicit activation on drinking in problem drinkers with different levels of PD.

Group differences in cue-induced memory activation indicate a functional difference between high and low PD problem drinkers that does not derive from self-perceptions or experimental demand. This difference may have clinical implications. For example, previous research has shown that in vivo exposure to alcohol cues coupled with prevention of a drinking response can reduce alcohol use in problem drinkers (Laberg & Ellersten, 1987). However, subsequent investigators proposed that individual differences in cue reactivity may influence the effectiveness of this procedure (Litt et al., 1990; McCusker & Brown, 1991; Rohsenow et al., 1994). Taken together with these findings, the present results imply that treatments designed to extinguish conditioned alcohol-related expectancies may be especially helpful for improving treatment outcome in distressed problem drinkers.

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