

# Normal Performance on a Simulated Gambling Task in Treatment-Naïve Alcohol-Dependent Individuals

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**Background:** Research suggests that substance abusers make more disadvantageous decisions on the simulated gambling task (SGT); such decisions are associated with deviance proneness and antisocial symptoms. This study examines decision making on the SGT in young adults with alcohol dependence who are treatment-naïve (TxN).

**Methods:** A total of 116 subjects (58 controls, 58 TxNs) were tested on the SGT, where participants choose cards from 4 different decks that vary in terms of the magnitude of the immediate gain (large/small) and the magnitude of long-term loss (larger/smaller). Participants were also assessed on measures of externalizing symptoms, personality traits reflecting social deviance, neuropsychological function, and the density of the family history of alcoholism.

**Results:** Treatment-naïves did not differ from controls on measures of SGT decision making. Simulated gambling task performance was not associated with externalizing symptoms, social deviance proneness, or a familial density of alcoholism. Although TxNs had higher levels of externalizing symptoms, social deviance, and familial density of alcoholism compared with controls, these variables were only modestly elevated compared with previous samples of long-term abstinent alcohol-dependent individuals who showed decision-making deficits on the SGT.

**Conclusions:** The results suggest that our sample of young adult TxN adults with alcohol dependence do not have global deficits in decision making as measured by the SGT, and that their poor decisions regarding their alcohol consumption are more specific to drinking.

**Key Words:** Simulated Gambling Task, Alcoholism, Cognition, Decision Making.

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**A**LCOHOLISM AND DRUG abuse are disorders in which people continue their use of harmful substances despite major negative consequences. Bechara et al. (1994) developed a simulated gambling task (SGT) that has been used in studies (Bechara et al., 2001; Grant et al., 2000) to examine the mechanisms that underlie the skewed decision-making style of substance abusers. The gambling task imitates real-life decision making in that it requires an individual to weigh reward versus punishment in an atmosphere of uncertain outcomes. Subjects are asked to choose between decks of cards that have small positive gains associated with relatively small long-term negative consequences (the good decks) and decks of cards with large positive gains associated with large long-term negative consequences (the bad decks). Over the long run, choices from the good decks result in winning money, while choices from the bad decks result in losing money. We all continually weigh the short-

term advantages against the long-term consequences of our behavior, but a hallmark of drug and alcohol abuse is persistence in a behavior (drinking) that returns short-term benefit (intoxication), but often leads to significant long-term negative consequences.

The SGT was initially developed to study patients with acquired sociopathy due to damage to the ventromedial prefrontal cortex in an effort to model the global decision-making deficits that presumably contribute to the enduring pattern of disinhibited behavior observed in this population (Bechara et al., 1994; Bechara et al., 1997). Such patients often take part in risky behaviors that are immediately gratifying while ignoring negative future outcomes. It is thought that they cannot see beyond the short-term rewards of their behavior to its potential long-term consequences (Bechara et al., 1994). Compared with controls, when playing the SGT, patients with ventromedial prefrontal lesions consistently chose to draw more cards from decks with larger immediate rewards and larger long-term net losses than from decks with smaller immediate rewards, smaller delayed punishments, and long-term net gains (Bechara et al., 1994, 1997). Although they performed poorly on the gambling task, such patients performed normally in other cognitive domains (Bechara et al., 1998). The cognitive domains assessed by Bechara and colleagues were verbal and performance intelligence quotient from the Wechsler Adult

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Intelligence Scale—Revised, memory and attention/concentration as measured by the Wechsler Memory Scale, visual retention measured by the Benton Visual Retention Test, and verbal ability from the COWAT. Additional tests conducted were the Wisconsin Card Sorting Task, the Facial Recognition Task, and the Judgment of Line Orientation Task.

Ventromedial prefrontal dysfunction may predispose an individual to make disadvantageous personal choices possibly leading to socially inappropriate or socially deviant behavior (Bechara et al., 1994, 1997) or to drink excessively even when it leads to significant problems. As noted above, the SGT was initially developed to study patients with acquired sociopathy due to damage to the ventromedial prefrontal cortex. Since that time, Bechara and colleagues have extended their work to show that patients with amygdala damage also show SGT impairments (Bar-On et al., 2003; Bechara et al., 1999).

A number of factors may contribute to the poor decisions that alcoholic individuals make in regard to their drinking, such as global problems in decision making associated with neuropsychological deficits that predate, or are a consequence of, their alcoholism, specific contextual factors (alcohol cues), and past learning specifically associated with drinking environments (such as conditioned responses), or peer pressure (Fein et al., 2004; Finn and Hall, 2004; Finn et al., 2002). Bechara et al.'s (1994) SGT has been administered to substance abusers by a number of researchers to investigate evidence of global problems in decision making. Studies show that on the SGT, alcoholic individuals (Bechara and Damasio, 2002; Bechara et al., 2001; Mazas et al., 2000), and drug abusers (Grant et al., 2000; Petry et al., 1998) have a pattern of disadvantageous decision making similar to that of ventromedial prefrontal lesion patients, characterized by favoring larger immediate rewards while disregarding long-term negative consequences. Clark and Robbins (2002) note the centrality of abnormal decision making to addictive behavior and hypothesize that this association between addiction and impaired decision making underlies performance deficits on the gambling task in substance abusers.

Evidence also suggests that these decision-making deficits are associated with diminished behavioral control, social deviance, and antisocial traits (Fein et al., 2004; Stout et al., 2005), known to predate and predict the development of alcoholism in prospective studies (Caspi et al., 1996; Chassin et al., 1999; Pulkkinen and Pitkanen, 1994). In fact, a number of studies suggest that global decision-making deficits assessed with laboratory tasks are observed only in those alcoholic individuals who have a history of antisocial behavior (Finn et al., 2002; Mazas et al., 2000; Petry, 2002). Such deficits either are not present (Finn et al., 2002) or are much milder (Mazas et al., 2000; Petry, 2002), in nonantisocial alcoholic individuals. The covariance of antisocial traits with decision-making deficits on laboratory tasks is consistent with

the hypothesis that global decision-making deficits may predispose to alcoholism, although it also may be that long-term alcohol abuse might contribute to antisociality and global decision-making deficits.

In a recent paper on long-term abstinent alcoholic individuals (abstinence duration ranging from 6 months to 13 years, with a mean abstinent duration of 6.7 years), we demonstrated that abstinent alcoholic individuals were impaired on the SGT compared with age-matched controls (Fein et al., 2004) and that impaired decision-making was associated with low levels of socialization. In a second paper (Harper et al., 2005), we showed that the same group of long-term-abstinent alcoholic individuals had reduced gray matter in the region of the amygdala, an area implicated in impaired gambling task performance (Bar-On et al., 2003; Bechara et al., 1999). Although we believe that these results partly reflect the consequences of long-term alcohol abuse, we could not rule out the possibility that the results reflected a factor associated with the genetic vulnerability to alcoholism and was in fact an antecedent rather than a consequence of alcoholism. In a third paper (Fein and Landman, 2005), we demonstrated that treated and treatment-naïve (TxN) individuals come from different populations in regard to their alcohol use. In that paper, we rejected the hypothesis that the TxN alcoholics and the treated alcoholics have similar alcohol use trajectories over time, with the TxN sample simply being observed earlier in their alcohol use histories. Instead we concluded that the two groups come from different populations in regard to their alcohol use in the early years of abusive drinking (in fact, the treated alcoholic individuals had alcohol doses over 50% higher than TxN alcoholic individuals in the years just after they began drinking heavily). This suggests that results from studies of alcoholic individuals in treatment or posttreatment (i.e., most studies of alcoholic individuals) cannot be generalized to untreated individuals (who comprise most of alcoholic individuals). If this holds true, one would not expect to see the same degree of decision-making impairments in TxN samples as were observed in the long-term-abstinent alcohol-dependent sample.

The current study was designed to help address these issues. Previous studies have used either active alcoholic individuals (Mazas et al., 2000), drug addicts (Grant et al., 2000), alcoholic individuals just before treatment (Bechara, 2001; Bechara and Damasio, 2002), or substance abusers in treatment (Petry et al., 1998). In the current study, we examine performance on the SGT in TxN alcohol-dependent individuals compared with age-matched and gender-matched controls. We also examine the association of SGT performance with socially deviant, antisocial personality traits; in various cognitive domains, and with alcohol use variables. Further analyses were performed comparing the TxN group and the long-term-abstinent alcohol-dependent sample from our previous paper (Fein et al., 2004).

## MATERIALS AND METHODS

### Participants

The TxN alcohol-dependent group and the control group were age- and gender-matched, each consisting of 24 women and 34 men. All participants were recruited from the community by café postings, newspaper advertisements, and a local Internet site. The inclusion criterion for the control group was a lifetime drinking average of less than 30 drinks per month, with no periods of more than 60 drinks per month. Treatment-naïve participants needed to meet DSM-IV (American Psychiatric Association, 2000) criteria for current alcohol dependence. Exclusion criteria for both groups were as follows: (1) lifetime or current diagnosis of schizophrenia or schizophreniform disorder; (2) history of drug (other than nicotine or caffeine) dependence or abuse; (3) significant history of head trauma or cranial surgery; (4) history of diabetes, stroke, or hypertension that required medical intervention or of other significant neurological disease; or (5) clinical evidence of the Wernicke–Korsakoff syndrome. All participants were informed of the study's procedures and aims and signed a consent form before their participation. Subjects participated in four sessions that lasted from 1.5 to 3 hours, which included clinical, neuropsychological, electrophysiological, and neuroimaging assessments. All participants were asked to abstain from using alcohol for at least 24 hours before any lab visits, and a Breathalyzer test was administered to all subjects before each session. We had no positive Breathalyzer test results. Subjects who completed testing were paid for time and travel, and those who completed the entire study were also given a completion bonus.

### Procedure

All participants were assessed using a computerized psychiatric Diagnostic Interview Schedule (Robins et al., 1998) (c-DIS). Participants were also interviewed on their drug and alcohol use using the lifetime drinking history methodology (Skinner and Sheu, 1982; Sobell and Sobell, 1990; Sobell et al., 1988), completed questionnaires assessing social deviance proneness, and family density of alcoholism and were administered the SGT along with a neuropsychological assessment. Medical histories were also reviewed and liver functions were tested.

### Assessments

*Personality and Externalizing Symptoms.* The presence of personality traits indicative of deviance proneness and externalizing disorders were assessed using the Psychopathic Deviance Scale of the MMPI-2 (MMPI\_Pd) (Hathaway, 1989), the Socialization Scale of the California Psychological Inventory (CPI\_So) (Gough, 1969), and the sum of positive responses to the antisocial personality and conduct disorder questions on the c-DIS (externalizing symptoms).

*Alcohol Use Variables.* Based upon subject's responses on the lifetime drinking history, alcohol use variables were defined. Alcohol lifetime duration refers to the number of months of alcohol consumption in the individual's lifetime, while peak duration refers to the number of months of peak alcohol use. Alcohol lifetime dose is the average number of drinks per month of alcohol consumption over the subject's lifetime, while peak dose is the number of drinks per month during their period of peak alcohol consumption. Age and level at first heavy use were also included as alcohol use variables.

*Familial Drinking Density.* The Family Drinking Questionnaire (Mann et al., 1985; Stoltenberg et al., 1998) was administered in the first session to assess the density of problem drinkers in the participant's family. Participants were asked to rate the members of their family as being alcohol abstainers, alcohol users with no problem or problem drinkers. Family Drinking Density 1 (FamDD\_1) was defined as the total number of problem drinking first-degree rela-

tives divided by the total number of first degree relatives. FamDD\_2 is the same as FamDD\_1, except it addresses the proportion of second degree relatives.

*Neuropsychological Measures.* A neuropsychological assessment was also administered, covering the domains of attention, auditory working memory, verbal skills, abstraction/cognitive flexibility/executive functioning, immediate and delayed memory, motor and psychomotor skills, reaction time, and spatial processing. The tests used to assess each domain were as follows: (1) attention [Stroop Color (Golden, 1975), Micro-Cog (MC) Numbers Forward, MC Numbers Reversed, MC alphabet, MC Word List 1 (Powell et al., 1993)]; (2) verbal fluency [COWAT (Benton and Hamsher, 1983), AMNART (Grober and Sliwinski, 1991)]; (3) abstraction/cognitive flexibility [Short Categories (Wetzel, 1982), Stroop interference score (Golden, 1975), Trail Making Test B (Reitan and Wolfson, 1985), MC Analogies (Powell et al., 1993), MC Object Match A and B (Powell et al., 1993)]; (4) psychomotor [Trails A (Reitan and Wolfson, 1985), Symbol Digit (Smith, 1968)]; (5) immediate memory [MC Story immediate 1 and 2 (Powell et al., 1993), Rey–Osterrieth Complex Figure test—immediate recall (Osterrieth, 1944), MC Word List 2 (Powell et al., 1993)]; (6) delayed memory [MC Story Delay 1 and 2 (Powell et al., 1993), MC Address delay (Powell et al., 1993), Rey–Osterrieth Complex Figure test—delayed recall (Osterrieth, 1944)]; (7) motor skills [Grooved Pegboard (Klove, 1963)]; (8) reaction time [MC Timers 1 and 2 (Powell et al., 1993)]; (9) spatial processing [MC Tic Tac 1 and 2, MC Clocks (Powell et al., 1993), Block Design (Wechsler, 1981)]; and (10) auditory working memory [PASAT at delays of 2.4, 2.0, 1.6, and 1.2 seconds (Gronwall, 1977)].

*SGT Administration.* The game begins with \$2000 of “fake” money. The participants' task is to try to win as much money as possible. They are asked to select 1 card at a time from 1 of the 4 decks shown. Some cards will add money and some cards will subtract money from their running total. Participants are told that they can switch from 1 deck to another at any time and as often as they wish. Each deck has a total of 60 cards, with the game ending after a total of 100 cards are selected. They are told that they will not know when the game will end, that they should continue to choose from whichever deck(s) they prefer until the game ends, that some decks are better than others, and that the computer does not change the order of cards after the game starts, make you lose at random, or make you lose money based on the last card chosen. The dependent variable for the gambling task is a measure of advantageous decision bias indexed as the number of cards chosen from the “good” decks minus the number of cards chosen from the “bad” decks.

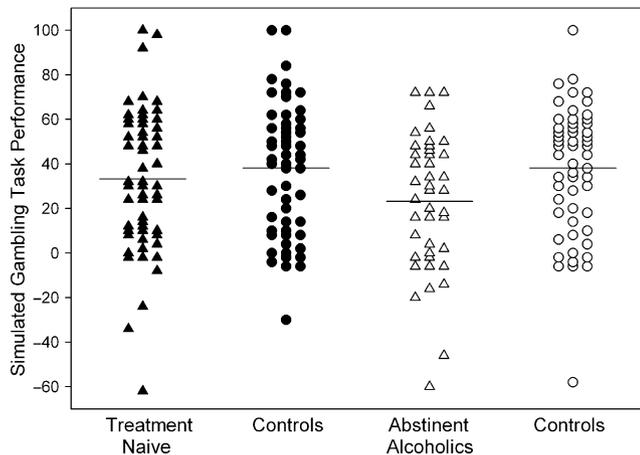
### Statistical Analysis

The data were analyzed using SPSS (SPSS Inc., 2004). Analysis of variance for unbalanced designs was carried out using the General Linear Models procedure. Pearson correlations were used to examine associations.

## RESULTS

### SGT Performance

Treatment-naïve individuals and controls did not differ in their performance on the simulated gambling task ( $F_{1,112} = 1.04$ ,  $p = 0.31$ ). Additionally, there were no gender ( $F_{1,112} = 0.002$ ,  $p = 0.96$ ) or group by gender interactions ( $F_{1,112} = 0.785$ ,  $p = 0.38$ ). Figure 1 presents the raw gambling game scores for the TxN sample and the controls used in this analysis, as well as for the abstinent alcoholic group and controls from our previous study.



**Fig. 1.** Scatter plots of the raw simulated gambling task (SGT) scores (number of cards drawn from the “good” decks minus the number of cards drawn from the “bad” decks) for the treatment naïve (TxN) group (▲), the TxNs controls (●), the abstinent alcoholic persons (△), and the abstinent alcoholic’s controls (○). The horizontal lines represent the mean for each of the groups. There were no group differences in SGT performance between the TxN sample and either their controls or the abstinent alcoholic sample.

#### *Alcohol Use, Family Drinking Density Measures, and SGT Performance*

No significant correlations were observed within the TxN sample between alcohol use variables and performance on the simulated gambling task. The TxN sample compared with controls showed a greater percentage of problem drinking second degree relatives ( $F_{1,112} = 7.93$ ,  $p < 0.01$ ) and a trend toward a greater percentage of problem drinking first degree relatives ( $F_{1,112} = 2.97$ ,  $p = 0.09$ ). However, when examined as a covariate, neither of these family density measures was associated with gambling task performance, nor were there any interactions of either of these covariates with group, gender, or group by gender.

#### *Externalizing Symptoms, Personality Measures, and SGT Performance*

The TxN group had more externalizing symptoms ( $F_{1,112} = 11.89$ ,  $p = 0.001$ ) than the control group. The TxN group also had lower scores on the CPI\_So scale ( $F_{1,112} = 38.13$ ,  $p < 0.001$ ) and higher scores on the MMPI\_Pd scale ( $F_{1,112} = 13.56$ ,  $p < 0.001$ ), both indicating greater deviance proneness. No significant correlations were observed between gambling task performance and any of the above measures.

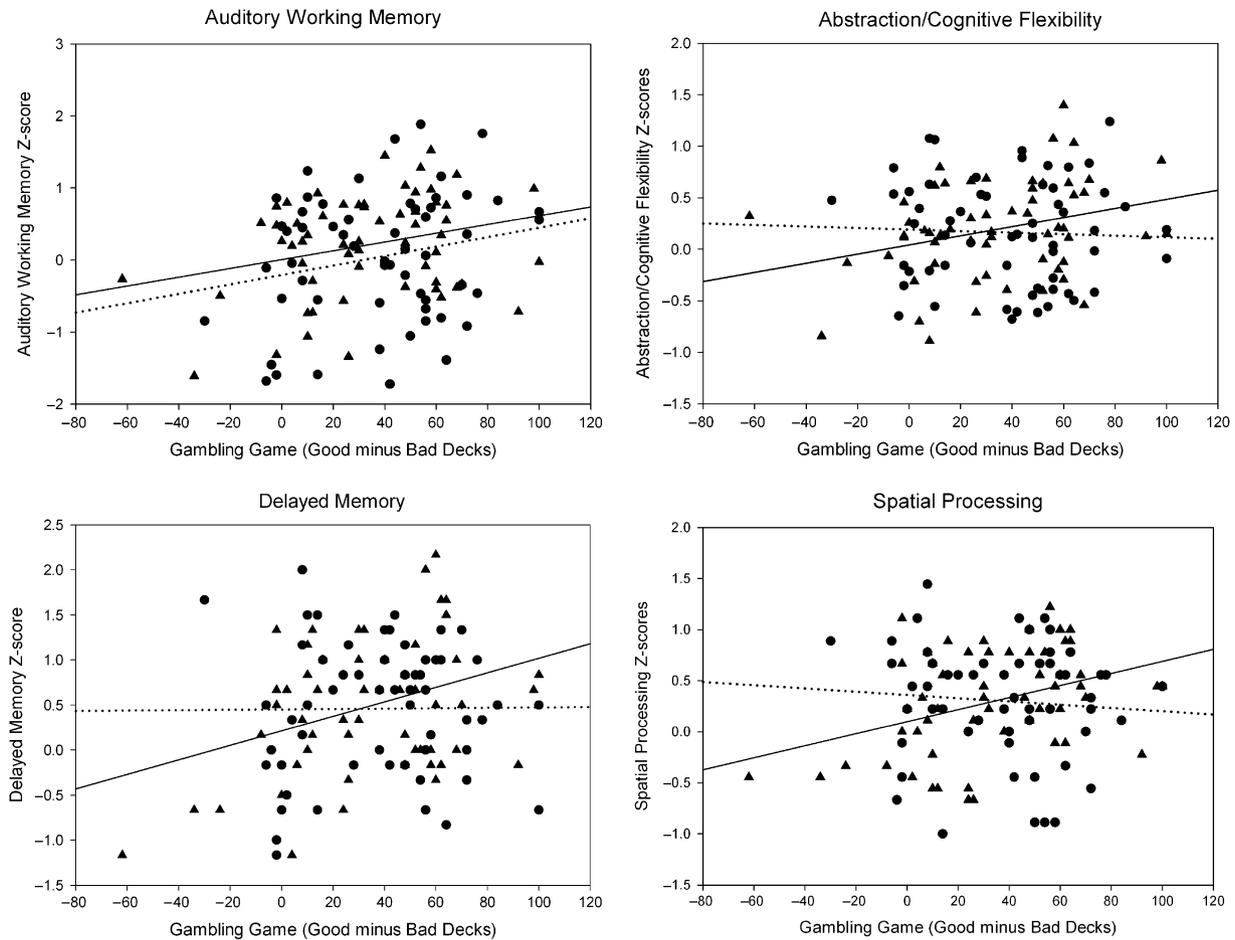
#### *Neuropsychological Performance*

The 2 groups differed only in the attention domain, with the TxN group performing significantly better than controls ( $F_{1,110} = 8.48$ ,  $p < 0.01$ ). Within the TxN group, gambling task performance was positively associated with auditory working memory ( $r = 0.28$ ,  $p < 0.05$ ), abstraction/

cognitive flexibility ( $r = 0.30$ ,  $p < 0.05$ ), delayed memory ( $r = 0.35$ ,  $p < 0.01$ ), and spatial processing ( $r = 0.37$ ,  $p < 0.01$ ). No associations were found between gambling task performance and neuropsychological variables in the control group. These data are displayed in Fig. 2.

#### *Differences Between Long-Term Abstinent Alcoholic Individuals and the TxN Group and Between the Two Control Groups*

As noted above, we previously reported impaired SGT performance in long-term abstinent alcoholic individuals versus normal controls (Fein et al., 2004). To try to make sense of the current result in the context of the prior findings, we examined how our current samples differed from the samples in the earlier study. The abstinent alcoholic sample was significantly older than the TxN sample, but this was to be expected given the nature of the sample (they had already been through treatment and achieved abstinence for a significant length of time). Age, however, did not appear to be an issue, given that the 2 control groups, who were age-matched to the alcohol groups, had nearly identical performances on the gambling task. Furthermore, neither of the alcohol or control groups showed any significant associations between age and gambling task performance (all  $r$ 's  $< \pm 0.22$ , all  $p$ 's  $> 0.16$ ). Overall, the abstinent alcoholic group demonstrated increased severity in terms of the alcohol use measures, a higher degree of FAM\_DD, and increased deviance proneness and externalizing symptoms than that of the TxN sample. Table 1 presents a summary of these comparisons. The TxN and long-term-abstinent alcoholic groups did not differ significantly in their performance on the simulated gambling task ( $F_{1,97} = 1.94$ ,  $p = 0.17$ ). However, in SGT performance, the TxN group's scores were 13% lower than their controls, but 30% higher than the abstinent alcoholic group's scores; thus, their scores were much more similar to those of controls than to those of abstinent alcoholic individuals. Power analysis was conducted to determine how large a sample would be necessary to establish whether the TxN's SGT performance is different from controls or from long-term-abstinent alcohol-dependent individuals. To achieve a power of 0.80 to establish that TxN performed worse than controls, there would need to be approximately 585 controls and TxN individuals in each group (780 for a power of 0.90). To achieve a power of 0.80 to establish that TxN performed better than long-term-abstinent alcohol-dependent individuals, about 150 subjects in each group would be needed (200 in each group for a power of 0.90). The control groups from both of the studies performed comparably on the SGT, suggesting that TxN and long-term-abstinent alcohol-dependent samples can be compared directly, without the need for each sample to have its own age-comparable control sample.



**Fig. 2.** Scatter plots and correlations between simulated gambling task (SGT) performance and various neuropsychological (NP) domains. The treatment-naïve (TxN) sample is represented by ▲s, and the solid line represents their association between SGT performance and NP scores. The controls are represented by ●s, and their association between SGT performance and NP scores are represented by a dotted line. Within the TxN group, gambling task performance was positively associated with auditory working memory ( $p < 0.05$ ), abstraction/cognitive flexibility ( $p < 0.05$ ), delayed memory ( $p < 0.01$ ), and spatial processing ( $p < 0.01$ ). Within the control group there were no significant associations.

## DISCUSSION

Our sample of young-adult treatment-naïve individuals with alcohol dependence (TxN) did not show any evidence of deficits in decision making on the SGT. Simulated gambling task performance was not associated with externalizing symptoms, personality indicators of social deviance, or a family density of alcoholism or measures of alcohol use. Simulated gambling task performance was associated with measures of working memory capacity, abstraction, and spatial processing within the TxN group, but the TxN group did not show any evidence of overall neuropsychological deficits compared with controls. In fact, the TxN group performed better than controls on the attention domain. We do not know whether this is a real finding (given that they performed better than controls on 1 of 10 domains at  $p < 0.01$ , this result would not be significant if corrected for multiple comparisons); nevertheless, it is indicative of the finding that the TxN group does not evidence cognitive impairment. The TxN group had higher levels of externalizing symptoms and higher

levels of deviance proneness than controls; however, the levels of externalizing symptoms and deviance proneness were not as high as that in our previous sample of long-term-abstinent alcoholic individuals, who had impaired decision making in our earlier study using the SGT (Fein et al., 2004). It is not clear exactly why the TxN group did not make poor decisions on the SGT, but the results suggest that this sample does not have the global decision-making deficits that are likely to be detected by the SGT. While TxN individuals clearly make poor decisions in their drinking habits, they do not make poor decisions on the SGT. This suggests that their poor decisions are more specific to their drinking and do not reflect a global deficit in decision making. In addition, the results provide further evidence that alcohol dependence is not a uniform, homogeneous disorder, but a disorder that encompasses a number of distinct populations.

There are a number of possible explanations for our failure to find evidence of decision-making deficits in the TxN sample. First, it may be that the decision-making deficits

**Table 1.** Characteristics of Participant Groups

| Variable   | Tx N study       |                      | Abstinent alcoholic study |                      | Effect size (%)        |                         |                                |
|--|------------------|----------------------|---------------------------|----------------------|------------------------|-------------------------|--------------------------------|
|  | Tx N<br>(N = 58) | Controls<br>(N = 58) | Abs. Alc.<br>(N = 43)     | Controls<br>(N = 58) | TxN versus<br>controls | Abs. Alc.<br>versus TxN | Controls<br>versus<br>controls |
| Age (y)  | 31.1 ± 7.8       | 31.3 ± 7.9           | 46.5 ± 6.6                | 44.6 ± 6.6           | 0.0                    | 53.9 <sup>a</sup>       | 46.0 <sup>a</sup>              |
| Years education  | 16.2 ± 1.5       | 16.5 ± 1.8           | 15.7 ± 2.1                | 16.2 ± 1.8           | 0.7                    | 2.0                     | 0.7                            |
| <i>Alcohol use variables</i>                           |                  |                      |                           |                      |                        |                         |                                |
| Duration of active drinking (mo)                       | 181.2 ± 95.2     | 132.8 ± 98.3         | 260.6 ± 93.7              | 251.2 ± 130.3        | 5.9 <sup>b</sup>       | 15.7 <sup>a</sup>       | 19.6 <sup>a</sup>              |
| Average lifetime drinking dose<br>(std. drinks/mo)     | 84.9 ± 43.3      | 6.6 ± 6.3            | 157.8 ± 131.8             | 6.8 ± 7.1            | 64.3 <sup>b</sup>      | 14.2 <sup>***</sup>     | 0.2                            |
| Duration of peak drinking (mo)                         | 55.6 ± 55.1      | 59.3 ± 90.9          | 74.4 ± 73.4               | 121.2 ± 136.8        | 0.0 <sup>b</sup>       | 2.6                     | 7.3 <sup>a</sup>               |
| Peak drinking dose (std. drinks/mo)                    | 150.9 ± 113.1    | 14.9 ± 13.6          | 317.4 ± 250.9             | 15.9 ± 18.0          | 41.9 <sup>b</sup>      | 17.5 <sup>***</sup>     | 0.1                            |
| Age first met criteria for heavy use                   | 21.2 ± 4.9       | NA                   | 23.5 ± 6.4                | NA                   | NA                     | 6.3 <sup>*</sup>        | NA                             |
| Level at first heavy use                               | 135.9 ± 42.4     | NA                   | 187.1 ± 117.5             | NA                   | NA                     | 9.1 <sup>**</sup>       | NA                             |
| <i>Family drinking density</i>                         |                  |                      |                           |                      |                        |                         |                                |
| FamDD_1 <sup>c</sup>                                   | 0.18             | 0.12                 | 0.41                      | 0.15                 | 2.6                    | 14.5 <sup>***</sup>     | 0.7                            |
| FamDD_2 <sup>d</sup>                                   | 0.20             | 0.11                 | 0.25                      | 0.14                 | 6.6 <sup>**</sup>      | 2.0                     | 0.7                            |
| <i>Deviance proneness &amp; externalizing symptoms</i> |                  |                      |                           |                      |                        |                         |                                |
| CPI Socialization Scale                                | 31.6 ± 5.4       | 37.2 ± 4.5           | 27.7 ± 5.9                | 37.1 ± 3.7           | 25.4 <sup>***</sup>    | 10.4 <sup>***</sup>     | 0.2                            |
| MMPI_Pd Scale  | 18.7 ± 4.5       | 16.1 ± 3.2           | 22.0 ± 4.2                | 16.0 ± 3.6           | 10.8 <sup>***</sup>    | 11.7 <sup>***</sup>     | 0.0                            |
| Externalizing Symptoms (#) <sup>e</sup>                | 9.8 ± 7.7        | 5.6 ± 5.1            | 14.2 ± 7.4                | 4.7 ± 4.3            | 9.6 <sup>***</sup>     | 9.1 <sup>**</sup>       | 0.0                            |
| Gambling game  | 33.2 ± 31.7      | 38.2 ± 29.2          | 23.1 ± 30.3               | 38.2 ± 28.2          | 0.9                    | 2.0                     | 0.1                            |

Measures are reported as mean ± standard deviation. Effect is significance:

\* $p \leq 0.05$ ; \*\* $p \leq 0.01$ ; \*\*\* $p \leq 0.001$ .

<sup>a</sup>Statistical comparisons on age and variables associated with subject's age are not appropriate since age was associated with the study selection criteria.

<sup>b</sup>Statistical comparisons of the groups on alcohol use variables are not valid since alcohol use was part of the group selection criteria.

<sup>c</sup>Total number of problem drinking first-degree relatives divided by the total number of first-degree relatives.

<sup>d</sup>Total number of problem drinking second-degree relatives divided by the total number of second-degree relatives.

<sup>e</sup>Sum of antisocial personality disorder and conduct disorder symptoms from the Diagnostic Interview Schedule.

NA, not applicable; std. drinks/mo, number of standard drinks per month; TxN, treatment-naïves; MMPI\_Pd, Psychopathic Deviance Scale of the MMPI-2; Abs. Alc., Abstinent alcoholics.

observed on the gambling task in other samples are a reflection of the long-term consequence of chronic alcohol abuse and physiological dependence on alcohol. Our sample of young, TxN alcoholic individuals had less severe symptoms of alcohol abuse/dependence and did not drink as heavily nor as long as our sample of abstinent alcoholic individuals who showed evidence of decision-making deficits (Fein et al., 2004). It might be that physiological dependence on alcohol leads the individual to pay more attention in general to short-term outcomes, because the short-term outcome of significant withdrawal symptoms is so motivationally significant for severe alcoholic individuals. In other words, as an individual becomes more and more physiologically dependent on alcohol, the individual becomes more and more concerned about the short-term outcome of withdrawal. As alcohol-seeking and consumption behaviors begin to dominate their lives, they develop the habit of constantly making decisions that put far more weight on short-term outcomes. It is also possible that the impact of long-term exposure to alcohol and physiological dependence on brain structures, such as the ventromedial prefrontal cortex and the amygdala, results in a condition quite similar to that displayed by the patients with ventromedial prefrontal cortex lesions (Bechara et al., 1994) or amygdala lesions (Bar-On et al., 2003; Bechara et al., 2003).

Second, as suggested above, it may also be that our TxN sample comes from a different population of alcohol-dependent individuals, who do not have either a high level of genetic vulnerability to alcoholism or a global decision-making deficit that predates their alcoholism. The TxN group had significantly fewer problem drinker family members compared with our treated alcoholic individuals, which suggests that they have a lower genetic vulnerability to alcoholism. Our TxN subjects also did not show very high levels of deviance proneness or externalizing symptoms compared with other samples of alcoholic individuals who demonstrate decision-making deficits, such as our sample of abstinent alcoholic individuals (Fein et al., 2004) or the antisocial alcoholic individuals in Mazas et al. (2000) or in Finn et al. (2002). It may be that the term "alcoholism" as commonly understood in our society refers only to the more virulent form of alcohol dependence that involves higher genetic vulnerability, SGT impairments, and more severe early alcohol use trajectories. The population studied here may have alcohol dependence that can remit as suggested by the work of Schulenberg et al. (1996), Sher and Gotham (1999), or Dawson et al. (2005).

Deviance proneness and antisocial traits are typically associated with decision-making deficits in substance abusers (Fein et al., 2004; Finn et al., 2002; Mazas et al.,

2000; Stout et al., 2005). In contrast, our data suggest that the decision-making problems in our TxN subjects may be specific to their drinking and not evident in other domains of their life. However, we did not carefully assess the decision making of our TxN subjects in other contexts or with drinking itself. If we are to understand the nature of decision-making problems in the range of types, or degrees of severity, of alcoholism, then it is essential that researchers attempt to characterize the decision making of alcoholic individuals across different domains (i.e., behaviors) and contexts (e.g., drinking vs nondrinking contexts). Future research should attempt to examine how individual alcoholic persons, well characterized in terms of personality, family history, cognitive abilities, and psychopathology, make decisions in domains such as alcohol consumption, sexual behavior, money management (including purchasing behavior), work-related behaviors, eating behaviors, interpersonal conflict, emotion-provoking situations, and other contexts that are motivationally relevant to the individual. Furthermore, it is also very important that researchers assess the influence of the typical context in which the individual makes these sorts of decisions. Animal research suggests that context is an important influence in determining drug administration behavior and response (Robinson and Berridge, 1993). For instance, our TxN subjects might make impulsive decisions when at a bar, or in a typical drinking context, but not in other situations, such as at home with family members or in artificial laboratory environments. Finally, it may be that the decision-making deficits in TxN subjects are subtler or somewhat different than those of our long-term abstinent alcoholic subjects or other samples of substance abusers that show deficits on the gambling task. Although the gambling task models real-life decisions that involve weighing short-term rewards and long-term consequences, it remains an artificial task and it may not be sensitive to more subtle deficits in decision making or to the influence of context on decision making. Given these issues, it may not be the best measure of impaired decision making in young alcoholic subjects.

In summary, our sample of young-adult treatment-naïve alcoholic persons did not show any evidence of deficits in decision making on the SGT in comparison with controls. Simulated gambling task performance was not associated with measures of antisocial symptoms or traits, which were not very elevated in the TxN alcoholic persons compared with other samples of alcoholic persons who perform poorly on the SGT. In addition, SGT performance was not associated with externalizing symptoms, personality indicators of social deviance, family density of alcoholism, or measures of alcohol use habits. This suggests that the poor decisions of TxN participants are more specific to their drinking and do not reflect global deficits in decision making. Additional research is needed on the nature and mechanisms associated with specific and global

decision-making deficits and their relationship with long-term outcomes in alcoholic individuals.

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