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Prediction of Comorbid Alcohol Use Disorders Using Factors Derived from the Positive and Negative Syndrome Scale in an Inpatient Psychiatric Hospital

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PREDICTION OF COMORBID ALCOHOL USE DISORDERS USING FACTORS DERIVED FROM THE POSITIVE AND NEGATIVE SYNDROME SCALE IN AN INPATIENT PSYCHIATRIC HOSPITAL

By

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The present study examined the relationship of schizophrenia-related cognitive deficits and comorbid alcohol use disorders (AUD) among inpatients with psychotic disorders. Relevant research has implicated the role of cognitive impairment in the development of AUD in general population and other psychiatric samples, and preliminary evidence lends credibility to associations between the positive (hallucinations, delusions) and negative (flat affect, lack of motivation) symptoms of schizophrenia and variations in cognitive deficits. Accordingly, it was hypothesized that any increased likelihood of AUD as a function of positive and negative symptomatology would be better accounted for by systematic variations in cognitive functions as they generally relate to the development of AUD. To test this association, a Principal Components Analysis was applied to the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) using data collected from a large sample of inpatients with schizophrenia or schizoaffective disorder. The resultant five component solution included the key Positive (POS), Negative (NEG) and Cognitive (COG) components needed to test the predicted mediation by COG on the relationship between AUD and POS/NEG. Next, interview and archival data pertaining to AUD were collected from 132 (23 female) randomly selected inpatient volunteers, with chart-ratings of AUD serving as the dependent measure in a planned logistic regression analysis. Although support for the primary prediction was not obtained, intriguing post-hoc analyses revealed a significant POS by COG interaction ($\Delta \chi^2(1) = 5.94, p < .05$). Follow-up of this interaction indicated that whereas there was no association between COG and AUD for those with fewer POS symptoms, each unit increase in the severity of COG was associated with a nearly four times decreased likelihood of problematic alcohol use for persons with worse POS, $\Delta \chi^2(1) = 7.20, p < .01$. These results are thought to stem from a “floor effect” in cognitive functioning, such that poor reality-testing acts to limit efforts to compensate for dysfunction and associated distress by drinking. Such findings, though unexpected, should nonetheless spur the dual-diagnosis literature, which at present provides little evaluation of the effects of schizophrenic symptomatology on the development of alcohol problems.
INTRODUCTION

For decades, researchers have sought to capture and define the heterogeneity observed in schizophrenic (SZC) symptomatology (e.g., Berman et al., 1997; Herbener & Harrow, 2001; Peralta & Cuesta, 1994; Vorgunti, Heslegrave, & Awad, 1997) and explore its implications. These endeavors undoubtedly have been complicated by the high rate of comorbid alcohol use disorders (AUD)\(^1\) in SZC populations (D’Mello, Boltz, & Msibi, 1995; Drake, Osher, & Wallach, 1989). Yet, despite the frequent co-occurrence of these disorders and its potential importance, the basis of their connection has not been the target of much research and consequently is poorly understood. This is particularly unfortunate, because it has been suggested that almost any use of psychoactive substances by persons with schizophrenia may be associated with significant adverse consequences (e.g., Bartels, Drake, & McHugo, 1992; Drake et al., 1989). Clearly, efforts to elucidate the nature of SZCs’ elevated risk for AUD are warranted. The present investigation was designed to pursue this course by analyzing how AUDs map onto meaningful, empirically derived subgroups of schizophrenia symptoms.

Specifically, taking into account recent research indicating that alcohol’s deleterious effects on behavior are often mediated by its impact on complex cognitive processes (e.g., Curtin et al., 2001), there is reason to believe that SZCs’ apparent hypersensitivity to alcohol may be due, in part, to the cognitive deficit symptoms commonly associated with schizophrenia. Such cognitive deficits could serve to lower the threshold at which intoxication typically is associated with alcohol-related impairment. These deficits might also be expected to contribute to disadvantageous decisions regarding alcohol use, thereby placing certain cognitively compromised SZCs at heightened risk for recurrent alcohol-related problems and, consequently, AUD. Thus, with regard to AUD-SZC comorbidity, progress toward understanding the connection may be achieved through closer attention to the general heterogeneity of schizophrenia, particularly as it relates to deficits in cognitive functioning.

My principal hypothesis was that because cognitive impairments vary as a function of particular schizophrenia symptom dimensions, alcohol’s effects on SZCs functioning – as well
as the rates of AUD comorbidity – should vary accordingly. The present effort to link the
cognitive and dual-diagnosis\textsuperscript{2} literatures appeared to be an especially promising line of inquiry
because, although non-cognitive symptoms of schizophrenia are frequently the focus of clinical
interventions, recent research has shown that nearly half of the variance in the functional
outcomes of this population can be accounted for by measures of cognitive functioning (Fuji &
Wylie, 2002; Green, 1996; Green et al., 2000; Martinez-Aran et al., 2002; Penades et al., 2001;
Velligan et al., 1997; Wilder-Willis et al., 2002).

**Background and Significance**

Although prevalence estimates have varied across studies, approximately one-third of
SZCs may suffer from current symptoms of AUD (D’Mello et al., 1995; Drake et al., 1989; Drake &
Mueser, 1996; Drake & Wallach, 1989; Regier et al., 1990). Improved insight into the
mechanisms underlying the comorbidity of these disorders seems especially important because
SZCs with comorbid AUD appear to evidence significantly greater impairment in their overall
functioning relative to those without addiction problems (Bunkley, 2002; Krausz et al., 1996;
Lammertink et al., 2001; McEvoy & Allen, 2003). For example, dually-diagnosed SZCs have
greater difficulty with daily living skills, managing finances, and maintaining stable housing
(Drake et al., 1989; Drake & Wallach, 1989; Osher et al., 1994) and evidence more medical
problems than their non-dually diagnosed counterparts (Drake et al., 1989). Substance use has
also been associated with hostile, threatening behavior among those with chronic mental
illnesses like schizophrenia (Arango et al., 1999; Drake et al., 1989; Smith & Hucker, 1994).
Relative to SZCs without addiction problems, comorbid AUD has been linked to abuse of other
drugs (Osher et al., 1994), greater susceptibility to depression, and higher rates of suicide
(Bartels et al., 1992; Heilä et al, 1997; Krausz et al., 1996). Although certain service-integration
issues may be involved (Calloway & Morrissey, 1998; Jackson et al., 2001; Lett, 1988; Ridgley,
Goldman, & Willenbring, 1990), the higher rates of rehospitalization observed among dually-
diagnosed SZCs as compared to the non-dually diagnosed in some studies (e.g., Drake et al.,
1989; Fischer, Owen, & Cuffel,1996; Jackson et al., 2001; Kivlahan et al., 1991; Moos & Moos,
1995; Osher et al., 1994)\textsuperscript{3} suggests that SZCs with AUD may be particularly unresponsive to

In some ways, the negative outcomes associated with comorbid AUD among SZCs
appear similar to those observed in non-SZC AUD populations. As such, the elevated
impairment of dually-diagnosed SZCs could be interpreted as indicative of the simple additive
effects of two disorders relative to the impact of an uncomplicated, single schizophrenia
diagnosis. Nonetheless, whereas the adverse consequences associated with primary AUD in non-psychiatric samples typically emerge after many years of heavy alcohol consumption, even relatively low levels of alcohol consumption can exert a significant negative impact on the functioning of SZCs (Bartels et al., 1992). Furthermore, some negative consequences associated with SZCs’ alcohol use may occur in connection with factors unlikely to be relevant to primary AUD, such as non-compliance with psychiatric treatment regimens. Although medication non-compliance is fairly common among SZCs without AUD, it appears to be even more prevalent among those who are dually diagnosed (Drake & Mueser, 2002; Drake et al., 1989; Hunt, Bergen, & Bashir, 2002; Jackson et al., 2001) and also has been found to covary systematically with alcohol consumption, rather than abuse diagnosis, in at least one study (Pristach & Smith, 1990). The association of medication non-compliance with simple use of alcohol -- regardless of quantity-frequency -- implies that almost any use of alcohol by this population risks the development of consequences consistent with AUD (Bartels, Drake, & Wallach, 1995; Drake et al., 1989). Indeed, one longitudinal investigation found that fewer than five percent of those with severe mental illnesses, like schizophrenia, were able to drink moderately without encountering problems in a number of psychosocial domains (social, vocational, psychological) and later development of some AUD at four to seven year follow-up (Drake & Wallach, 1993; Drake, McHugo, & Noordsey, 1993).

Of course, there is not unequivocal evidence of marked adversity associated with any alcohol use by SZCs (Drake & Mueser, 1996; Fowler et al., 1998). Certainly, it is acknowledged that the elevated risk of AUD among SZCs does not mean all who imbibe will manifest them. Indeed, some research suggests that SZCs who drink alcohol may be less impaired, at least prior to the onset of schizophrenia, than those who do not imbibe (Arndt et al., 1992; D’Mello et al., 1995; Kirkpatrick et al., 1996; Krausz et al., 1996; Sanguineti & Samuel, 1994). For example, one study observed that SZCs with a history of heavy alcohol use were older at the time of their first mental health contact, which may suggest a more chronic, insidious course of illness for their non-drinking counterparts (Zisook et al., 1992). However, currently abstinent SZCs with a history of alcohol use, by definition, must have possessed the discipline, resources, and skills necessary to quit, and thus might constitute a sample with more limited cognitive impairment. Further, given that “history” was defined in this study as heavy use of alcohol, rather than as alcohol-related symptoms, this comparison group might represent a select sample of SZCs with better cognitive functioning and a relatively benign alcohol use history. Nonetheless, despite some mixed evidence with regard to differences in premorbid functioning
– which itself is difficult to assess – there is agreement that SZCs with addiction problems experience worse outcomes over the course of their illness compared to those with schizophrenia alone (e.g., Kirkpatrick et al., 1996; Soyka, 1994).

Mixed results on the correlates of AUD and alcohol use among SZCs also could be partially attributed to the relative inattention to the heterogeneity of schizophrenia in the extant literature, particularly as it relates to cognitive deficits that may be critical to the development of AUD. Yet the probable confound introduced by treating highly variable SZCs as a single group has only briefly been alluded to in the dual-diagnosis literature (e.g., Carpenter et al., 1999a; D’Mello et al., 1995; Pishkin & Bourne, 1981; Woodward et al., 1991). To their credit, some investigators have described their dually diagnosed samples as “variable” (e.g., Pristach & Smith, 1990; Zisook et al., 1992), but differences have only rarely been evaluated systematically (e.g., Arndt et al., 1992; Bunkley, 2002; Hambrecht & Haffner, 1996; Kirkpatrick et al., 1996). The relative lack of consensus about how to characterize the apparent heterogeneity of schizophrenia (Berman et al., 1997) may be partially to blame for this oversight. However, improved understanding of dual diagnosis may depend on efforts, like the present study, to address this diagnostic ambiguity empirically.

Certainly, a detailed discussion of the various typologies of schizophrenia is beyond the scope of this paper (for review, see Hoenig, 1983), but the potential role of variations or subtyping of symptoms in dual diagnosis is germane to the present purpose. In the following discussion, I argue for the value of considering such connections by integrating loosely connected and otherwise separate concepts from the AUD and schizophrenia literatures.

**Variations in Schizophrenia Symptoms: Potential Impact on Dual Diagnosis**

The latest revision of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; American Psychiatric Association [APA], 1994) reflects a culmination of research and theory regarding the heterogeneity of schizophrenia in its recognition of three distinct subtypes of schizophrenia (i.e., Paranoid, Disorganized, Catatonic), two additional classifications for more ambiguous presentations (i.e., Undifferentiated, and Residual) and one for co-occurring mood episodes (i.e., Schizoaffective Disorder). Research on prevalence rates of AUD among subtypes of schizophrenia suggests that Alcohol Abuse Disorder may be higher among Paranoid-Type SZCs than in any other inpatient psychiatric diagnostic group (Alterman, Erdlen, & Murphy, 1981) and that those SZCs who have used substances heavily were least likely to be identified as having Schizophrenia, Disorganized-Type (Zisook et al., 1992). Although providing some preliminary support for the notion that rates of AUD may vary as a function of
schizophrenia subtype, the use of relatively small samples and differing definitions of schizophrenia with earlier editions of the DSM may limit generalizability of these results.

To my knowledge, there has been little other research examining the relationship of AUD to DSM schizophrenia subtypes, and several practical problems with this strategy may be partly responsible for the omission. For example, the DSM instructs diagnosticians to determine subtype classifications on the basis of the current symptom presentation (APA, 1994). This could complicate longitudinal research as less-stable diagnostic specifications (e.g., Undifferentiated, Residual) may vary from admission to admission (Goldberg & Weinberger, 1995). Furthermore, although the DSM subtypes may be reliably diagnosable in theory, they may not be in practice. Specific subtypes may be largely irrelevant to the treatment community inasmuch as each is generally treated in the same manner (i.e., psychotropic medication). It is also plausible that the current controversy over the existence and characterization of SZC subtypes (e.g., Cardno & Farmer, 1995; Carpenter et al., 1999a; Garver, 1997; Goldberg & Weinberger, 1995; Lenzenweger, 1999; Seaton, Goldstein, & Allen, 2001) has contributed to the neglect of subtype classifications in the relevant dual-diagnosis literature. Indeed, some have argued that assignment to subtypes risks loss of valuable information concerning variance along symptom dimensions (Lenzenweger, 1999).

However, examinations of the relationships between schizophrenia symptoms and use patterns of specific substances, like alcohol, have not revealed any consistent pattern of association (e.g., Miles et al., 2003; Mueser, Drake, & Wallach, 1998). Whereas some research has suggested that the dually-diagnosed show fewer severe negative symptoms (e.g., Kirkpatrick et al., 1996; Soyka, 1994), others have found no symptomatic differences among groups defined by lifetime history of substance problems in general (Cuffel & Chase, 1994) or AUD specifically (D’Mello et al., 1995; Krausz et al., 1996; Zisook et al., 1992). In other research, however, SZCs who engaged in heavy alcohol consumption (5 or more drinks per episode) were rated as having less severe thought disorder and fewer positive symptoms than their abstinent counterparts (Dixon et al., 1991; Messias & Bienvenu, 2003; Osher et al., 1994). In contrast, others have observed higher proportions of auditory hallucinations (Bunkley, 2002; Hambrecht & Hafner, 1996), disorganized speech (Cuffel, Heithoff, & Lawson, 1993) and delusions (Barbee et al., 1989; Bunkley, 2002; Strakowski et al., 1994), mainly paranoid or suspicious delusions (e.g., Bunkley, 2002; Kirkpatrick et al., 1996; Messias & Bienvenu, 2003) among the dually-diagnosed relative to SZCs without addiction problems. An alternate position holds that the relationship of schizophrenia symptoms and dual diagnosis is dependant on AUD
severity, but evidence in this connection is similarly mixed. For example, whereas some have noted that SZCs with Alcohol Abuse Disorder, but not Dependence Disorder, reported more delusions and hallucinations than SZCs without AUD (Hambrecht & Hafner, 1996), others have observed a positive association between the total number of self-reported hallucinations with Alcohol Dependence symptoms, but not Alcohol Abuse symptoms (Brunette et al., 1997).

At first glance, it may be difficult to make sense of these apparently contradictory findings with regard to the relationship of schizophrenia symptoms, alcohol use, and AUD. However, upon closer examination, several possible explanations emerge. First, the computation of bivariate correlations of AUD diagnosis with dichotomous symptom variables (present vs. absent) may obscure important variations in severity along these symptom dimensions as they relate to AUD. Second, although simple tallies of particular SZC symptoms (e.g., hallucinations) can provide a reasonable proxy of symptom severity, it is unclear if these effects are truly additive. Further, the computation of zero-order bivariate correlations (e.g., number of delusions and AUD) does not account for the potential shared variance among symptoms, which is necessary to assess the unique contributions of each SZC symptom (or sets of symptoms) to dual diagnosis. A multivariate consideration of SZC symptoms, measured on a continuum of severity, should yield a more meaningful account of the range of clinical presentations observed in schizophrenia (Peralta & Cuesta, 1994) and its correlates.

In an effort to address these shortcomings, the present study made use of the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987). This widely used, psychometrically-sound instrument provides a standardized measure of the SZCs’ overall clinical profile (Kay, Opler, & Lindenmayer, 1988; Kay, Opler, & Lindenmayer, 1989; Mass et al., 2000) while also allowing for independent and multivariate examination of schizophrenia symptom dimensions. The PANSS was initially designed to measure the Type I and Type II- syndromes of schizophrenia, a typology based on the relative predominance of positive (e.g., delusions, hallucinations, floridly bizarre behavior, inappropriate affect), and negative (e.g., flattened affect, avolition, alogia, social/emotional withdrawal) symptoms, respectively, as well as their proposed underlying etiologies: abnormal dopaminergic activity and structural neurological abnormalities, respectively (Berrios, 1985; Crow, 1980). PANSS items are rated on a seven-point continuum of severity and summed to yield four scores: Positive symptoms (seven items), Negative symptoms (seven items), General Psychopathology (16 items), and a Composite score (Kay et al., 1987; Kay et al., 1988).
To my knowledge, just two studies have systematically examined the prevalence of AUD as a function of schizophrenia symptoms indexed by the PANSS. In the first study, conducted with psychiatric outpatients in Spain, higher rates of AUD were found in SZCs relative to non-SZC psychiatric controls (Arias, Padin Calo, & Fernandez Gonzalez, 1997) similar to research with U.S. samples. However, new insights can be gleaned from the finding that these elevated rates of AUD were found only among SZCs with predominant Positive symptoms, with lower rates of Alcohol Dependence Disorder observed in SZCs exhibiting prominent Negative symptoms and those of “mixed” presentation (positive and negative symptoms), (Arias et al., 1997). However, the second study, also conducted in Spain, found no differences in rates of substance disorders between psychiatric groups defined by the PANSS subscales (Santamarina & Iglesias, 2001). Of course, given the potential for cross-cultural differences in rates as well as definitions of AUD (e.g., Gureje et al., 1997; Schmidt & Room, 1999), generalization of these study results to a U.S. sample may be limited. Additionally, because these studies were published only in Spanish, it is unknown if the PANSS cutoff scores used for classification into Positive and Negative subgroups were those suggested by the authors of this instrument based on work with US samples (see Kay et al., 1988) or even if these cutoff scores have cross-cultural applicability. Furthermore, the overall validity of these PANSS subgroups has been questioned by some theorists because nearly one-third of SZCs can be classified as “Mixed” (Goldberg & Weinberger, 1995), a rather nebulous category in which the relative predominance of Positive and Negative symptoms may vary considerably across studies.

In recent years, the Positive and Negative subscales of the PANSS have come to be regarded less as categorical and more as dimensional constructs measured on continuous scales of increasing severity and potentially subject to multivariate consideration. Yet, to my knowledge, there has been no direct examination of the prevalence of AUD as a function of the PANSS dimensions. In the course of examining predictors of homelessness in a SZC sample, however, researchers found that although higher PANSS Positive subscale scores were observed among the homeless SZCs as compared to their domiciled counterparts, these differences were eliminated when only non-addicted, medication compliant SZCs were considered in the analyses (Opler et al., 1994). It perhaps can be inferred from these results that substance problems may account for at least some of the variance in the PANSS Positive subscale scores in this sample. However, it is unclear whether the proportion of variance accounted for by substance problems, or AUD in particular, would have achieved statistical significance had medication compliance -- which appears to be associated with substance use
in this population (e.g., Drake & Mueser, 2002; Drake et al., 1989) -- not been a criterion for inclusion in follow-up analysis.

Given that no consistent pattern of relationships between alcohol use and/or AUD and the symptoms/subtypes of schizophrenia has been observed, this line of research has thus far done little to illuminate the nature of the AUD-SZC connection. Although the relevance of SZC symptoms to dual diagnosis has continued to be a matter of some debate and speculation, the idea that alcohol use/problems may emerge as a consequence of factors associated with SZC symptomatology is a common feature of several dual-diagnosis conceptualizations (see Mueser et al., 1998). For example, based on research indicating that SZCs may develop alcohol problems with lesser consumption than those with AUD alone (Bartels et al, 1992; Drake et al., 1989), the “Supersensitivity Hypothesis” proposes that SZCs may be considerably more vulnerable to alcohol’s deleterious effects on their psychological functioning than are normal or other psychiatric groups (Mueser et al., 1998). Of course, a direct test of this theory calls for alcohol challenge procedures, which are, for ethical and pragmatic reasons, generally not feasible. Thus, although the idea that SZCs are particularly sensitive to alcohol may be theoretically appealing, the possible mechanisms of this effect have not yet been elaborated.

One possibility is that because cognitive deficits are associated with the SZC symptomatology, lesser amounts of alcohol may be needed to produce significant alcohol-related cognitive impairment which, in turn, leads to adverse consequences that are either not recalled or not linked to alcohol use when SZCs are presented with opportunities to drink. In this connection, the inconsistent pattern of relationships obtained between the positive and negative symptoms of schizophrenia, alcohol use, and AUD in previous research may be due, in part, to the relative lack of attention to variations in cognitive deficits at the core of the SZC symptomatology -- even in the rare case where these data were available (e.g., Sevy et al., 2001). This appears to be a gross oversight not only because certain cognitive impairments may be critical to the development of AUD in general, but also because variations in cognitive deficits seem to covary with the clinical symptoms of schizophrenia. These issues are discussed next.

Parallels among the Cognitive Deficits of Schizophrenia, Acute Intoxication, and AUD

A comparative analysis of the cognitive deficit symptoms commonly associated with schizophrenia and the sorts of cognitive errors that can result from acute alcohol consumption reveals some noteworthy similarities that potentially can speak to SZCs elevated risk for alcohol problems. For example, one study found that although SZCs were as capable as normal controls in learning simple rules for identifying physical attributes of various stimuli (e.g.,
distinguishing stimuli on the basis of color), when increasingly complex rules (e.g., apply color, size, and shape) were required for attribute identification, SZCs performed more poorly than normals. That is, whereas additional pieces of information tended to enhance the performance of normal participants on attribute identification tasks, they proved to be detrimental to SZCs’ performance under similar conditions (Pishkin & Bourne, 1981). Although not specifically tested in this study, it stands to reason that such cognitive errors probably extend to dealing with stimuli of varying complexity within a multifaceted, environmental context. In some ways, these symptomatic cognitive deficits resemble the “alcohol myopia” that can result from acute intoxication, in which attention is restricted to explicit cues in the immediate environment, with inadequate detection of, or a failure to process, more subtle or peripheral features of the environmental context (cf. Curtin et al., 2001; Steele & Josephs, 1990).

Research within the general alcohol literature also supports the idea that the problematic effects of alcohol on behavior result from alcohol’s deleterious impact on controlled cognitive processes, such as those required for dealing with abstract/complex environmental stimuli, competing demands on cognitive resources, and changing response contingencies, which are necessary to respond appropriately to situational demands (e.g., Casbon et al., 2003; Curtin et al., 2001). For instance, performance on tasks involving working memory, a controlled cognitive process that involves updating a planned response to incorporate new information, appears to be hindered by moderate doses of alcohol in normal population samples (e.g., Casbon et al., 2003; Dougherty et al., 1999; Dougherty et al., 2000; Kirchner & Sayette, 2003). Similarly, research with SZCs has consistently demonstrated their performance deficits on tasks that involve encoding or other demands on working memory (e.g., Barch et al., 2002; Coleman et al., 2002; Elhevåg, Fisher, & Goldberg, 2003; Fossati et al., 1991; Gold et al., 2003; Green, 1996; Green et al., 2000; Hartman et al., 2002; Park & Holzman, 1992; Park et al., 2003; Rund, 1998; Spindler et al., 1997; Zorrilla et al., 2003). In a relevant study, SZCs and non-SZC psychiatric controls participated in a gambling paradigm in which data concerning odds ratios were provided to enhance betting decisions. Results indicated that SZCs’ poorer performance relative to matched controls was not attributable to simple risk-taking (high bets regardless of odds ratios), but rather to slower, less-efficient decision-making in response to the changing information (Hutton et al., 2002).

Thus, it could be argued that prior to engaging in alcohol consumption, the sober baseline cognitive functioning of many SZCs may be at a level not unlike moderately intoxicated normal individuals, thereby perhaps lowering their threshold for typical alcohol-related
impairment. In other words, lesser amounts of alcohol may be needed to result in significant alcohol-related cognitive impairment such as working memory deficits, behavioral disinhibition, and impaired judgment (Curtin et al., 2001), all of which can lead to adverse consequences beyond drinking alone (Giancola et al., 1996a). However, perhaps due to ethical concerns, there have been no studies that attempt to characterize the effects of acute alcohol exposure under experimental conditions that could address the specific hypothesis that SZCs are especially sensitive to alcohol’s deleterious effects on cognitive functions because relative to average drinkers, they begin with compromised cognitive functioning in the sober state.

Furthermore, if it is assumed that almost any use of alcohol by SZCs may be associated with negative consequences, as the evidence seems to suggest (e.g., Bartels et al., 1992; Drake et al., 1989), then the cognitive processes involved in decisions to drink or not to drink become critically important to the development and operation of comorbid AUD in this population as well. In general, as alcohol use progresses, the reasons both to use and to avoid use are likely to accumulate, such that the problem drinker’s reactions to alcohol cues (e.g., bars, drinking acquaintances, etc.) may become increasingly complex and elicit competing response sequences (cf. Breiner, Strizke, & Lang, 1999). Thus, as addiction develops, the decision processes surrounding management of drinking are apt to involve escalating cognitive demands, due to conflicting inclinations to indulge the desire to drink (approach) and to avoid the adverse consequences of consumption (avoidance). Naturally, problematic drinking can emerge from a singular focus on approach inclinations (Breiner et al., 1999). In some sense, this reflects simplistic decision making, taking the easy way out – indulgence and immediate gratification. In contrast, the characteristically distal and delayed consequences of alcohol use may require the use of rather complex cognitive rules to make the connections between alcohol consumption and subsequent negative outcomes necessary to refrain from use (cf. Orford, 1985). If it is presumed that the same cognitive-behavioral processes relevant to the development of addiction within the general population extend to persons with schizophrenia, SZCs who are compromised in cognitive functions critical to avoidance inclinations may show impaired capacity to resolve drinking decisions in favor of abstinence (Bellack & Gearon, 1998; Brunette et al., 1997; Carey & Correia, 1998; Drake, Rosenberg, & Mueser, 1996; Drake et al., 1990; Goldfinger et al., 1996; Smith et al., 2000; Test et al., 1989), thereby placing them at elevated risk for adverse alcohol-related consequences and AUD. Indeed, SZCs’ self-reported drinking motives commonly reflect “approach” inclinations with immediate consequences, such as enhancement and coping (Dixon et al., 1990; Spencer, Castle, & Michie, 2002).
In this connection, performance deficits on tasks involving delayed retrieval, over and above any problems with encoding and working memory, are commonly observed in persons with schizophrenia (Green, 1996; Green et al., 2000; Keefe et al., 2002; Rund, 1998). Certainly, a failure to adequately encode personal historical events could impact SZCs' capacity to access past experiences concerning the adverse consequences associated with their alcohol use, particularly because such consequences are often temporally delayed. Inasmuch as recalled material may be subject to distortion (Smelson et al., 2002) of varying degrees across individuals in general population samples (Stanovich & West, 1998), the cognitive impairments associated with schizophrenia symptomatology may make certain SZCs especially prone to such bias. Even if SZCs were able to effectively recall the negative consequences associated with their use of alcohol, as noted above, their capacity for abstract reasoning with complex stimuli is commonly impaired (Pishkin & Bourne, 1981). Thus, as factors pertinent to alcohol use grow increasingly numerous and complex, SZCs decisions to use or not to use may be resolved on the basis of motives that are most simple and immediate to them, which tends to mean greater inclinations to approach and consume alcohol.

Indeed, much like persons with frontal lobe dysfunctions (Morris et al., 1995), many SZCs are compromised in their ability to weigh the risks, benefits, and probabilities of situational factors in making decisions. This may become particularly problematic when such decisions involve mental health concerns. For example, in one study, psychiatric patients were told a hypothetical story about someone with his/her illness along with specific information on available treatment options and the advantages/disadvantages thereof. Next, participants were asked to advise a treatment to the hypothetical patient and offer some rationale for their choice. Although SZCs were as able as other psychiatric patients to select and express a treatment option, their abilities to understand relevant information (e.g., medications, side-effects, etc.), reason about the risks and benefits of treatments options, and/or appreciate the nature of the situation and the relevant consequences were significantly impaired (Grisso & Appelbaum, 1995; Grisso, Abbelbaum, & Hill-Fotouhi, 1997). Thus, SZCs may resolve drinking decisions with little appreciation for or consideration of the potential consequences of indulgence.

Certainly, deficits in cognitive processes critical to complex decision-making are observed in patients with AUD alone, and some research evidence suggests that simple AUD, in both its acute and chronic expression, impairs abilities to weigh the risks, benefits, and probabilities in making advantageous decisions (e.g., Homer et al., 1999b; Mazas, Finn, & Steinmetz, 2000; Rosenberg et al., 1990; Sullivan et al., 1993). Generalized intellectual deficits,
impaired visual-spatial abilities, and impairments in higher cognitive functions (abstract reasoning, planning, and attention) are commonly associated with prolonged, regular heavy alcohol consumption (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2001). Although the domains of alcohol-related cognitive impairment may appear on the face of it to be quite similar to those impacted by schizophrenia -- and indeed persons with primary AUD can show deficits in cognitive functioning relative to the general population -- such impairments rarely fall within the clinical range of severity (Krabeendam et al., 2000; Nixon, Hallford, & Tivis, 1996). Indeed, only a small proportion of the heaviest drinkers may develop irreversible brain damage (e.g., Korsakoff’s syndrome). Although the precise quantity/frequency of alcohol consumption resulting in significant cognitive impairment remains unclear, the most severe cognitive deficits, like those associated with schizophrenia, likely would take many years of continued heavy use to develop in non-SZCs. Nonetheless, there exists some data to suggest that even mild to moderate drinking over a prolonged period of time can also adversely impact cognitive functioning (Giancola et al., 1996b; NIAAA, 2001). Thus, it could be argued that evidence for worse cognitive impairment among the dual-diagnosed simply may result from the cumulative effects of alcohol use on cognitive performance.

For several reasons, however, any long-term effects of chronic, low level alcohol consumption is probably of minimal import with regard to the schizophrenia-related cognitive deficit symptoms to be explored in the present study. First, despite the independent associations of AUD and schizophrenia with cognitive impairment, most research indicates little evidence for a synergistic impact of the co-occurrence of AUD on the cognitive functioning of SZCs (Addington & Addington, 1997; Cleaveland & Denier, 1997; Cleghorn et al., 1991; Deschmukh et al., 2002; Holthausen et al., 2002; Nazzaro, 2002; Nixon et al., 1996; Seaton et al., 2001; Sevy et al., 2001; Sullivan et al., 2003), perhaps because schizophrenia alone exerts a floor effect where cognitive impairment is concerned. Second, whereas the cognitive deficits associated with schizophrenia seldom improve (Rund, 1998; Seaton et al., 2001), cognitive functions often recover among those with primary AUD with a few months of abstinence (Clark, 1984; Cleaveland & Denier, 1997). Finally, although many years of regular, moderate alcohol consumption may adversely affect cognitive functioning in normals, SZCs are probably less likely to engage in such consistent alcohol consumption patterns simply because they are more likely to experience significant adverse consequences at low-level doses, perhaps resulting in external constraints on opportunities for continued use (e.g., hospitalization, or incarceration).
To summarize, I have argued that because compromised cognitive functioning is associated with schizophrenia symptomatology, lesser amounts of alcohol may be needed to produce the sorts of alcohol-related cognitive impairment that contribute to dysregulated behaviors and their resultant harmful outcomes. Moreover, cognitively-impaired SZCs may be particularly inclined to initiate drinking episodes despite a history of aversive consequences from them because the negative outcomes of past alcohol use are either (a) not recalled, or (b) not cognitively associated with alcohol use. For them, the factors affecting decisions to drink or not to drink may be limited to the immediate, concrete cues (alcohol’s positive effects) that favor approach, to the relative neglect of more delayed avoidance considerations that tend to require complex cognitive processes, including recall of past negative consequences and the conscious connection of them to alcohol use. Furthermore, to the extent that cognitive deficits vary within the SZC population, certain subgroups of SZCs may be particularly susceptible to maladaptive decisions regarding both the initiation of alcohol use and the curtailment of use once a drinking episode has commenced. Consequently, empirical efforts to explain cognitive symptom variability may be critical to the identification of those SZCs in need of AUD intervention. A promising step in this direction may come from preliminary evidence indicating a relationship between the variability in the clinical symptoms of schizophrenia and specific cognitive deficits.

Cognitive Deficits Vary with Positive and Negative Schizophrenia Symptoms

Evidence concerning cognitive deficits in schizophrenia often points to diffuse, generalized impairments irrespective of DSM schizophrenia subtype (Goldberg & Weinberger, 1995). However, there exists a body of research suggesting that certain cognitive impairments do in fact vary with negative (flattened affect, alogia, amotivation) and positive (hallucinations, delusions) symptomatology. Specific relationships between negative symptoms and cognitive deficits in sensory integration (Arango, Kirkpatrick, & Buchanan, 2000), social problem solving ability (Green, 1996), and executive functions (Brier et al., 1991; Dinn et al., 2002; Johnson-Selfridge & Zalewiski, 2001) have been obtained, whereas other research suggests more generalized cognitive impairment with increasing negative symptomatology. Such associations have been less consistently obtained with regard to positive symptoms (e.g., Arango et al., 2000; Brier et al., 1991; Dinn et al., 2002; Green, 1996; Hughes et al., 2002; Rund, 1998), but recent research indicates a link between positive symptomatology and specific types of cognitive distortions, some of which may be pertinent to alcohol decisions.

For example, SZCs who present with certain Schneiderian “first-rank symptoms” (e.g., third person auditory hallucinations, passivity phenomena, thought insertion/ broadcasting) have
been found to evidence specific cognitive deficits in source-monitoring, that is, identifying the origin of newly acquired information. In one relevant study, SZCs and matched controls were presented with several varied visual/verbal stimuli and asked to generate verbal associations to them. Though SZCs were as able as control subjects to recognize the visual/verbal stimuli as coming from an external source (i.e., provided by the researchers), those with “first rank” symptoms were more likely to identify self-generated associations as coming from an external source as well – a result not inconsistent with their pathology. That is, an inability to identify oneself as the origin of self-generated information may lead to faulty conclusions that experiences were derived outside oneself, perhaps taking the form of positive symptoms like hallucinations and delusions (Keefe et al., 2002). By extension, perhaps alcohol-related experiences may not be perceived as self-relevant among those SZCs with these types of positive symptoms, thereby hindering the cognitive processes involved in formulating connections between alcohol use and consequent problems necessary to refrain from use.

Other research suggests that delusions, another unquestionably “positive” symptom, may be associated with specific types of cognitive distortions as well (Garety, 1991). Specifically, impairment in the ability to gather information pertinent to the formulation of hypotheses and to evaluate the likelihood of competing hypotheses have been proposed to underlie the development of delusions in some SZCs (Hemsley & Garety, 1986). Compared to normals and other psychiatric groups, delusional patients generally request fewer items of information before arriving at judgments and also tend to express more certainty in their validity of faulty conclusions. In other words, deluded SZCs were more likely to respond quickly to new, potentially contradictory probabilistic information, suggesting that they are highly influenced by more proximal stimuli and make little use of information previously provided to them (Garety, 1991; Huq, Garety, & Hemsley, 1988). These cognitive errors may be especially evident when the material is self-relevant and emotionally salient (McGuire et al., 2001), as perhaps with alcohol experiences. Thus, for SZCs with delusions, drinking decisions may be based on more immediate, salient cues that generally favor consumption – perhaps placing this subgroup at increased risk for AUD development.

In contrast, intact cognitive functioning is typical of the DSM-IV Paranoid Type (Kremen et al., 2000; Palmer et al, 1997) and paranoid subtype SZCs appear better able to attend to the contextual aspects of complex stimuli than those presenting with other subtypes of schizophrenia (Strauss, 1993). Thus, whereas other SZCs may be sensitive to alcohol’s deleterious effects because they start off cognitively compromised in the sober state, those of
the Paranoid Subtype may not evidence the sorts of cognitive errors associated with “alcohol myopia” with low levels of use. Nonetheless, certain cognitive errors have been observed among Paranoid-Type SZCs that potentially shed light on the little research suggesting their elevated AUD rates (e.g., Alterman et al., 1981). One study, for example, showed that paranoid SZCs were more likely than non-paranoid SZCs and normals to report correlations between events that are in reality, uncorrelated (Brennan & Hemsley, 1984) -- a result congruent with their psychopathology. Therefore, it is plausible that paranoid SZCs may be prone to formulate erroneous associations with regard to their ostensibly alcohol-related problems, relating these adverse consequences to something other than alcohol. Such deficits could possibly contribute to disadvantageous decisions regarding alcohol use when drinking opportunities arise. Other research employing neurophysiological methods showed that severe auditory hallucinations, particularly among those of the paranoid subtype, were associated with significantly more errors based on “slips”, i.e., knowing the right answer but failing to carry out the appropriate behavioral response (Mathalon et al., 2002). Thus Paranoid-Type SZCs who suffer hallucinations may engage in alcohol use despite knowledge of past adverse consequences from it, thereby perhaps contributing to their risk for AUD.

To summarize, preliminary evidence supports the connection of both generalized and specific cognitive deficits not only with the negative symptoms of schizophrenia, but also with certain positive symptoms. However, these relationships may have been obscured in some research (e.g., Arango et al., 2000; Green, 1996; Rund, 1998) because the relative contributions of each symptom cannot be considered when sum totals of positive symptoms alone are used for analysis without regard to their severity. The inconsistent results observed across studies may also be due to subtle differences in how these positive and negative symptom dimensions were operationalized. In this connection, the PANSS offers an important advantage as a structured and reliable method for evaluating schizophrenia symptom dimensions. Furthermore, recent findings from factor-analytic research with the PANSS suggest a method by which the relative contributions of each SZC symptom may be better captured for the study of symptom dimensions, including core cognitive impairments.

The PANSS and Cognitive Deficit Symptoms of Schizophrenia

Over the past decade, several studies have examined the cognitive correlates of the PANSS subscales (Kay et al., 1987), but results have been mixed. In most studies, SZCs are rated on the PANSS, administered a battery of neuropsychological tests, and bivariate correlational analysis are conducted (e.g., Bell et al., 1994b; Berman et al., 1997; Collins et al.,
1997; Daban et al., 2003; Potkin et al., 2002; Vorunganti et al., 1997; Wong et al., 1997; Yazici et al., 2002). Overall, this research indicates that a relationship probably exists between cognitive deficits and the symptoms of schizophrenia as measured by the PANSS subscales, but computation of large numbers of correlations (one hundred or more) with relatively small samples of SZCs (fifty or fewer) limit any definitive interpretations concerning these relationships (i.e., Berman et al., 1997; Vorunganti et al., 1997; Wong et al., 1997). Furthermore, restriction of samples to only those with the highest level of cognitively functioning (i.e., those capable of completing the research procedures, mental-status exam scores, exclusion of organic or dually-diagnosed) in some studies also may account, in part, for the variable findings and potentially may limit generalizability of these results to the population to be sampled in the present study. Moreover, most of these studies failed to report, let alone statistically account for, any shared variance of these symptom subscales (e.g., Berman et al., 1997, Yazici et al., 2002). In one study that did report these figures (Collins et al., 1997), the Positive and Negative subscales were in fact significantly correlated ($r = 0.58$). Moreover, the relative contribution of individual items to scores on the subscales and associated variance in cognitive functions were not evaluated. This appeared to be important in at least one study (Ndlovu, Stirling, & Hellewell, 1998), in which it was observed that self-monitoring performance (indexed by tests in which examinees draw and subsequently identify designs) was worse among patients with higher Positive Subscale scores, but was unrelated to the severity of the Positive subscale item measuring hostility, even after controlling for IQ.

Furthermore, there is a growing body of research on the PANSS indicating that the Positive, Negative, and General Psychopathology Subscales may not adequately characterize the heterogeneity of schizophrenia. In one investigation, for example, PANSS data collected in a sample of “subchronic” SZCs were subjected to a factor analysis prior to evaluating the neurocognitive correlates of the negative symptoms of schizophrenia. It was noted in this study that the Negative subscale item measuring abstract thinking was associated with Verbal IQ, but not with the resultant negative factor (Vollema, Guersten, & Kuipers, 1995). Similarly, other research has indicated that the Positive and General Psychopathology subscales demonstrate only modest internal consistency. Item P2, for example, showed a non-significant item-total correlation with the Positive subscale in one study (Peralta & Cuesta, 1994). Considered together, such findings suggest that some of the items originally assigned to Positive and Negative subscales may be tapping something other than positive and negative symptoms of schizophrenia.
Indeed, it is generally agreed that a separate symptom dimension or dimensions, in addition to, and to some extent independent of, the positive and negative symptom composites are needed to account for the variance in schizophrenia symptoms (Anonymous, 2002; Arndt, Alliger, & Andreasson, 1991; Cuesta & Peralta, 1995; Dollfus & Everitt, 1998; Grube, Bilder, & Goldman, 1998; Nakaya, Juwa, & Ohmori, 1999; Peralta & Cuesta, 1995; Peralta, Cuesta, & deLeon, 1994; Smith, Mar, & Turoff, 1998). One such symptom dimension, originally identified as “Disorganization” encompasses disorganized thought and behavior (e.g., Peralta & Cuesta, 1994; Cardno & Farmer, 1995) and consistently has been linked to impairment of overall neurological functioning, sensory integration, and sequencing of complex motor activities (Arango et al., 2000; Daban et al., 2003). Disorganized symptomatology also has been shown to be related to deficits on tasks of semantic priming (Moritz et al., 2003) as well as tasks involving working memory (Barch et al., 2002). In one study, SZCs with disorganized symptoms were found to be unable to maintain context relevant information in working memory in a way that allowed for the selection of appropriate behavioral responses in a Context Processing Task (Barch et al., 2003). Given that similar cognitive deficits have been observed in alcohol challenge studies with college students (e.g., Casbon et al., 2003), it is plausible that for SZCs with Disorganized symptoms in particular, lesser amounts of alcohol may be needed to produce the alcohol-related impairments of cognitive control that contribute to disinhibited behaviors and their associated negative consequences.

Although it is generally agreed that positive, negative, and disorganized symptomatology constitute the core symptoms of schizophrenia (Bell et al., 1994b; Carpenter, Kirkpatrick, & Buchanan, 1999b; Lançon et al., 2000), the latest factor-analytic research with the PANSS suggests additional factors may be needed to account for schizophrenia heterogeneity (Mueser, Curran, & McHugo, 1997; Peralta & Cuesta, 1999). Specifically, models involving Positive (POS), Negative (NEG), Cognitive (COG), Excitement/Hostility (EXC/HOS), and Anxious/Depressed (ANX/DEP) factors have been obtained in research with diverse groups, including male and female SZCs in several different countries (e.g., U.S., France, Japan, Greece), outpatient SZC veterans (Bell et al., 1994a), and in inpatients with schizophrenia and/or schizoaffective disorder (e.g., El Yazanji et al., 2002; Fredrikson et al., 1997; Hayashi et al., 2002; Lançon et al., 2000; Lindemayer, Bernstein-Hyman, & Grochowski, 1994; Lykourous et al., 2000). Though POS, NEG, and COG were of particular interest in this study, the consistent emergence of this factor pattern in persons with schizophrenia, both with and without diagnosable affective disturbances, underscores the relevance of additional symptom
dimensions beyond the core three to schizophrenia syndromes more generally. In other words, the five-factor model seems to broadly capture the range of clinical presentations common not only to the primary *DSM-IV* subtypes of schizophrenia, but also probably to the schizoaffective disorders. See Table 1.

Table 1

*Description of Item Content Typically Attached to the PANSS Factors*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Constructs measured</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEG</td>
<td>Flattened affect, impoverished or halted speech, lack of motivation, social and emotional withdrawal</td>
</tr>
<tr>
<td>POS</td>
<td>Distortions in thought content and perception, including hallucinations, bizarre ideation, and delusions of persecution or grandiosity</td>
</tr>
<tr>
<td>COG</td>
<td>Formal thought disorder (e.g., derailment, loose association), concrete reasoning, mental confusion, impaired awareness, poor decision making, low insight, and distractibility or difficulty sustaining attention. Scores correlate with objective performance on neuropsychological tests of attention, memory, and executive cognitive functions.</td>
</tr>
<tr>
<td>ANX/DEP</td>
<td>Feelings and/or physical signs of apprehension, uncertainty, and fear; depressive outlook; emotions of guilt, shame, and remorse; preoccupation with physical malfunctioning</td>
</tr>
<tr>
<td>EXC/HOS</td>
<td>Poor management of behavioral or emotional urges resulting in impulsive expressions of anger or resentment, from oppositional defiance or belligerence to verbal or physical assaults</td>
</tr>
</tbody>
</table>

Yet, despite the apparent consistency in the symptom constructs tapped by the PANSS factors, there is continued disagreement about which and how many PANSS items load on each factor, and the few confirmatory factor analyses that have been conducted have not diminished the uncertainty much (e.g., Cuesta & Peralta, 1995; Fitzgerald et al., 2003; Mueser et al., 1997; Nakaya et al., 1999). Consequently, the precise PANSS items comprising COG have yet to be established (Bryson et al., 1999). The most common candidates seem to be P2 - Conceptual
Disorganization, N5 – Difficulty in Abstract Reasoning, GP10 - Disorientation, and GP11 – Poor Attention, on which higher scores implicate worse cognitive impairment in these areas (Bell et al., 1994a; Fredrikson et al., 1997; Kay & Sevy, 1990; Lancon et al., 2000; Lepine et al., 1989; Lépine et al., 1989; Lindenmeyer et al., 1994; Lindenmeyer et al., 1995; Mass et al., 2000; Lykourous et al., 2000; Peralta & Cuesta, 1994; Von Knorring & Lindstromm, 1995). Nonetheless, despite some variability in constituent items, the consistent emergence of a COG factor accounting for 7 to 10% unique variance in the PANSS (Lancon et al., 2000; Mass et al., 2000) across samples varying in size, DSM schizophrenia diagnosis, demographic characteristics, treatment modality, and state of illness (Bryson et al., 1999) is reassuring. Certainly, it lends support to my contention that cognitive deficits must be considered in studies examining the heterogeneity of schizophrenia and its clinical correlates – including AUD.

Additionally, relevant research suggests that COG effectively taps the cognitive deficits associated with SZC symptomatology. For example, in a sample of patients with schizophrenia and schizoaffective disorder, over one-third of the variability in COG, as identified by Bell and colleagues (1994a), could be accounted for by performance on the Wisconsin Card Sort Test (WCST), a measure of executive cognitive functions, and the Slosson Intelligence Test (Bell et al., 1994b). Likewise, in a sample of recently admitted SZC inpatients, higher scores on the COG factor (i.e., worse symptoms) were associated with poorer performance on tests of sustained attention/vigilance, encoding, and processing of shifts in modality (vision and audition) of stimulus presentation (Mass et al., 2000). In other research conducted with a large outpatient sample of non-organic SZCs, a COG factor defined by five items (P2, N5, GP4, GP11, GP12) was found to be a reliable, internally-consistent, temporally-stable index of schizophrenia-related cognitive impairment. Furthermore, COG scores in that study were associated with performance on measures of sustained attention, executive cognitive functions, verbal learning and memory, and intelligence (Bryson et al., 1999). More recently, scores on COG defined by four items (P2, N5, GP5, GP11) were found to be positively related to SZCs' performance on objective measures of attention, working memory, and achievement (Potkin et al., 2002).

Thus, on the basis of previous research, it is presently argued that COG provides a reasonable proxy for evaluating the cognitive symptoms associated with the SZC symptomatology. Making use of COG for the purposes of the present study obviates the need for more direct tests of cognitive functioning, which perhaps would entail the administration of a comprehensive neurocognitive battery. Clearly, the selection of the optimal test battery capable of measuring all pertinent cognitive constructs would prove to be a daunting task under any
circumstances. Furthermore, the time required to administer such a battery of tests would not only prove too onerous for most researchers, but also may be largely inappropriate given the limited attentional capacity of many SZCs (e.g., Barbee et al., 1989; Cornblatt & Keilp, 1994; Drake, Alterman, & Rosenberg, 1993a; Drake, Rosenberg, & Mueser, 1996; Wolford et al., 1999). One could plausibly restrict the sample to only those SZCs capable of completing the experimental procedures, but that would risk a biased pattern of results. However, a recent principal components analysis of common neuropsychological tests administered to boys at risk for substance use disorders showed that independent indices of attention, planning, abstract reasoning, foresight, judgment, self-monitoring, and motor control (all of which may be involved in critical drinking decisions) were in fact interdependent and related, with a significant portion of the variance in performance on these tasks accounted for by a single factor (Giancola et al., 1996a). In this connection, the significant correlations found between COG and several different tests of cognitive functioning could suggest that this PANSS factor is tapping the same latent cognitive construct. Hence, use of COG seems defensible.

Certainly, it is possible that several variables may account for the relationships observed between COG and objective measures of cognitive functioning, but this is precisely why multivariate consideration of variations in cognitive deficits along with other symptoms of schizophrenia is needed. Indeed, in one study, whereas neuropsychological test performance was found to be unrelated to global functioning (GAF), scores on COG were (Bell et al., 1994b), suggesting that overall severity may be an important consideration in evaluation of the clinical correlates of COG -- like AUD. Similar to the results obtained in research employing non-standardized indices of positive and negative symptomatology noted above, there is evidence that COG shares a significant proportion of variance with the POS and NEG factors obtained from factor analyses of the PANSS. For example, Bryson and colleagues (1999) found that within their outpatient SZC sample, correlations with COG ranged from \( r = 0.25 \) to \( 0.54 \) for POS, and \( r = 0.29 \) to \( 0.56 \) for NEG, depending on its constituent items. Similar bivariate correlations have been observed when P2, N5, GP10, and GP11 comprise COG among chronic SZCs \( (r = 0.43 \) POS and \( r = 0.30 \) NEG), but not with acutely relapsed SZCs. In the latter group, COG was significantly associated with POS \( (r = 0.48) \) but not NEG (Lancon et al., 2000). These results not only suggest a connection between cognitive heterogeneity and the clinical symptoms of schizophrenia, but also extend previous research in this area (e.g., Arango et al., 2000; Berman et al., 1997; Vorunganti et al., 1997) in that they reflect systematic, multivariate analysis of the relative contributions of each schizophrenia symptom to this relationship.
Such attention to the shared variance among schizophrenia symptom dimensions may be especially important when evaluating their potential relationships to AUD. For example, one study found that smoking was positively related to PANSS Negative subscale total scores. However, this apparent association with negative symptomatology could be misleading in that the simple bivariate correlations were reported between smoking and PANSS items on the Negative and General Psychopathology subscale tapping impaired cognition (e.g., attention, orientation, deficits in abstract thinking), (Patkar et al., 2002). Thus, the pathological process underlying the manifestation of smoking behavior may actually represent COG, but in the absence of multivariate analysis of symptoms, these effects cannot be disentangled. If not for this equivocation, added support for the predicted role of cognitive deficits to the development of AUD in this population could have been reasonably gleaned from another result of this study that pointed to a significant positive association between smoking and AUD. The present investigation should serve to resolve some of this ambiguity, by analyzing the unique, shared, and interactive relationships of the clinical and cognitive deficit symptoms of schizophrenia with the sorts of problems experienced by SZCs who drink alcohol.

The Present Study: Summary and Specific Aims

Preliminary evidence lends credibility to associations between the positive and negative symptom of schizophrenia and variations in certain cognitive deficits. Thus, the observation of any differential susceptibility to AUD as a function of these symptoms may be due to systematic variations in cognitive functions as they generally relate to the development of AUD. Factor analysis provides a method by which the relative contributions of each SZC symptom may be considered in analysis of broad symptom dimensions, including cognitive deficits, as they relate to AUD. Indeed, factor-analytic studies of the PANSS have invariably yielded COG (formal thought disorder, mental confusion, distractibility, concrete reasoning), which not only is associated with objective measures of cognitive functions but also appears to be moderately correlated with NEG (flat affect, alogia, social withdrawal) and POS (distortions in thought content and perception). Thus, evaluation of factor-analytically derived PANSS dimensions provides a strategy for testing, for the first time, relationships between AUD and the clinical symptoms of schizophrenia (POS, NEG) as a function of variations in cognitive deficits (COG). Although COG may not be the optimal index of schizophrenia-related cognitive impairments, it offered practical advantages for execution of the present study as a first step in examining how AUDs may map onto meaningful, empirically-derived subgroups of SZC symptoms.
In the present investigation, the relationship between AUD and SZC symptomatology was evaluated using PANSS data collected from a large, randomly selected sample of inpatients meeting *DSM-IV* (APA, 1994) criteria for either Schizophrenia or Schizoaffective Disorder upon their admission to a state psychiatric facility. Inpatients with primary diagnoses of Schizoaffective Disorder (SZA) were included in the sample not only because dual diagnosis would be analyzed with respect to symptom dimensions -- not diagnoses -- but also because research evidence points to the similarity of SZA and the subtypes of schizophrenia with regard to (a) the five-factor model of SZC symptoms as assessed by the PANSS, as noted previously, (b) specific cognitive functions, and (c) the rates and correlates of AUD diagnoses.

An exploratory factor analytic approach to the PANSS data was undertaken in the present study because although previous factor analytic studies have consistently obtained clear POS, NEG, and COG factors, their constituent items as well as the number of additional factors (zero, one, or two) has been more variable across studies, which makes the required à priori specification of factors for confirmatory analytic approaches less clear. Thus, hypotheses concerning the factor solution providing the best fit to the sample data was limited only to the prediction of clear, theoretically interpretable POS, NEG, and COG factors. However, on the basis of other factor-analytic research with similar samples, the COG factor emerging from the present analysis was expected to be minimally comprised of P2 - Conceptual Disorganization, N5 - Difficulty in Abstract Thinking, GP10 - Disorientation, and GP11 – Poor Attention, although other items that loaded significantly on this factor were included if theoretically relevant.

A subset of the factor analysis sample was then randomly selected and evaluated with the Alcohol Use Disorders Identification Test (AUDIT; Bohn et al., 2001). The results of this brief screening interview were used to validate ratings of AUD derived from independent chart reviews, which yielded a more refined analysis of AUD for the purpose of defining the key dependent variable in the present study. Once acceptable associations between scores on the AUDIT and the chart ratings were obtained, the relationship of the computed PANSS factor scores to chart ratings of AUD was evaluated using regression analyses. More specifically, given the role that deficits in cognitive functions may play in the development of AUD in this population, it was hypothesized that a significant portion of the relationship observed between AUD and the clinical dimensions of schizophrenia (POS, NEG) would be accounted for, i.e., mediated by, COG. Examination of the influence of other demographic, treatment, and diagnostic variables associated with cognitive deficits and/or co-occurring with AUD on the predicted mediational relationship was also pursued on a post-hoc basis.
METHOD

Participants

The present investigation was primarily based on archival data obtained from hospital records and a comprehensive database maintained at the Florida State Hospital (FSH) in Chattahoochee, Florida. In general, persons admitted to FSH must be at least 18 yrs old and meet legal criteria for admission as specified in Chapter 394 of the Florida Statutes (Baker Act) or as ordered by the court for evaluation. Specifically, a person may meet criteria for involuntary admission to FSH if they have been assessed as (a) dangerous to self or others (b) unable to care for him/herself adequately in the community as a result of psychiatric problems, (c) incompetent to proceed through the court system due to psychiatric problems, or (d) adjudicated as Not Guilty by Reasons of Insanity. Persons may also be admitted to FSH on a voluntary basis if they have a diagnosable mental illness, are capable of providing informed consent to treatment, and are expected to respond to such treatment. It should be noted that some persons meeting admission criterion (c) may be legally competent to consent to treatment (i.e., make informed treatment decisions) but still incompetent to proceed through the legal system. Additionally, some persons involuntarily committed under criterion (d) may be competent to consent to treatment.

At the time of this study, over 11,000 administrations of the PANSS were included in the FSH database, the culmination of data collected on admission and every six months from each “resident” on each of his/her hospitalizations. From this database, I first selected cases from currently hospitalized residents whose PANSS admission data had been collected in 1995 or later (n=770). For those residents who were admitted to FSH on more than one occasion since 1995, I selected the most recent admission for analysis (n=611). For example, if Resident A was hospitalized from 1995-1996 and then re-admitted in 1999, I selected data from the 1999 admission for the present analysis. Finally, I selected only those cases with DSM-IV (APA, 1994) primary Axis I diagnoses of Schizophrenia or SZA disorder and complete PANSS data.
Seven cases were later excluded from the analyses following initial selection and evaluation because subsequent chart review suggested ambiguous primary diagnosis of Schizophrenia or SZA disorder [e.g., unclear database entry which could not be clarified \(n=1\), secondary diagnosis of substance-induced psychosis \(n=1\) or psychosis due to medical illness \(n=1\), or the primary schizophrenia diagnosis had been modified over the course of treatment to bipolar disorder \(n=2\), polysubstance dependence \(n=1\), or malingering \(n=1\).] Another was excluded from further consideration after chart review revealed a secondary diagnosis of dementia due to alcohol \(n=1\), which might confound interpretation of the effects of interest in this investigation.

Following these exclusions, the final sample for the first phase of the study (i.e., factor analysis of the PANSS) included \(N=436\) \((n=97\) female\) residents, with an average age at current admission of 40.82 years \((SD = 10.4; \text{Range} = \{19, 68\})\). The average length of stay at PANSS administration was 8.32 days, \(SD = 3.2\), Median = 7 days, Range = 17. Seventy-one (16.3\%; \(n=36\) female) were civilly committed, but the majority were identified as forensic admissions and either adjudicated as Not Guilty by Reasons of Insanity (33.5\%; \(n=146\), 19 female) or as Incompetent to Proceed to Trial (50\%; \(n=218\), 42 female). In terms of their overall severity of functioning, GAF recorded on admission averaged 39.86 \((SD = 9.7; \text{Range} = \{15, 70\})\). Primary DSM-IV Axis I diagnoses recorded on admission were: 47.0\% SZA Disorder \((N=205; n= 63\) female\), 33.4\% Paranoid Type \((N=146; n = 23\) female\), 14.0\% Undifferentiated Type \((N=61; n=7\) female\), 4.1\% Disorganized Type \((N=18; n=2\) female\), and less than 1\% Residual Type \((N=5; 1\) female\) and Catatonic Type \((N=1)\). A substantial proportion of the sample also was diagnosed with one or more secondary Axis I (55.5\%) and/or Axis II (35.5\%) disorders on admission (see Table 2). Of particular note, 77 residents (17.6\%) received an AUD diagnosis, of whom a majority \((n=59\) or 76.6\%) were diagnosed with a concurrent drug use disorder. In total, the frequency of any substance disorder on admission was 47.7\% \((N=208)\).

Database cases included in the first phase of the investigation were assigned randomly generated numbers and subsequently identified by sequential random number selection as “potential participants” for the next phases of the investigation involving AUD evaluation. In accordance with FSH Internal Review Board requirements, participation in interview/chart review procedures was limited to residents who were (a) currently hospitalized, (b) competent to consent to treatment (verified at the time of the consent), and (c) considered to be clinically appropriate for participation by the resident’s treatment team.

Of the initial 100 randomly selected cases, 97 were identified as competent to consent to
Table 2
**Frequency of Comorbid Disorders in the Phase One Sample.**

<table>
<thead>
<tr>
<th>Axis I Disorder</th>
<th>Frequency N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anxiety Disorder</strong></td>
<td></td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>2</td>
</tr>
<tr>
<td>Post-Traumatic Stress Disorder</td>
<td>2</td>
</tr>
<tr>
<td><strong>Mood Disorder</strong></td>
<td></td>
</tr>
<tr>
<td>Major Depressive Disorder</td>
<td>2</td>
</tr>
<tr>
<td>Dysthmia</td>
<td>1</td>
</tr>
<tr>
<td>NOS</td>
<td>1</td>
</tr>
<tr>
<td><strong>Any Substance Disorder</strong></td>
<td>226 (51.8%)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>77 (17.6%)</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>2</td>
</tr>
<tr>
<td>Cannabis</td>
<td>29</td>
</tr>
<tr>
<td>Cocaine</td>
<td>23</td>
</tr>
<tr>
<td>Hallucinogen</td>
<td>1</td>
</tr>
<tr>
<td>Inhalant/PCP/Unknown</td>
<td>51</td>
</tr>
<tr>
<td>Polysubstance</td>
<td>43</td>
</tr>
<tr>
<td><strong>Secondary Schizophrenia Disorder</strong></td>
<td>31 (7.1%)</td>
</tr>
<tr>
<td>Schizophreniform</td>
<td>1</td>
</tr>
<tr>
<td>SZA</td>
<td>12</td>
</tr>
<tr>
<td>Paranoid Type</td>
<td>7</td>
</tr>
<tr>
<td>Undifferentiated Type</td>
<td>6</td>
</tr>
<tr>
<td>Psychotic Disorder, NOS</td>
<td>5</td>
</tr>
<tr>
<td><strong>Other Axis I Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Attention Deficit</td>
<td>1</td>
</tr>
<tr>
<td>Cognitive Disorder/Dementia</td>
<td>4</td>
</tr>
<tr>
<td>Impulse Control</td>
<td>1</td>
</tr>
<tr>
<td>Reading</td>
<td>1</td>
</tr>
<tr>
<td>Tourette’s Syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Pedophilia</td>
<td>2</td>
</tr>
</tbody>
</table>
Table 2 - continued

<table>
<thead>
<tr>
<th>Axis II Disorder</th>
<th>Frequency N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Developmental Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Borderline Intellectual Functioning</td>
<td>17</td>
</tr>
<tr>
<td>Mental Retardation (Mild or Unspecified)</td>
<td>26</td>
</tr>
<tr>
<td><strong>Personality disorder</strong></td>
<td>121 (27.7%)</td>
</tr>
<tr>
<td>Antisocial</td>
<td>64</td>
</tr>
<tr>
<td>Borderline</td>
<td>19</td>
</tr>
<tr>
<td>Dependent</td>
<td>1</td>
</tr>
<tr>
<td>Histrionic</td>
<td>1</td>
</tr>
<tr>
<td>Narcissistic</td>
<td>2</td>
</tr>
<tr>
<td>Obsessive Compulsive</td>
<td>2</td>
</tr>
<tr>
<td>Paranoid</td>
<td>4</td>
</tr>
<tr>
<td>Schizoid</td>
<td>4</td>
</tr>
<tr>
<td>Not Otherwise Specified</td>
<td>24</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
</tr>
<tr>
<td>Childhood Antisocial Behavior</td>
<td>1</td>
</tr>
</tbody>
</table>

treatment and hence participation in this study. In order to obtain a sample of sufficient size to detect the effects of interest (see Statistical Power below) an additional 100 cases were randomly selected from the remaining sample. Of these, 92 were identified as competent to consent to treatment. The resulting sample of 189 residents (n=47 female) constituted the initial pool of eligible participants. Of these, 10 (n=2 female) were excluded because participation was considered clinically contraindicated by their respective treatment teams.

Therefore, 179 residents of the initial randomly selected pool of 200 potential participants met all the necessary selection and approval criteria for participation in subsequent phases of the study. One female and two male residents who were identified as legally competent to consent to treatment/participation were deemed by the interviewer (MLR) to be unable to adequately convey an understanding of the purpose of the study and/or respond to the interviewer’s questions due to either acute symptoms of their mental illness or medication sedation. Thus although legally competent to consent to treatment, these three were
considered unable to provide informed consent to participate and consequently excluded from further consideration. Thirty other residents eligible for participation were discharged from the hospital before informed consent procedures could be initiated. Of the eligible participants who were approached for the study, 108 (74%; \(n=19\) female) volunteered for the entire study and signed the consent form, which included an agreement to a review of chart records. Another five residents (\(n=3\) female) consented to chart review, but declined the AUDIT interview.

Given the disproportionate number of female residents in the initial pool of eligible participants (24.5%), and with consideration of probable attrition due to clinical/legal ineligibility, discharge, and refusal, it appeared unlikely that a sample with equal numbers of male and female participants yielding adequate statistical power to test the hypotheses of interest would be obtained. Therefore, I over-sampled male participants in order to procure a sample of sufficient size should I need to test the key hypotheses with males alone. To accomplish this end, I selected 47 male cases from the database sample in order of their randomly assigned number, among those who were currently hospitalized. Of these, one was excluded because he was not competent to consent to treatment and another was competent to consent to treatment but deemed unable to do so at the time of the consent procedures. Twelve others were deemed clinically inappropriate to participate in the study by their respective treatment team and were excluded per FSH Research guidelines. Nine male residents were discharged before an interview could be scheduled. Of the remaining 25 males who were approached for the study, 19 (76%) of them offered their full consent to participation.

In total, AUDIT data were available for \(N=127\) residents (\(n=19\) female), with another five consenting to chart review. Six of these 132 residents were among the aforementioned eight excluded after primary Axis I diagnoses were clarified by chart review. Thus, data from a final sample of \(N=126\) (\(n=21\) female) were available to test the predicted relationship between AUD and SZC symptoms. See RESULTS for additional detail on the characteristics of this sample.

**Measures**

**Global Scale of Adaptive Functioning**

The Global Scale of Adaptive Functioning (GAF; APA, 1994) is a 100-point scale that provides an index of the resident’s overall functioning within the context of physical or environmental restrictions. The GAF was initially coded by the admitting psychiatrist and recorded from the aforementioned database maintained by FSH for use in this study. The GAF can be regarded as a reasonable proxy for the overall severity of a resident’s illness upon admission and was used as a covariate in the planned analyses, as discussed below.
Positive and Negative Syndrome Scale

The PANSS by Stanley R. Kay, Ph.D., Lewis A. Opler, M.D., Ph.D., and Abraham Fiszbein, Ph.D. is a 30-item symptom rating scale that was designed to measure core positive and negative symptoms of schizophrenia and related psychopathology (e.g., anxiety, depression). Symptoms are rated along a seven-point continuum of severity on the basis of a semi-structured interview with the patient as well as observations of the patient’s behavior during the past seven days as reported by hospital staff, family, or other collateral informants. The PANSS appears to be a psychometrically sound instrument in the measure of the SZCs’ overall clinical profile (Kay, Opler, & Lindenmayer, 1988, Kay, Opler, & Lindenmeyer, 1989; www.mhs.com). See Table 3 for selected item descriptions.

FSH psychology staff (doctoral or master’s level psychologists, pre-doctoral psychology interns) are subjected to extensive training on the PANSS via videotape and manual review as recommended by the authors of the instrument. Examiners are required to meet acceptable standards for inter-rater reliability (minimum 90% agreement within one rating point) prior to independent PANSS administration and are re-trained annually and/or when obvious problems with examiner drift are noted. Due to limited resources, FSH has been unable to have two examiners routinely administer the PANSS concurrently, but the instrument is straightforward and training offers a reasonable expectation of inter-rater reliability (Von Knorring & Lindstromm, 1995). Admission PANSS ratings are usually completed within the first week of the resident’s admission to the hospital and the resulting data were maintained in a comprehensive database. At FSH, the PANSS items are rated along a seven-point severity scale (1= Absent, 2=Minimal, 3=Mild, 4=Moderate, 5=Moderate-Severe, 6=Severe, 7=Extreme) on the basis of patient responses to a structured clinical interview (SCI-PANSS; Kay, 1991; Opler, Kay, Lindenmayer, & Fiszbein, 1992), behavioral observations, and reports from staff, family, or referral sources.14

Briefly, the PANSS interview proceeds in four phases. The first phase (10-15 min) is relatively unstructured and non-directive, with open-ended inquiry into the resident’s mental health history and precipitants to hospitalization. In the second (10-15 min) and third (5-10min) phases, specific areas of psychopathology are probed with increasingly focused and directive questioning techniques. In the final phase (5-10min), the full range of psychopathology is evaluated and previous ambiguous responses are clarified (Kay et al., 1987; Kay & Sevy, 1990).
Table 3

*Positive and Negative Syndrome Scale* by Stanley R. Kay, Ph.D., Lewis A. Opler, M.D., Ph.D., and Abraham Fiszbein, M.D. FSH psychology personnel rate the following items on the basis of structured interview (SCI-PANSS; Kay, 1991), behavioral observations, and reports from staff, family, or referral sources.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
<td>Minimal</td>
<td>Mild</td>
<td>Moderate</td>
<td>Moderate-Severe</td>
<td>Severe</td>
<td>Extreme</td>
</tr>
</tbody>
</table>

**Positive Scale**

P2 Conceptual disorganization

Impairments in thought processes as evidenced by an inability for goal-directed communication, e.g., tangential or circumstantial speech, loose associations, illogical speech, or thought blocking.

**Negative Scale**

N5 Difficulty in abstract thinking

Difficulty in classifying objects or concepts, generalizations and similarities. Problems are resolved using only concrete or egocentric thought processes.

**General Psychopathology Scale**

GP10 Disorientation

Unawareness or confusion about whom one is (name, birth date), where they are, or passage of time (may be due to withdrawal).

GP11 Poor attention

Attention deficits evident through easy distractibility, or difficulty sustaining attention or re-adjusting attention to new stimuli.

GP13 Disturbance of volition

Impairment in the voluntary initiation, maintenance, and control of one’s thoughts and behaviors.

GP15 Preoccupation

Fixed focus on thoughts, feelings, or other internal experiences that interferes with reality-testing and behavior.

Alcohol Use Disorders Identification Test

The AUDIT (Bohn et al., 1995; Babor et al., 2001) is a brief, two-part screening tool developed specifically for the early identification of problem drinking (see www.nih.niaaa.org) that includes a core questionnaire (Part I) and a physical examination instrument (Part II). In the present study, the ten-item questionnaire (Part I) was used to evaluate and establish the validity of independent chart ratings of AUD (see below). The AUDIT yields a total score (max 40), and three subscale scores: Hazardous Alcohol Use (consumption patterns), Dependence Symptoms, and Harmful Alcohol Use. The content of the Harmful Alcohol Use and Dependence Symptom items is similar to those found in other common self-report alcohol measures but are scored according to their frequency of occurrence in Likert format (0-4), rather than by their presence or absence (Bohn et al, 1995). A total cut-point score of eight has been shown to yield optimal separation of hazardous and harmful drinkers as well as alcohol dependent individuals from normals in general population samples (Allen et al, 1997; Babor et al., 2001) and those with severe mental illness (Dawe, Seinen, & Kavanagh, 2000). See Appendix A-1.

Although the AUDIT is certainly not an infallible measure of AUD, it possesses several features that make it a particularly desirable choice for use with the current sample. First, all items refer directly to alcohol and its effects in the past year (Bohn et al, 1995; Babor et al., 2001). Such fact-based items about the recent past are considered optimal for obtaining pertinent data from those with severe psychiatric problems (Drake, McHugo, & Diesanz, 1995). Second, AUDIT items tapping alcohol consumption patterns appear first in the inventory. This may be important because some research has shown that psychiatric patients readily admit to consumption, but may be less inclined to describe their alcohol problems (Smith & Pristach, 1990). Questions that suggest a link between alcohol use and problems may inflate estimates of AUD among SZCs with prominent paranoia who may be prone to identify correlations among variables, which are not, in reality, correlated (Brennan & Hemsley, 1984). Thus, for SZCs with paranoia, inquiries concerning use patterns without explicit or implied associations to problems may be preferred. Third, research has supported the concurrent validity of the AUDIT total score (cutoff =8) and its subscales with AUD diagnoses obtained via structured interview with SZCs, even after accounting for all possible demographic predictors (Dawe et al., 2000). Finally, the AUDIT typically takes less than ten minutes to administer, thereby better accommodating the limited attentional capacity commonly associated with schizophrenia (Barbee et al., 1989).
Clinician Rating Scale

The Clinician Rating Scale (CRS; Mueser et al., 1995a) was specifically developed to assess alcohol problems in persons with severe mental illness, as self-reports of alcohol problems are often difficult to obtain in this population. The CRS was initially designed for use by clinicians who maintain regular contact with patients over a six-month period so that data available from a variety of sources may be considered. Research has generally supported its utility in the identification of AUD (Carey, Cocco, & Simmons, 1996; Drake et al., 1996; Mueser et al., 1995a; Osher et al., 1994). The CRS was modified for use in the present study (see Appendix A-2). Specifically, instructions for the CRS were altered to better correspond with the study procedures (i.e., chart ratings) and the critical time period was extended to one-year, rather than six-months, to correspond with the independent measure of AUD, the AUDIT. Chart ratings based on the CRS constituted the present measure of AUD and served as the primary dependent variable in the planned regression analyses.

Procedures

The present study was conducted in three phases. First, I examined the factor-structure of the PANSS using data previously collected from a sample of currently hospitalized residents with DSM-IV (APA, 1994) diagnoses of schizophrenia or SZA disorder upon their most recent admission to FSH. A subset of this sample was then randomly selected for evaluation of AUD. Specifically, residents offering their voluntary consent to participate in the study were administered the AUDIT (phase 2) in order to establish the validity of independent chart ratings of AUD (phase 3), which served as the dependent variable in the planned analyses.

Informed Consent and AUDIT Interview

Appointments were arranged to meet individually with eligible participants on their respective hospital unit/ward so as not to interfere with other scheduled therapy activities. Next, via the attending psychologist, I sought the approval of the treatment team before approaching a resident for participation in this study. After reviewing the informed consent form (see Appendix B-1), the resident’s ability to comprehend the purpose, advantages, and disadvantages of the study was assessed and recorded (see Appendix B-2). The resident was permitted to ask questions and any misunderstandings concerning the study were addressed by the examiner. Following completion of the informed consent procedures, residents volunteering for the study were administered the AUDIT via interview, using procedures outlined by Babor and colleagues (2001). Residents were given a certificate of participation (Appendix B-3), regardless of whether or not they completed the interview.
AUD Chart Ratings

In the third and final phase of the investigation, four pre-doctoral psychology graduate students who were employed by FSH via contract with Florida State University independently evaluated charts maintained on residents providing their informed consent to participation in the study. Chart review procedures were initiated within one month of the conclusion of phase two of the investigation, with cases assigned to teams of two raters according to the resident’s hospital unit/ward.15 Raters were blind to residents’ scores on the AUDIT.

Prior to evaluation of the chart records, each rater was trained on the CRS borrowing from procedures recommended by the authors of this instrument (Mueser et al., 1995b). Raters first reviewed sections of the DSM-IV (APA, 1994) pertaining to substance use disorders and schizophrenia. Each rater also read and studied a summary of the existing literature on the assessment of comorbid AUD among persons with schizophrenia (Reardon & Lang, 2001). Group discussions were held to ensure sufficient understanding of this background material, to correct any misconceptions, and to highlight its relevance to the current research. Next, each rater reviewed sections of Mueser and colleagues (1995) Toolkit for Evaluating Substance Abuse in Persons with Severe Mental Illnesses pertaining to the evaluation of AUD using the CRS. In a subsequent meeting, the CRS was reviewed and case vignettes were discussed in order to illustrate key concepts and to provide raters with practice applying the CRS.

The CRS was adopted as an alternate means to the assessment of comorbid AUD not only because it allowed for a more refined analysis of AUD severity, but also because reliance on AUD diagnoses recorded in the database seemed questionable based on research indicating pervasive problems with reliable diagnosis of AUD by personnel overseeing inpatient psychiatric populations (e.g., Anath et al., 1989; Barbee et al., 1989; Blanchard et al., 2000; Goethe & Ahmadi, 1991; Kirchner et al., 1998). Furthermore, the DSM-IV criteria for AUD are generally understood to involve high levels of consumption and the consequences of alcohol use typical of the general population, but it appears that the negative consequences of alcohol use in SZC population can emerge with lesser consumption than in the average drinker and may lead to different sorts of alcohol problems than those commonly observed in the general population (cf. Drake et al., 1989; Bartels et al., 1995). Accordingly, in addition to applying DSM-IV (APA, 1994) AUD criteria, raters were instructed to consider the special consequences of substance disorders observed in persons with severe mental illnesses as outlined by Mueser et al (1995) when evaluating for AUD during the year prior to residents’ current admission (see Table 4).
<table>
<thead>
<tr>
<th>Consequence</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Housing instability</td>
<td>Evicted from dwelling due to alcohol; homelessness</td>
</tr>
<tr>
<td>Symptom relapse unrelated to stressors</td>
<td>Worsening of psychotic symptoms</td>
</tr>
<tr>
<td>Treatment non-compliance</td>
<td>Medication non-compliance, failure to attend clinic appointments</td>
</tr>
<tr>
<td>Violent behavior/threats of violence</td>
<td>Fights, throwing things, foul language (when intoxicated)</td>
</tr>
<tr>
<td>Sudden, unexplained mood shifts</td>
<td>Depression, anger, euphoria, anxiety, expansive mood</td>
</tr>
<tr>
<td>Suicide ideation or attempts</td>
<td>Thoughts or talk about self-harm or suicide; thinking of death or plans to self-harm</td>
</tr>
<tr>
<td>Cognitive Impairment a</td>
<td>Confusion, memory deficits, or poor planning unrelated to relapse of schizophrenia</td>
</tr>
<tr>
<td>Difficulty budgeting funds</td>
<td>Often attempts to borrow or steal money, or pawns own or others possessions</td>
</tr>
<tr>
<td>Prostitution</td>
<td>Exchanging sex for money or material goods (food, clothing, or alcohol)</td>
</tr>
<tr>
<td>Social Isolation</td>
<td>Social withdrawal/avoidance</td>
</tr>
<tr>
<td>Social Difficulties</td>
<td>Frequent arguments/fights with significant others (family, friends, spouse)</td>
</tr>
<tr>
<td>Employment Difficulties</td>
<td>Often tardy or absent, arguments with co-workers, docked pay, job loss</td>
</tr>
<tr>
<td>Hygiene and health problems</td>
<td>Poor personal hygiene/grooming, medical complications, weight loss</td>
</tr>
<tr>
<td>Legal problems</td>
<td>Disorderly conduct, Driving While Intoxicated, shoplifting, etc.</td>
</tr>
</tbody>
</table>

Note. Adapted from the *Toolkit for Evaluating Substance Abuse in Persons with Severe Mental Illness* by Mueser et al., 1995a.

a This consequence alone was not used in the present study for making ratings of AUD
Of course, it has been argued that almost any use of alcohol by persons with schizophrenia may be associated with significant negative consequences; however, the CRS allowed for the important distinction between what appeared to be uncomplicated use of alcohol (2 – “Use without Impairment”) and alcohol use that was clearly linked to negative outcomes. Thus, although chart raters were attentive to the unique problems associated with alcohol use in this population, the results of the planned analyses may be generally interpreted with respect to alcohol problems, without undue prejudice against simple use of alcohol by persons with schizophrenia. Other research has supported the reliability/validity of using patient records to assess alcohol problems (Mansson et al., 1993), and in the present study, data pertinent to CRS-AUD ratings were extrapolated from self-report, admission laboratory tests, psychiatrist diagnoses, and collateral reports (referral information, legal, family reports) as noted in the chart progress notes, treatment plan, and discharge summary. Demographic, diagnostic, and treatment data were also coded from the consenting residents’ charts (Appendix A-3) in order to evaluate their influence on the association of AUD with the observed PANSS factors. Raters were instructed to limit chart review to 30 minutes. Semi-monthly meetings were held with the primary investigator to ensure adherence to these criteria and guard against drift.

**Data Analysis Strategy**

**Statistical Power**

Several approaches have been used to determine the optimal sample size for factor analytic procedures, with most based on participants-to-variable ratios (see Floyd & Widaman, 1995). As a general rule of thumb, however, a sample of at least 300 cases is needed for factor analysis (Tabachnik & Fidell, 2001). The present sample of \(N=446\) cases falls within the Good to Very Good range, as identified by Comrey and Lee (1992).

Using tables provided by Cohen (1992) and Cohen & Cohen (1988), 70 participants are needed to detect an estimated large effect size (ES = 0.35) of the model \(R^2\) at an alpha of 0.05 with eight partially correlated “predictor” variables (PANSS factors, age, gender, GAF). A minimum sample size of 100 is required for sufficient power to detect a medium effect size (ES = 0.08) for the primary effect of interest, (i.e. mediation of POS/NEG by COG on AUD). The present sample of \(N=126\) provided more than 90% power to detect the overall model effect and over 85% power to test the predicted mediation effect (Cohen, 1992; Cohen & Cohen, 1988).

**Exploratory Factor Analysis of the PANSS**

An exploratory Principal Components Analysis (PCA) was conducted with the thirty PANSS items. The present study utilized the following criteria for factor extraction: (a) Kaiser
Criterion (eigen-values > 1), (b) Cattell’s (1966) Scree test (a graphical plot of the eigen-values should reveal a place where the decrease of eigen-values appears to level off to the right of the plot, and no more than the number of components to the left of this point are retained), (c) internal consistency, and (d) theoretical interpretability. Applying recommendations outlined in Tabachnik & Fidell (2001), PANSS items with loadings greater than, or equal to, 0.32 were retained in the final solution. Items with component loadings less than 0.32 were not considered in subsequent analyses, unless there was a strong theoretical rationale for retention.

Factors were rotated using the oblique (Promax) method. This approach allows for inter-correlated factors, which may better characterize psychological phenomena insofar as the majority of psychiatric patients present with several co-occurring symptoms (Mueser et al., 1997; Peralta & Cuesta, 1999). This approach also allows for factor score intercorrelations, which is important because a moderate degree of shared variance among factor scores is needed to test the predicted mediational effect of COG on POS/NEG. Factor scores were computed using the Bartlett Method, which is similar to the more commonly used Regression method, but allows for unbiased factor scores that correlate only with their own factors.

Reliability and Validity of CRS-AUD Classifications

The inter-rater reliability of the CRS-AUD ratings obtained for each participant was evaluated by (a) the percent agreement between raters for each category on the CRS, and (b) the Kappa statistic, which measures the pairwise agreement among raters making category judgments corrected for expected chance agreement (Carletta, 1996).

Correspondence between self-reported alcohol problems on the AUDIT (i.e., total score, subscale scores, and cutoff classifications) and CRS-AUD classifications obtained from chart review were evaluated using non-parametric statistics.

Prediction of Alcohol Use Disorders by the PANSS

A hierarchical logistic regression analysis, with CRS-AUD as the dependent variable (1=problem alcohol use, 0= non-problem alcohol use or abstinence) was conducted. Because young age and male gender are commonly associated with AUD (e.g., Cuffel & Chase, 1994; Drake & Wallach, 1993; Mueser et al., 1990; Mueser et al., 1992b), variance in AUD attributable to these demographics was controlled by entering these variables in step one of the equation, along with GAF (index of overall severity). Inclusion of age in these analyses becomes particularly critical if alcohol problems can be attributed simply to greater years of opportunity. In this sense, age can be considered a proxy for duration of alcohol use, which may be related both to cognitive impairment and severity of AUD, and thus important to statistically control.
Inclusion of gender also was considered important because female SZCs have been shown to have better premorbid functioning than males (Blanchard et al., 2000) and higher rates of AUD have been observed among males in general and psychiatric populations (APA, 1994; Kandel et al., 1997; Mueser et al., 1990). PANSS factors other than POS, NEG, and COG were also entered in step one of the model to account for variance in AUD attributable to psychological symptoms of other comorbid disorders and/or a diagnosed affective episodes (i.e., SZA).

We also examined the bivariate associations of other pertinent individual difference variables with the key constructs of interest (i.e., COG, AUD) with significant associations ($p<.05$) used to determine whether the variable should be tested as a covariate (entered in step one) in the planned regression analyses. Some of the individual difference variables tested were: ethnicity, education, commitment status, primary diagnosis, number and type of comorbid conditions (e.g., history of brain injury, seizure disorder, drug disorder, antisocial personality, developmental disorder), medicated at time of admission/medication change, medication type, age at first hospitalization, age of AUD onset, number and duration of previous hospitalizations, duration of current hospitalization. We also examined the impact of methodological variables on the study results, such as method of PANSS administration, PANSS rater staff position, inter-rater discrepancies, etc. Only variables which entered the equation as significant covariates (i.e., $p<.05$) were retained in the final model. Assessment of the significance of beta coefficients (independent variables) was pursued only if the overall model effect was significant.

Four steps are required to test the predicted mediational effect of COG on the relationship between POS/NEG and AUD (Baron & Kenny, 1986; Kenny, Kashy, & Bogler, 1998). First, the significance of the correlations between POS/NEG and COG (the “mediator”) were evaluated. Second, the significance of the unique and shared contributions of POS and NEG to AUD was evaluated by entering these variables in step two of the regression analysis. Third, the association of AUD with COG, controlling for POS and/or NEG, was evaluated by entering COG in step three of the regression equation. The fourth step involves the evaluation for evidence of either complete or partial mediation. In the case of complete mediation, the relationship between POS/NEG and AUD is completely removed when COG is controlled. In other words, entering COG into the regression equation results in a statistically significant change in the POS/NEG beta-coefficients (observed in step two), reducing these effects to non-significance. Such a result would suggest that the relationship between POS/NEG and AUD is a spurious one, entirely accounted for by COG. Alternately, an actual, but indirect, relationship between POS/NEG and AUD may exist by way of COG. Such partial mediation is indicated
when the first three steps above are satisfied, but the fourth is not (Joiner, personal communication April 11, 2002; Kenny et al., 1998).

**Supplemental Analyses**

In order to evaluate the magnitude of any indirect effects of POS/NEG through COG on AUD, Structural Equation Modeling (SEM) techniques were applied to a scaled-down model that included the key constructs of interest. Additionally, the potential moderating effects of COG on the relationship of AUD with POS and NEG were assessed on a post-hoc basis by evaluating the significance of interaction terms (i.e., POS X COG, NEG X COG) entered in the hierarchical regression analyses. The significance of these interactions would suggest that degree of impact of COG on AUD depends on the severity of POS and/or NEG.
RESULTS

Preliminary Analyses

Data entry checks were performed on all cases for the following variables: Date of Admission, PANSS/MCAS Administration Date, Length of Stay at PANSS Administration, Length of Stay at Consent,\(^{17}\) Method of PANSS Administration (interview and observation vs. observation only), Age,\(^{18}\) Gender, Primary Diagnosis, PANSS items, and AUDIT total and subscale scores. Data recorded from code sheets (see Appendix A-3) and the AUDIT interview forms were also examined for accuracy in a random subset of twenty-five cases (20%), with an additional 10% of cases checked for recording of Type of Current Medication (i.e., antipsychotics, mood stabilizers, anxiolytics, etc.). If data entry errors were found for any particular variable via these random checks, data recorded for this variable were subsequently verified in the entire sample.\(^{19}\) All data entries pertaining to AUD-related variables (i.e., CRS, patterns of consumption, etc.) were examined for accuracy, and no entry errors were found.

Next, the distributions of all variables were examined for possible violations of the assumptions of the planned analyses. Univariate outliers were identified by the median-interquartile method and reined in by substituting the original values with the median +/- two interquartile ranges (Tabachnick & Fidell, 2001). After applying these corrections, variable distributions were evaluated for violations of the assumptions of normality, linearity, and/or homoscedasticity via review of skewness/kurtosis statistics, tests of normality (i.e., Kolmogorov-Smirnov), normal probability plots, and histograms. Further information in this connection is provided below where relevant to the analyses described.

Factor Analysis of PANSS

Assumptions of Exploratory Factor Analysis

In the Phase One sample (\(N=436\)), a total of 273 univariate outliers were identified across the 30 PANSS items and reined in via the median-interquartile method (\(P4 = 7, P7 = 5, N1 = 38, N2 = 27, N3 = 38, N4 = 38, GP2 = 3, GP4= 12, GP6 = 1, GP7 =6, GP8 = 5, GP9 = 28,\)
GP10 = 16, GP13 = 4, GP14 = 5, GP15 = 7, GP16 = 6). Following these corrections, no further violations of the assumptions of normality, homoscedacity, or linearity were observed.

Given the confirmed univariate normality for each of the PANSS items, the possibility of a curvilinear relationship among pairs of items was considered unlikely. Obviously, with 30 variables, visual inspection of all pairwise scatterplots to check for deviations from multivariate normality would be impractical (approximately 900 plots); however, a review of several bivariate scatterplots, though suggestive of the presence of some multivariate outliers, was not indicative of curvilinear relationships among variables that would violate the assumption of multivariate normality (required for statistical inferences about the number of components extracted).

Multivariate outliers were identified by performing a series of multiple linear regressions in which each PANSS item was regressed on all other PANNS items, and examining the significance of Mahalanobis Distance values ($\chi^2(30) = 59.7, p<.001$) obtained from each model (Tabachnik & Fidell, 2001). A total of 12 multivariate outliers were identified using this method. PCA’s performed both with and without these cases yielded the same pattern of component loadings; therefore, for simplicity, only the more inclusive analyses are reported below.20

Examination of the bivariate correlations among pairs of PANSS items indicated that a substantial number of them exceeded 0.30, a finding which supports the factorability of the PANSS in the present sample. Furthermore, the Bartlett Test for the PCA was significant, $\chi^2(435) = 8070.28, p<.001$, indicating that the correlations in the matrix were significantly different from zero. Finally, the Kaiser-Meyer-Olkin (KMO) statistic, a measure of sampling adequacy, was 0.93. This value far exceeds the recommended cutoff of 0.60 for a satisfactory factor analysis to proceed (Tabachnik & Fidell, 2001).

Descriptive statistics, including bivariate correlations, means, and standard deviations for each of the PANSS items are provided in Appendix C-1.

Factor Analysis Results

Principal Components Analysis (PCA) and Principal Axis Factoring (PAF) with oblique rotation (PROMAX) was performed through SPSS (v. 11.5) using the 30 items of the PANSS for the sample of 436 residents. As is commonly observed, PCA and PAF yielded a highly similar pattern of results, suggesting that measurement error did not significantly contribute to the present solution. For simplicity, and because PCA is the preferred method of analysis when factor scores are to be used for subsequent analyses (Tabachnik & Fidell, 2001), only the PCA results are presented here.
Although there was not a particularly clear scree (Eigenvalues = 11.29, 2.99, 2.21, 1.48 and 1.20), PCA yielded five components with eigenvalues greater than one (Kaiser Criterion). The rotated loadings of variables on components, sums of squared loadings (SSL), and un-rotated percentage of variance explained are shown in Table 5. Loadings noted in bold represent “on-factor” loadings, whereas those in regular font represent “off-factor” loadings. All initially extracted communalities were high (greater than 0.50); however, further examination of the Pattern Matrix of component loadings indicated that item N7 failed to load significantly (i.e., >0.32) on any extracted component. Therefore, this item was dropped and the PCA was repeated. The results of this subsequent analysis are presented in Table 5 in parentheses. Although the general pattern of loadings was highly similar to the first analysis, originally extracted components II and IV yielded slightly lower SSLs after N7 was dropped -- hence the difference in order of these factors. Following removal of N7, each component appeared non-trivial (i.e., accounted for a significant proportion of the variance) and well-defined by the items. As can be seen in Table 5, “on-factor” loadings were higher than “off-factor” loadings and only a few items (e.g., P2, P3, P4, P6, N6) showed relatively high communalities with other components. The average loading for “on-factor” items was 0.67, whereas the average loadings for “off-factor” items was just 0.04. Cronbach’s Alpha statistics, which were computed on the basis of “on-factor” items, supported the internal reliability of each of the components for use as independent variables in the present research (I = 0.91, II = 0.87, III = 0.83, IV = 0.76, V = 0.83). Application of orthogonal (Varimax) rotation yielded an identical factor pattern and highly similar factor loadings.21

Additionally, each of the components was readily interpretable. An item was included in the interpretation of a component on the basis of its highest loading across the five components. Following removal of item N7, the first extracted component appeared consistent with the expected NEG dimension. It captured the characteristic negative symptoms of schizophrenia, such as blunted affect, passive/active emotional/social withdrawal, poor rapport, lack of conversational flow, and motor retardation. The second component was identified as COG and involved symptoms of conceptual disorganization, abstract thinking deficits, disorientation, poor attention, poor judgment/insight, volitional disturbance, preoccupation, and odd mannerisms/posturing. The third component tapped hallucinations, delusions (general, persecutory, grandiosity), and unusual thought content consistent with a POS dimension. The content of the fourth and fifth components was comparable to the ANX/DEP and EXC/HOS factors obtained in other research (see INTRODUCTION, Table 1).
Table 5
Principal Components Analysis Results: Rotated Factor Loadings

<table>
<thead>
<tr>
<th></th>
<th>Factor I (I)</th>
<th>Factor III (II)</th>
<th>Factor II (III)</th>
<th>Factor V (IV)</th>
<th>Factor IV (V)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>.127 (.138)</td>
<td>-.144 (-.137)</td>
<td><strong>.979 (.963)</strong></td>
<td>-.048 (-.044)</td>
<td>-.075 (-.059)</td>
</tr>
<tr>
<td>P2</td>
<td>.010 (.020)</td>
<td><strong>.550 (.548)</strong></td>
<td>.423 (.421)</td>
<td>-.121 (-.121)</td>
<td>-.018 (-.010)</td>
</tr>
<tr>
<td>P3</td>
<td>.050 (.060)</td>
<td>.327 (.313)</td>
<td><strong>.525 (.521)</strong></td>
<td>.212 (.206)</td>
<td>-.363 (-.339)</td>
</tr>
<tr>
<td>P4</td>
<td>-.429 (-.416)</td>
<td>.338 (.347)</td>
<td>.197 (.197)</td>
<td>.010 (.015)</td>
<td><strong>.516 (.500)</strong></td>
</tr>
<tr>
<td>P5</td>
<td>-.150 (-.139)</td>
<td>-.121 (-.110)</td>
<td><strong>.828 (.813)</strong></td>
<td>-.042 (-.037)</td>
<td>.081 (.087)</td>
</tr>
<tr>
<td>P6</td>
<td>.162 (.170)</td>
<td>-.275 (-.258)</td>
<td><strong>.604 (.594)</strong></td>
<td>.124 (.131)</td>
<td>.338 (.335)</td>
</tr>
<tr>
<td>P7</td>
<td>.101 (.116)</td>
<td>-.171 (-.161)</td>
<td>.072 (.084)</td>
<td>.003 (.006)</td>
<td><strong>.894 (.883)</strong></td>
</tr>
<tr>
<td>N1</td>
<td><strong>.853 (.845)</strong></td>
<td>.008 (.007)</td>
<td>.032 (.036)</td>
<td>.051 (.051)</td>
<td>-.192 (-.178)</td>
</tr>
<tr>
<td>N2</td>
<td><strong>.868 (.861)</strong></td>
<td>.009 (.014)</td>
<td>.116 (.120)</td>
<td>-.022 (-.020)</td>
<td>-.053 (-.042)</td>
</tr>
<tr>
<td>N3</td>
<td><strong>.634 (.636)</strong></td>
<td>.097 (.102)</td>
<td>.085 (.094)</td>
<td>-.167 (-.166)</td>
<td>.299 (.305)</td>
</tr>
<tr>
<td>N4</td>
<td><strong>.836 (.830)</strong></td>
<td>.035 (.041)</td>
<td>.089 (.093)</td>
<td>-.082 (-.079)</td>
<td>.043 (.051)</td>
</tr>
<tr>
<td>N5</td>
<td>.163 (.160)</td>
<td><strong>.796 (.795)</strong></td>
<td>-.143 (-.140)</td>
<td>-.141 (-.139)</td>
<td>-.054 (-.059)</td>
</tr>
<tr>
<td>N6</td>
<td><strong>.647 (.639)</strong></td>
<td>.451 (.452)</td>
<td>-.222 (-.214)</td>
<td>-.071 (.069)</td>
<td>-.027 (-.026)</td>
</tr>
<tr>
<td>N7</td>
<td>.224</td>
<td>.139</td>
<td>.290</td>
<td>.047</td>
<td>.287</td>
</tr>
<tr>
<td>GP1</td>
<td>-.194 (-.192)</td>
<td>.024 (.033)</td>
<td>.179 (.173)</td>
<td><strong>.650 (.653)</strong></td>
<td>.020 (.012)</td>
</tr>
<tr>
<td>GP2</td>
<td>-.109 (-.107)</td>
<td>-.054 (-.047)</td>
<td>.018 (.018)</td>
<td><strong>.809 (.810)</strong></td>
<td>.111 (.104)</td>
</tr>
<tr>
<td>GP3</td>
<td>.057 (.061)</td>
<td>.022 (.013)</td>
<td>-.004 (.001)</td>
<td><strong>.739 (.732)</strong></td>
<td>-.120 (-.110)</td>
</tr>
<tr>
<td>GP4</td>
<td>.066 (.069)</td>
<td>.030 (.034)</td>
<td>.139 (.140)</td>
<td><strong>.537 (.537)</strong></td>
<td>.039 (.041)</td>
</tr>
<tr>
<td>GP5</td>
<td>.217 (.220)</td>
<td><strong>.394 (.392)</strong></td>
<td>-.026 (-.018)</td>
<td>.250 (.249)</td>
<td>.095 (.097)</td>
</tr>
<tr>
<td>GP6</td>
<td>.290 (.287)</td>
<td>-.241 (-.238)</td>
<td>-.194 (-.187)</td>
<td><strong>.723 (.722)</strong></td>
<td>.119 (.117)</td>
</tr>
<tr>
<td>GP7</td>
<td><strong>.667 (.656)</strong></td>
<td>.076 (.081)</td>
<td>-.195 (-.190)</td>
<td>.281 (.283)</td>
<td>-.065 (-.065)</td>
</tr>
<tr>
<td>GP8</td>
<td>.179 (.190)</td>
<td>-.065 (-.057)</td>
<td>-.090 (-.075)</td>
<td>-.059 (-.056)</td>
<td><strong>.870 (.858)</strong></td>
</tr>
<tr>
<td>GP9</td>
<td>-.024 (-.011)</td>
<td>.143 (.145)</td>
<td><strong>.836 (.823)</strong></td>
<td>.099 (.100)</td>
<td>-.137 (-.122)</td>
</tr>
<tr>
<td>GP10</td>
<td>.099 (.102)</td>
<td><strong>.818 (.809)</strong></td>
<td>-.093 (-.085)</td>
<td>-.015 (-.018)</td>
<td>-.086 (-.083)</td>
</tr>
</tbody>
</table>
Table 5 - continued

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>GP11</td>
<td>.022 (.029)</td>
<td><strong>.728 (.727)</strong></td>
<td>-.020 (-.012)</td>
<td>-.101 (-.101)</td>
<td>.233 (.228)</td>
</tr>
<tr>
<td>GP12</td>
<td>.109 (.112)</td>
<td><strong>.354 (.366)</strong></td>
<td>.303 (.298)</td>
<td>-.143 (-.136)</td>
<td>.192 (.185)</td>
</tr>
<tr>
<td>GP13</td>
<td>.128 (.124)</td>
<td><strong>.561 (.570)</strong></td>
<td>-.012 (-.014)</td>
<td>.235 (.241)</td>
<td>.027 (.015)</td>
</tr>
<tr>
<td>GP14</td>
<td>-.251 (-.235)</td>
<td>.259 (.262)</td>
<td>-.212 (-.194)</td>
<td>.176 (.176)</td>
<td><strong>.794 (.779)</strong></td>
</tr>
<tr>
<td>GP15</td>
<td>.061 (.059)</td>
<td><strong>.392 (.409)</strong></td>
<td>.132 (.127)</td>
<td>.151 (.160)</td>
<td>.201 (.185)</td>
</tr>
<tr>
<td>GP16</td>
<td><strong>.695 (.692)</strong></td>
<td>.062 (.069)</td>
<td>.075 (.081)</td>
<td>.002 (.004)</td>
<td>.141 (.146)</td>
</tr>
<tr>
<td>% Variance&lt;sup&gt;a&lt;/sup&gt;</td>
<td>37.65 (37.14)</td>
<td>7.37 (10.28)</td>
<td>9.98 (7.62)</td>
<td>3.99 (5.11)</td>
<td>4.94 (4.11)</td>
</tr>
<tr>
<td>SSL&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.59 (4.47)</td>
<td>3.64 (3.62)</td>
<td>3.69 (3.49)</td>
<td>2.87 (2.87)</td>
<td>3.16 (2.96)</td>
</tr>
</tbody>
</table>

Note. Pattern matrix component loadings for PCA with oblique (PROMAX) rotation. Loadings in parentheses reflect results of extraction after item N7 was deleted.
<sup>a</sup> Variance based on un-rotated SSL divided by number of items in model
<sup>b</sup> These figures were based on pattern matrix loadings

Examination of the PCA Solution<sup>22</sup>

Confirmatory Factor Analysis (CFA) with Maximum Likelihood estimation was applied to evaluate how well the five-factor model reproduced the observed data. Items were allowed to load only on their respective factors, as noted in bold font in Table 5, and constrained from loading on other factors. Inter-correlations among the factors were set free. Although the five-factor model effectively converged to a final solution, its fit to the observed data was not as strong as expected: Null Model $\chi^2$ (406, N=436) = 23900.22; tested model, $\chi^2$ (367, N=436) = 1835.45, $p<.001$, RMSEA = 0.096 (90% Confidence Interval = {0.092, 0.10}). However, the chi-square difference test clearly indicated significant improvement in fit of the current five-factor model relative to the independence (null) model, $\Delta \chi^2(39) = 22064.77$, $p<.001$. Furthermore, standard fit indices, which are less sensitive to sample size, were generally adequate: CFI = 0.94, NNFI = 0.94, AGFI = 0.73. The average squared multiple correlations of the items with their respective factors was 0.52 ($SD = 0.14$; Range {0.34-0.78}), suggesting that items were fair indicators of their respective components.
Factor Score Computation

Oblique rotated factor scores for each component were computed via the Bartlett Method, using results obtained from the second PCA with item N7 deleted. Squared multiple correlations (SMC) of “on-factor” items with their respective component scores were generally consistent with those obtained in the above CFA. With the internal consistency of the components adequately established, each component can be considered a reasonably reliable indicator of the construct measured and thus suitable for application in further analyses. In the present study, the component scores served as independent variables in the planned analyses (see below). Inter-correlations among component scores are presented in Table 6.

Table 6
Factor Score Correlation Matrix

<table>
<thead>
<tr>
<th></th>
<th>NEG</th>
<th>COG</th>
<th>POS</th>
<th>ANX/DEP</th>
<th>EXC/HOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEG</td>
<td>0.648</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COG</td>
<td>0.514**</td>
<td>0.506</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td>0.306**</td>
<td>0.516**</td>
<td>0.598</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ANX/DEP</td>
<td>0.338**</td>
<td>0.310**</td>
<td>0.353**</td>
<td>0.505</td>
<td></td>
</tr>
<tr>
<td>EXC/HOS</td>
<td>0.327**</td>
<td>0.510**</td>
<td>0.537**</td>
<td>0.359**</td>
<td>0.633</td>
</tr>
</tbody>
</table>

Note. Component scores were computed via the Bartlett Method based on PCA with oblique (PROMAX) rotation. Diagonal elements reflect the average SMCs of the “on-factor” items with their respective component.

** p<.01

Checks on Participant Selection and AUD Evaluation

Assessment of Selection Bias

As detailed in Methods above, residents selected for the factor analysis of the PANSS may have been excluded from further participation in subsequent phases of the study via random selection, if incompetent to consent treatment, or if deemed clinically inappropriate by his/her treatment team. Other eligible residents were not evaluated for AUD because they either declined participation or were discharged before an interview could be arranged. Therefore, it was important to establish that selection/exclusion procedures did not introduce
any systematic bias with regard to the PANSS factors and/or AUD, which would hinder the interpretation of the key analyses of the relationship of SZC symptoms to alcohol problems.

Although men were oversampled (see Method), the gender distribution among initially randomly selected residents (80.1% male) and those not selected (74.9% male) were not significantly different, $\chi^2(1) = 1.69$, n.s. However, the initial pool of selected residents was slightly younger ($M = 39.76$ yrs, $SD = 10.1$) than those not selected ($M = 42.13$ yrs, $SD = 10.7$), $t(434) = 2.38$, $p < .05$. No significant differences were observed either for commitment status, overall severity (GAF) or for any of the PANSS factor scores, $p$'s > .10.

Compared to initially selected residents who were legally competent to consent to treatment (and hence participation in this study), those identified as incompetent ($n=11$) were more likely to be civilly committed (10% vs. 73%), $\chi^2(1) = 33.81$, $p < .001$. They also showed worse overall functioning (GAF: $M = 33.18$, $SD = 7.17$ vs. $M = 39.88$, $SD = 10.3$) as well as more severe NEG ($M = 0.73$, $SD = 1.0$ vs. $M = -.07$, $SD = 1.0$) and COG symptoms ($M = 0.63$, $SD = 1.2$ vs. $M = -.04$, $SD = 1.0$), $t(174) = 2.12$, $p < .05$, $-2.45$, $p < .05$, and $-2.11$, $p < .05$, respectively, compared to those competent to consent to treatment. No other differences in relative gender proportions, age, or other PANSS factors were observed, $p$'s > .10. Additionally, there were no significant differences among those who were legally competent to consent but deemed unable to volunteer at the time of evaluation ($n=4$) relative to those considered able to do so if they so chose, $p$'s > .10. Of course, given the small number of those appearing unable to provide informed consent, power to adequately test for these differences was poor.

Residents excluded from the study by their treatment team for clinical reasons showed worse COG ($M = 0.48$, $SD = 1.1$) and EXC/HOS ($M = 0.49$, $SD = 1.2$) scores than residents considered eligible to approach for participation ($M = -.04$, $SD = 1.0$ and $M = -.02$, $SD = 1.0$), $t(185) = -2.25$, $p < .05$ and $-2.21$, $p < .05$, respectively. No other differences in commitment status, age, gender proportions, or other PANSS factors were observed, $p$'s > .10 between clinically excluded and clinically eligible residents.

No significant differences in GAF, commitment status, gender proportions, or PANSS factor scores were observed between eligible residents who were currently hospitalized and those subsequently lost to discharge, nor between volunteers and those refusing to participate.

In terms of FSH diagnoses of AUD, the initial randomly selected sample was statistically as likely to have been diagnosed on admission with AUD (28.6%) as those not selected (19.9%), $\chi^2(1) = 3.70$, n.s. Moreover, statistically comparable proportions of AUD diagnosis were found among those participating in the informed consent procedures relative to those
excluded due to incompetence and/or inability to consent to treatment, clinically inappropriateness, or discharge. Relative to volunteers, residents who refused participation were statistically as likely to have been assigned an AUD diagnosis by hospital staff, $p > .05$. Of course, comparisons based on staff recorded *DSM-IV* diagnoses of AUD should be interpreted with caution, not only because of the well-documented problems with reliable diagnosis in hospital settings (e.g., Anath et al., 1989; Barbee et al., 1989; Blanchard et al., 2000; Goethe & Ahmadi, 1991; Kirchner et al., 1998) but also because coding of AUD diagnosis in the present study was dependant, in part, on participation. Specifically, FSH staff identified AUD was elaborated by chart review for participating residents, but based solely on admission diagnosis for residents unselected or systematically excluded from participation.

**Reliability of Chart Ratings**

Twenty six residents were discharged and thirteen others were transferred to another unit before two independent chart assessments could be completed. The relative proportions of discharges and transfers differed significantly across the two teams of raters (8 vs. 18 charts and 11 vs. two charts, respectively), $\chi^2(2) = 10.07$, $p<.001$, though both teams completed a comparable number of ratings for continuously hospitalized residents on their original units (44 and 43). These data may be relevant, because in certain cases of discharge, charts are reduced in preparation for storage. Furthermore, a within-hospital transfer may extend the delay between ratings within the same team if one of the raters was required to wait until the resident’s new location could be established. Indeed, CRS ratings of AUD completed on currently hospitalized residents were more consistent than ratings of discharged residents, $\chi^2(1) = 8.28$, $p<.01$. Importantly, however, the reliability of CRS-AUD ratings (i.e., discrepant vs. non-discrepant) did not appear to vary with the transfer of a resident when compared to discharged and currently hospitalized residents [$\chi^2(1) = 0.84$, n.s., and $\chi^2(1) = 1.08$, n.s., respectively]. Moreover, assignment of the CRS-AUD categories did not depend on whether or not the resident was currently hospitalized, discharged, or transferred, [$\chi^2(6) = 7.61$, n.s.]

There were no significant differences in the number of days from the date of informed consent to the date chart ratings were completed, either between teams ($M = 91.07$ days, $SD = 22.4$ vs. $M=106.33$ days, $SD = 25.7$), $t(123) = 1.91$, $p>.05$ or within members of the same team ($M = 20.39; SD = 18.20$ vs. $M =13.78$ days $SD = 20.12$), $t(123) = 1.92$, $p>.05$. Complete chart ratings were obtained an average of 102.23 days ($SD = 24.4$; *Med* = 98) following initiation of informed consent procedures. The number of days to complete chart ratings did not appear to
vary as a function of the number of CRS-AUD discrepancies/agreements between raters \[ t(123) = -0.18, \text{n.s.} \] or CRS-AUD category assignments \[ F(3, 121) = 1.08, \text{n.s.} \]

On average, raters completed their code sheets at a rate of 23.00 min \((SD = 6.75, \text{Med} = 20)\) per chart, but there were significant differences in this regard across raters, \( F(3, 245) = 57.34, p<.001 \). Tukey HSD follow-up tests showed that one rater spent significantly less time \((M=17.87, SD = 6.77)\) and another significantly more time \((M=29.84, SD = 1.27)\) than the other two raters \((M=22.5, SD = 4.68 \text{ and } M=21.47, SD = 6.31)\) in chart review.\(^{261}\) Importantly, however, these time differences did not appear to vary as a function of CRS-AUD discrepancies/agreements between raters \[ t(121) = -1.60, \text{n.s.} \] or CRS-AUD category assignments \[ F(3, 119) = 0.79, \text{n.s.} \]

Preliminary examination of the inter-rater reliability of the CRS-AUD ratings indicated 74.6% agreement between raters for each category on the CRS (average across teams). An acceptable level of inter-rater reliability for each category on the CRS was also achieved as assessed by Intra-class/Alpha Coefficient \([\text{ICC} = 0.87, F(125, 125) = 7.49, p<.001]\) and the more conservative Cohen’s Kappa statistic \([0.63]\). With regard to dichotomous problem vs. non-problem distinctions on the CRS \(1 \text{ and } 2 = \text{non-problem}, 3+ = \text{problem}\), raters agreed in 87.3% of cases (average across teams). Inter-rater reliability estimates for this problem vs. non-problem distinction remained excellent even after controlling for chance agreement \((\text{Cohen’s Kappa} = 0.75)\). In order to optimize the stability of the CRS ratings, discrepancies were resolved by consensus of the raters and the primary investigator prior to the analyses.

**Correspondence of CRS Ratings with the AUDIT**

In the present study, 59 residents (46.8%) were assigned an AUD classification on the CRS, including \(n=10 \text{ (7.9%) Dependence (Level 4)} \) and \(n=49 \text{ (38.9%) Abuse (Level 3)} \). Thirty residents (23.8%) were noted to have engaged in non-problematic use of alcohol in the year prior to admission (Level 2), and the remaining 37 residents were identified as having abstained from alcohol during this time period (Level 1). No resident was identified with Severe Alcohol Dependence (Level 5). Of the 67 residents without an identified AUD in the year prior to their hospitalization, evidence for a past AUD at some point in their lifetime was noted in the chart records of 23 residents \((n=20 \text{ Abuse}; n = 3 \text{ Dependence})\). Thus, the percentage of the present sample identified with a current or lifetime AUD on the CRS was 65.1% \((82 \text{ out of } 126)\).

A total of 121 cases were available for cross-comparisons of the CRS-AUD categorizations with self-reported alcohol problems on the AUDIT. Applying the standardized cutoff score indicative of problematic use on the AUDIT \((\text{i.e., Total Score } >= 8)\), 74% of the
abstainers (Level 1) and 78.6% of those identified as having used alcohol without impairment (Level 2) scored in the non-problematic range on the AUDIT [$\chi^2(1) = 17.29, p < .001$], whereas 61% of residents with Abuse (Level 3) and 88.9% of those with Dependence (Level 4) obtained scores of eight or higher on the AUDIT [$\chi^2(1) = 5.586, p < .001$].

Descriptive statistics for the AUDIT Total and Subscale Scores (Hazardous Use, Harmful Use, and Dependence Symptoms) for each of the CRS classifications are provided in Table 7. Closer examination of the Total and Subscale scores suggested violations of the assumptions of normality required for mean comparisons, even after correcting for univariate outliers. Bimodal distributions were evident, as a disproportionately higher number of residents in the Abstainer (Level 1) and Use without Impairment (Level 2) categories obtained AUDIT scores of zero.

Table 7

<table>
<thead>
<tr>
<th></th>
<th>Abstainer (n=35)</th>
<th>Use w/o Impairment (n=28)</th>
<th>Abuse (n=49)</th>
<th>Dependence (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hazardous Use</td>
<td>2.71 (3.9)</td>
<td>3.32 (4.0)</td>
<td>5.73 (4.0)</td>
<td>7.11 (4.4)</td>
</tr>
<tr>
<td>Harmful Use</td>
<td>1.94 (2.9)</td>
<td>1.46 (2.7)</td>
<td>2.82 (2.9)</td>
<td>5.67 (3.2)</td>
</tr>
<tr>
<td>Dependence Symptoms</td>
<td>1.77 (3.1)</td>
<td>1.03 (2.4)</td>
<td>2.42 (3.0)</td>
<td>3.67 (3.8)</td>
</tr>
<tr>
<td>AUDIT Total</td>
<td>6.82 (10.3)</td>
<td>5.85 (8.3)</td>
<td>11.10 (8.7)</td>
<td>17.56 (10.7)</td>
</tr>
</tbody>
</table>

Accordingly, non-parametric analyses were used to compare AUDIT scores across CRS groups. Results showed significant overall differences in Hazardous Use [$\chi^2(3) = 17.26, p < .01$], Harmful Use [$\chi^2(3) = 11.47, p < .01$] and AUDIT Total [$\chi^2(3) = 24.63, p < .001$] scores between the CRS groups. There were no group differences on the Dependence Symptoms subscale, [$\chi^2(3) = 5.45, p > .1$], perhaps because of the restricted range of scores obtained on this scale. Follow-up Mann-Whitney U comparisons indicated that relative to Abstainers (Level 1), residents meeting CRS Abuse (Level 3) and Dependence (Level 4) criteria acknowledged more Hazardous Use [$U = 472.5; Z = 3.54, p < .01$ and $U = 69.5; Z = 2.65, p < .01$, respectively]. Dependant-, but not
Abuse-, level drinkers also reported more Harmful Use of alcohol than Abstainers \( U = 55.0; Z = 3.14, p < .01 \). More importantly, perhaps, residents identified as CRS Abuse (Level 3) and Dependence (Level 4) by chart review self-acknowledged more Hazardous Use \( U = 446.5; Z = 2.55, p < .01 \) and \( U = 64.0; Z = 2.22, p < .05 \), respectively] and Harmful Use \( U=495.0; Z = 2.16, p < .05 \) and \( U=29.0; Z=3.66, p < .001 \), respectively] compared to those able to drink non-problematically (Level 2). AUDIT Total Scores were higher among the two AUD groups (Level 3 and 4) relative to abstainers \( U = 547.5; Z = 2.83, p < .01 \) and \( U = 56.5; Z = 2.98, p < .01 \) and residents who consumed alcohol without impairment \( U = 410.5; Z = 2.93, p < .01 \) and \( U = 34.0; Z = 3.27, p < .01 \), respectively].

However, no differences in self-reported Hazardous Use, Harmful Use, or AUDIT Total scores were found between abstainers (Level 1) and non-problematic drinkers (Level 2), \( p's > 0.2 \). Additionally, although residents identified as Dependent (Level 4) by chart review self-admitted to more Harmful Use than those identified with Abuse (Level 3), scores on the Hazardous Use Subscale and the overall AUDIT Total did not vary with the increased ratings of AUD severity on the CRS, \( p's > 0.1 \). With just 10 individuals (nine with AUDIT data) identified as Dependent on the CRS, the statistical power available to compare this group classification with members of other CRS categories may have been limited.

Given the unequal CRS group sizes and with consideration of the above findings concerning correspondence with the AUDIT [i.e., Level 1 = Level 2 < Level 3 = Level 4], the four CRS groups were collapsed into non-problem (CRS-NP; Levels 1 & 2) and problem (CRS-P; Levels 3 & 4) categories. Applying the AUDIT cutoff for problematic use, a statistically significant 71.1% overall correspondence rate with CRS-AUD problem (Level 3 or 4) vs. non-problem (Level 1 or 2) classifications was obtained, \( \chi^2(1) = 22.0, p < .001 \). Non-parametric comparisons generally paralleled the above findings, in that residents in the CRS-P group reported significantly more Hazardous Use \( U = 1052.5; Z = 4.07, p < .001 \), Harmful Use \( U = 1276.0; Z=3.03, p<.01 \), and Dependence Symptoms \( U = 1475.0; Z = 2.12, p < .05 \) than those assigned to the CRS-NP. Thus, higher AUDIT Total Scores were observed among residents identified as CRS-P \( U=1048.5; Z=4.06, p < .001 \) compared to CRS-NP. Specifically, following correction for univariate outliers identified in the two groups, average AUDIT Total Scores were classified within the non-problematic range (<8) for residents identified as CRS-NP \( M=4.84, SD = 6.04 \) and within the problematic range (=>8) for CRS-P residents \( M = 12.34, SD = 9.79 \).

Considered together, results of the AUDIT interview support the validity of the CRS with regard to chart ratings of non-problem (Levels 1 & 2) and problem (Levels 3 and 4) alcohol use.
Prediction of AUD by the PANSS Factors

Statistical Assumptions

Using data obtained from the study sample ($N=126$), the distributions of scores on the five PCA components was examined for possible violations of the assumptions of the planned regression analysis. A single univariate outlier was identified in the ANX/DEP dimension and reined in by the median-interquartile method (Tabachnick & Fidell, 2001). Subsequent inspection of variable distributions, normal-probability plots, and skewness/kurtosis statistics indicated no further violations of the assumptions of linearity, normality, or homoscedacity.

Visual inspection of bivariate scatterplots did not suggest the presence of any curvilinear relationships or outlying cases among pairs of PANSS component scores. However, additional efforts were undertaken to identify multivariate outliers by performing a series of multiple linear regressions in which each PANSS component was regressed on the other four and the significance of Mahalanobis Distance values ($\chi^2(5) = 20.52, p<.001$) obtained from each model was examined (Tabachnick & Fidell, 2001). One multivariate outlier was identified (Mahalanobis Distance = 24.77) in all five regression models and was excluded from further analysis. Thus, a total of 125 residents were included in the planned logistic regression analysis.

Sample Characteristics

Table 8 presents the demographic and clinical characteristics of the study sample, of which 67 were identified as non-problem drinkers (CRS = 1 or 2) and 58 as problem drinkers (CRS = 3 or 4) via CRS chart ratings. A description of the sample follows, with significant differences between CRS groups noted where applicable.

The current hospitalization was the first for less than 1% of the sample. Indeed, a comparison of age at first hospitalization with age upon most recent admission to FSH suggests that the sample represents a relatively chronic group, with an average duration of mental illness of about 15.7 yrs ($SD=9.5$, $Med = 18$). The frequency and duration of previous inpatient psychiatric hospitalizations varied rather widely, however, ranging from zero to over 16 stays, and lasting less than one to nearly 15 years (> 178 mos), respectively. Despite the high rate of comorbid substance disorders observed, only three residents were noted as having participated in any formal substance disorder treatment apart from psychiatric hospitalization. Although residents identified as CRS-P had more prior inpatient psychiatric hospitalizations than those identified as CRS-NP, $t(123) = 2.27, p<.05$, the CRS groups did not differ with regard to the total duration of previous hospitalizations, $t(123) = -0.46$, n.s. No other significant differences in past treatment history variables were observed.
Table 8

Sample Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Entire Sample (N=125)</th>
<th>CRS-NP (n=67)</th>
<th>CRS-P (n=58)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean (SD)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age on Admission</td>
<td>39.5 (9.4)</td>
<td>40.1 (9.3)</td>
<td>38.7 (9.6)</td>
</tr>
<tr>
<td>Education (Years)</td>
<td>10.8 (1.9)</td>
<td>10.7 (2.0)</td>
<td>10.9 (1.7)</td>
</tr>
<tr>
<td><strong>Frequency (N)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>21 (16.8)</td>
<td>13 (19.4)</td>
<td>8 (13.8)</td>
</tr>
<tr>
<td>Male</td>
<td>104 (83.2)</td>
<td>54 (80.6)</td>
<td>50 (86.2)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian/White</td>
<td>48 (38.4)</td>
<td>22 (32.8)</td>
<td>26 (44.8)</td>
</tr>
<tr>
<td>African-American/Black</td>
<td>68 (54.4)</td>
<td>41 (61.2)</td>
<td>27 (46.6)</td>
</tr>
<tr>
<td>Hispanic American/Latino</td>
<td>6 (4.8)</td>
<td>2 (3.0)</td>
<td>4 (6.9)</td>
</tr>
<tr>
<td>Other or Unknown</td>
<td>3 (2.4)</td>
<td>2 (3.0)</td>
<td>1 (1.7)</td>
</tr>
<tr>
<td><strong>Treatment Variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean (SD)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at 1st Hospitalization</td>
<td>27.4 (9.6)</td>
<td>27.7 (9.2)</td>
<td>27.1 (10.1)</td>
</tr>
<tr>
<td>Lifetime Hospitalizations</td>
<td>6.0 (4.2)</td>
<td>5.2 (3.8)</td>
<td>6.9 (4.4)</td>
</tr>
<tr>
<td>Length of Previous Hospitalizations (in months)</td>
<td>68.0 (57.3)</td>
<td>65.8 (58.5)</td>
<td>70.6 (56.2)</td>
</tr>
<tr>
<td>GAF on admission</td>
<td>40 (10.6)</td>
<td>41.1 (9.7)</td>
<td>38.8 (11.4)</td>
</tr>
<tr>
<td>LOS at Consent (in days)</td>
<td>404.8 (306.0)</td>
<td>359.6 (278.0)</td>
<td>457.0 (330.2)</td>
</tr>
<tr>
<td><strong>Commitment Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Civil</td>
<td>12 (9.6)</td>
<td>7 (10.4)</td>
<td>5 (8.6)</td>
</tr>
<tr>
<td>Forensic</td>
<td>113 (90.4)</td>
<td>60 (89.6)</td>
<td>53 (91.4)</td>
</tr>
<tr>
<td><strong>Medication Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Typical or Atypical Neuroleptic</td>
<td>122 (97.6)</td>
<td>65 (97.0)</td>
<td>57 (98.3)</td>
</tr>
<tr>
<td>Other</td>
<td>3 (2.4)</td>
<td>2 (3.0)</td>
<td>57 (1.7)</td>
</tr>
</tbody>
</table>
The average duration of current hospitalization at the time of informed consent was 404.8 days ($SD = 306.0$, Median = 328; Range $= \{76, 1241\}$). A majority of the sample had been committed to FSH via the criminal court system, including 52 residents (41.3%) adjudicated Not Guilty by Reasons of Insanity and 62 residents (49.2%) who were determined to be Incompetent to Proceed to Trial. Of these, 27 had been accused of committing non-violent criminal acts, 48 of violent criminal acts, and 38 of both non-violent and violent acts. There were no significant differences between CRS groups with regard to commitment status or type of offense.

In terms of their overall severity in functioning, GAF scores recorded on admission ranged from 15 to 70. At the point of chart ratings, residents’ overall functioning had remained much the same as on admission, $r(125)_{GAF \text{ database/chart}} = 0.75$, $p < .001$ with 65.6% of the sample’s GAF scores unchanged, 15.2% worsened, and 19.2% improved. Overall, however, residents improved an average of 1.39 ($SD = 7.7$) units on the GAF, $t(125) = 2.03$, $p < .05$. There were no significant differences in GAF, or changes thereof, between the CRS-P and CRS-NP groups.

In addition to their primary SZC diagnosis, a majority of the sample was diagnosed with one or more comorbid Axis I ($n=74; 58.7\%$) and/or Axis II ($n=53; 42.1\%$) disorders upon admission to FSH. Over the course of their hospitalization, diagnoses were clarified by hospital personnel and a total of 86 (68.3%) concurrent Axis I and 58 (46.0%) Axis II disorders were recorded from chart records (See Table 9). The figures presented in Table 9 indicate only single instances of each disorder and do not take into account their co-occurrence within the same individual. Indeed, nearly half of the sample presented with two (23.2%), three (19.8%) or more (4.8%) comorbid conditions. Of particular note, 21 of the 74 comorbid diagnoses on admission were AUDs with 13 more identified over the course of hospitalization. Another 13 residents received diagnoses of Polysubstance Dependence with alcohol specified as one of the substances of abuse. Thus, 46 residents (36.8%) had their alcohol problems documented by the hospital personnel overseeing their treatment. Thirty-nine (31.2%) residents were diagnosed with other drug disorders upon their admission to FSH, with 26 others identified with non-AUD substance disorders over the course of their hospitalizations (i.e., 52% of the sample). A majority of those diagnosed with AUD (76.1%) were also diagnosed with another drug disorder. Overall, problem drinkers were diagnosed with more comorbid conditions ($M=2.89$, $SD = 1.11$) compared to non-problem drinkers ($M=2.20$, $SD=1.03$) $t(123) = 3.57$, $p < .001$. Specifically, those in the CRS-P group were more likely to have been diagnosed with a comorbid drug disorder (67.8% vs. 38.8%; $\chi^2(1) = 10.57$, $p < .001$) as well as antisocial personality disorder ($\chi^2(1) = 10.57$, $p < .001$) than those in the CRS-NP group.
Table 9

Frequency (%) of DSM-IV Diagnoses Recorded from Chart Records

<table>
<thead>
<tr>
<th>Primary Diagnoses</th>
<th>Entire Sample</th>
<th>CRS-NP</th>
<th>CRS-P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>64 (51.2)</td>
<td>39 (58.2)</td>
<td>25 (43.1)</td>
</tr>
<tr>
<td>Paranoid Type</td>
<td>39 (31.2)</td>
<td>26 (38.8)</td>
<td>13 (22.4)</td>
</tr>
<tr>
<td>Undifferentiated</td>
<td>18 (14.4)</td>
<td>8 (11.9)</td>
<td>10 (17.3)</td>
</tr>
<tr>
<td>Disorganized</td>
<td>5 (4.0)</td>
<td>4 (6.0)</td>
<td>1 (1.7)</td>
</tr>
<tr>
<td>Residual</td>
<td>2 (2.0)</td>
<td>1 (1.5)</td>
<td>1 (1.7)</td>
</tr>
<tr>
<td>Schizoaffective</td>
<td>61 (48.8)</td>
<td>28 (41.8)</td>
<td>33 (56.9)</td>
</tr>
<tr>
<td>Depressed Type</td>
<td>5 (4.0)</td>
<td>4 (6.0)</td>
<td>1 (1.7)</td>
</tr>
<tr>
<td>Bipolar Type</td>
<td>50 (40.0)</td>
<td>20 (29.8)</td>
<td>30 (51.7)</td>
</tr>
<tr>
<td>Not Specified</td>
<td>6 (4.8)</td>
<td>4 (6.0)</td>
<td>2 (3.4)</td>
</tr>
</tbody>
</table>

Comorbid Diagnoses

<table>
<thead>
<tr>
<th>Any Substance Disorder ² ³</th>
<th>Entire Sample</th>
<th>CRS-NP</th>
<th>CRS-P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>33</td>
<td>7</td>
<td>26</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Cannabis</td>
<td>15</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Cocaine</td>
<td>16</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Polysubstance</td>
<td>35</td>
<td>14</td>
<td>21</td>
</tr>
</tbody>
</table>

Secondary Psychotic Disorder

| Schizoaffective          | 4             | 1      | 3     |
| Paranoid Type            | 3             | 1      | 2     |
| Psychotic Disorder, NOS  | 3             | 2      | 1     |

Other Axis I Disorders

| Obsessive Compulsive Disorder | 1 | 1 | 0 |
| Panic Disorder               | 1 | 0 | 1 |
| Post-Traumatic Stress Disorder | 1 | 0 | 1 |
| Cognitive Disorder/Dementia  | 2 | 2 | 0 |
| Pedophilia                   | 2 | 2 | 0 |
| Sexual Sadism                | 1 | 1 | 0 |
| Tourette’s Syndrome          | 1 | 1 | 0 |

<table>
<thead>
<tr>
<th>Any Substance Disorder ² ³</th>
<th>Entire Sample</th>
<th>CRS-NP</th>
<th>CRS-P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>33</td>
<td>7</td>
<td>26</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>1</td>
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<tr>
<td>Cannabis</td>
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<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Cocaine</td>
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<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Polysubstance</td>
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Table 9 - continued

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<th>Developmental Disorders</th>
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<tr>
<td>Borderline Intellectual Functioning</td>
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</tr>
<tr>
<td>Mental Retardation (Mild or Unspecified)</td>
<td>12</td>
<td>8</td>
<td>4</td>
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</table>

<table>
<thead>
<tr>
<th>Personality Disorder</th>
<th>41 (32.8%)</th>
<th>18 (26.9%)</th>
<th>23 (39.7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antisocial</td>
<td>22</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>Borderline</td>
<td>6</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Schizoid</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Schizotypal</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Not Otherwise Specified</td>
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<td>7</td>
<td>6</td>
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</tbody>
</table>

This figure excludes three residents diagnosed with Nicotine Dependence; b n=3 Alcohol Dependence and n=30 Alcohol Abuse; c n=15 cases included alcohol in poly substance diagnosis (CRS-NP = 7, CRS-P = 8); two of the CRS-NP also had a separate AUD diagnosis.

CRS Groups and the PANSS: Logistic Regression Analyses

Descriptive statistics and bivariate correlations for the PANSS component scores are presented in Table 10. Simple t-test comparisons revealed no significant differences between component scores as a function of CRS group membership (see Appendix C-2).

Table 10

PANSS Component Scores: Correlations and Descriptive Statistics

<table>
<thead>
<tr>
<th></th>
<th>NEG</th>
<th>COG</th>
<th>POS</th>
<th>ANX/DEP</th>
<th>EXC/HOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEG</td>
<td>1.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COG</td>
<td></td>
<td>.447**</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td></td>
<td></td>
<td>.252**</td>
<td>.551**</td>
<td>1.11</td>
</tr>
<tr>
<td>ANX/DEP</td>
<td></td>
<td>.341**</td>
<td>.382**</td>
<td>.342**</td>
<td>0.89</td>
</tr>
<tr>
<td>EXC/HOS</td>
<td></td>
<td>.283**</td>
<td>.572**</td>
<td>.587**</td>
<td>.391**</td>
</tr>
</tbody>
</table>

Mean (SD): -0.035 (1.1), -0.024 (1.0), -0.050 (1.0), -0.068 (0.9), -0.08 (1.0)

(Min., Max.): (-1.88, 2.38), (-1.79, 2.51), (-2.19, 2.17), (-1.98, 2.54), (-2.10, 2.51)

Note. Entries on the diagonals represent covariances [** p<0.01 level (2-tailed)]
A hierarchical logistic regression analysis was conducted with CRS-ratings of AUD as the dependent variable (1=problem alcohol use, 0=non-problem alcohol use or abstinence) and NEG (flat affect, alogia, social withdrawal), POS (distortions in thought content and perception) and COG (formal thought disorder, mental confusion, distractibility, concrete reasoning) as the key independent variables. In addition to the planned covariates (age, gender, GAF, ANX/DEP, EXC/HOS), a dichotomous variable representing the presence (coded 1) or absence (coded 0) of a drug use disorder (DUD) was also entered in step one of the model.35

Although the significant inter-correlations among NEG and POS with COG satisfied the first requirement for testing the mediational effect of COG on the relationship between POS/NEG and AUD (Kenny et al., 1998), no evidence for either unique or shared contributions of POS/NEG to CRS-AUD was found in the planned hierarchical regression analyses. COG approached significance as a marker of CRS-AUD group membership, but neither complete nor partial mediation by COG was supported. See Table 11.

Therefore, the potential moderating effects of COG, POS, and NEG on CRS-AUD were assessed on a post-hoc basis. When interaction terms were entered in stepwise fashion in the final step, the overall model remained significant \( \chi^2(10) = 22.81, p<.05 \) and a significant POS X COG interaction was obtained, \( \text{EXP(B)} = 0.65, \text{Wald} = 4.58, p<.05; \Delta \chi^2(1) = 4.94, p<.05. \) The inclusion of this interaction term improved the sensitivity of the model (i.e., capacity to detect problem alcohol use) from 60.3% to 65.5%, compromising specificity (i.e., capacity to detect non-problem use) only slightly from 71.6% to 70.1%.

To follow up this interaction, a median-split of scores on POS was computed and the effects of COG on AUD as a function of high- or low- positive symptomatology were tested. A Levene’s Test indicated statistically equivalent variance in COG across these groups (Low POS \( s^2 = 0.66 \) and High POS \( s^2 = 0.92; F(1, 123) = 3.08, \text{n.s.}, \)) including after accounting for variance attributable to the covariates \( F(1, 123) = 0.22, \text{n.s.}, \) meaning that the observed interaction is unlikely to have emerged simply from restricted range of COG in one of the groups. Regarding the identification of alcohol problems, results suggested that whereas there was no association between COG and AUD for residents with fewer POS symptoms, \( \Delta \chi^2(1) = 0.01, \text{n.s.}, \) each unit increase in the severity of COG symptoms was associated with a nearly four times decreased likelihood of problematic use for persons with higher POS symptoms, \( \text{Wald} = 4.56, p<.05; \Delta \chi^2(1) = 6.48, p<.01. \) In other words, the apparently protective impact of schizophrenia-related cognitive deficits on AUD depended on the severity of positive symptomatology.36
### Table 11

**Logistic Regression Results**

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>EXP(B) 95% CI</th>
<th>Wald</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE</strong></td>
<td>-0.01 (0.02)</td>
<td>1.00 {0.95, 1.03}</td>
<td>0.09</td>
</tr>
<tr>
<td><strong>GENDER</strong></td>
<td>0.08 (0.5)</td>
<td>1.08 {0.38, 3.11}</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>GAF</strong></td>
<td>-0.02 (0.02)</td>
<td>1.00 {0.94, 1.02}</td>
<td>0.70</td>
</tr>
<tr>
<td><strong>ANX/DEP</strong></td>
<td>-0.32 (0.2)</td>
<td>0.73 {0.46, 1.14}</td>
<td>1.90</td>
</tr>
<tr>
<td><strong>EXC/HOS</strong></td>
<td>0.17 (0.2)</td>
<td>1.19 {0.76, 1.85}</td>
<td>0.60</td>
</tr>
<tr>
<td><strong>Other Drug Disorder</strong></td>
<td>1.11 (0.4)</td>
<td>3.04 {1.40, 6.61}</td>
<td>7.86, p&lt;.01</td>
</tr>
</tbody>
</table>

**Step One** $\chi^2(6) = 13.48, p<.05$

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>EXP(B) 95% CI</th>
<th>Wald</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE</strong></td>
<td>-0.01 (0.02)</td>
<td>1.00 {0.95, 1.04}</td>
<td>0.05</td>
</tr>
<tr>
<td><strong>GENDER</strong></td>
<td>0.28 (0.6)</td>
<td>1.32 {0.43, 4.08}</td>
<td>0.23</td>
</tr>
<tr>
<td><strong>GAF</strong></td>
<td>-0.02 (0.02)</td>
<td>0.98 {0.94, 1.02}</td>
<td>1.02</td>
</tr>
<tr>
<td><strong>ANX/DEP</strong></td>
<td>-0.23 (0.2)</td>
<td>0.80 {0.49, 1.29}</td>
<td>0.87</td>
</tr>
<tr>
<td><strong>EXC/HOS</strong></td>
<td>0.23 (0.3)</td>
<td>1.26 {0.76, 2.09}</td>
<td>0.81</td>
</tr>
<tr>
<td><strong>Other Drug Disorder</strong></td>
<td>1.13 (0.4)</td>
<td>3.08 {1.40, 6.77}</td>
<td>7.87, p&lt;.01</td>
</tr>
<tr>
<td><strong>NEG</strong></td>
<td>-0.23 (0.2)</td>
<td>0.79 {0.53, 1.18}</td>
<td>1.29</td>
</tr>
<tr>
<td><strong>POS</strong></td>
<td>-0.04 (0.3)</td>
<td>0.96 {0.59, 1.57}</td>
<td>0.03</td>
</tr>
</tbody>
</table>

**Step Two** $\Delta \chi^2(2) = 1.32, \text{n.s.}$

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>EXP(B) 95% CI</th>
<th>Wald</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE</strong></td>
<td>-0.01(0.02)</td>
<td>1.00 {0.95, 1.04}</td>
<td>0.076</td>
</tr>
<tr>
<td><strong>GENDER</strong></td>
<td>0.33 (0.6)</td>
<td>1.40 {0.45, 4.38}</td>
<td>0.326</td>
</tr>
<tr>
<td><strong>GAF</strong></td>
<td>-0.02(0.02)</td>
<td>0.98 {0.94, 1.02}</td>
<td>1.162</td>
</tr>
<tr>
<td><strong>ANX/DEP</strong></td>
<td>-0.19(0.3)</td>
<td>0.83 {0.51, 1.35}</td>
<td>0.584</td>
</tr>
<tr>
<td><strong>EXC/HOS</strong></td>
<td>0.38(0.3)</td>
<td>1.46 {0.85, 2.50}</td>
<td>1.866</td>
</tr>
<tr>
<td><strong>Other Drug Disorder</strong></td>
<td>1.12(0.4)</td>
<td>3.07 {1.38, 6.83}</td>
<td>7.58, p&lt;.01</td>
</tr>
<tr>
<td><strong>NEG</strong></td>
<td>-0.13 (0.2)</td>
<td>0.88 {0.57, 1.35}</td>
<td>0.358</td>
</tr>
<tr>
<td><strong>POS</strong></td>
<td>0.09 (0.3)</td>
<td>1.09 {0.65, 1.84}</td>
<td>0.110</td>
</tr>
<tr>
<td><strong>COG</strong></td>
<td>-0.49 (0.3)</td>
<td>0.62 {0.36, 1.07}</td>
<td>2.96, p=0.085</td>
</tr>
</tbody>
</table>

**Step Three** $\Delta \chi^2(1) = 3.17, p=0.08$

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>EXP(B) 95% CI</th>
<th>Wald</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COG</strong></td>
<td>-0.49 (0.3)</td>
<td>0.62 {0.36, 1.07}</td>
<td>2.96, p=0.085</td>
</tr>
</tbody>
</table>

**Overall Model** $\chi^2(9) = 17.98, p<.05$
Figure 1 provides an illustration of the interaction of POS and COG on CRS group membership, controlling for the effects of the covariates, which were set equal to their respective mean values. This graph shows that among those with more severe positive symptoms, those with relatively intact cognitive functioning had the highest risk for alcohol problems (CRS-P), whereas those with more severe cognitive deficits had the lowest risk (CRS-NP). In contrast, persons with less severe symptomatology showed an approximately 42% odds of alcohol problems regardless of their scores on COG.

Figure 1. The interaction of POS and COG on CRS group membership. Based on median-split of POS, those with more severe positive symptoms are depicted by the line with squares and persons with less severe positive symptomatology are represented by the line with diamonds.

Supplemental SEM Analyses
Using Lisrel (v. 8.53), Structural Equation Modeling (SEM) was applied to assess the magnitude of the hypothesized indirect effects of POS and NEG on AUD by way of COG. The
The hypothesized model is presented in Figure 2. Circles represent latent variables and rectangles symbolize measured variables. Asterisks denote estimated parameters.

**Figure 2.** Hypothesized Structural Equation Model: The indirect effects of POS and NEG on AUD by way of COG

Inasmuch as only 125 cases were available for this analysis, the measurement model could not be estimated on the basis of all 29 PANSS items suggested by the original exploratory factor analytic procedures. Therefore, each latent symptom variable (i.e., POS, COG, NEG) was constrained to a value equal to the variance of its respective observed component score multiplied by one minus its reliability \( SD^2 \times (1 - \text{reliability}) \). The reliability of the component scores was estimated as the average SMC for “on-factor” items, as noted in Table 5 above. Given that measurement of the latent AUD construct was dichotomous (i.e., not normally distributed) a poly-serial/Pearson correlation matrix was computed. The fit of the current scaled-down model was estimated using a Weighted Least Squares procedure, which analyzes the correlation matrix weighted by the asymptotic covariance matrix of the observed variables. All error variances were fixed to zero and the covariance of POS and NEG was set to 0.289 (the
covariance of the observed factor scores). Table 12 presents the correlations, variances, and fixed parameter estimates of the indicators in the model.

Table 12

*Correlation Matrix ofObserved Variables in the SEM*

<table>
<thead>
<tr>
<th></th>
<th>AUD</th>
<th>COG</th>
<th>NEG</th>
<th>POS</th>
</tr>
</thead>
<tbody>
<tr>
<td>AUD</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG</td>
<td>-0.088a</td>
<td>1.186</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COG</td>
<td>-0.156a</td>
<td>0.447b</td>
<td>0.999</td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td>-0.006a</td>
<td>0.252b</td>
<td>0.551b</td>
<td>1.107</td>
</tr>
<tr>
<td>Fixed Parameter</td>
<td>1.0</td>
<td>0.493</td>
<td>0.417</td>
<td>0.445</td>
</tr>
</tbody>
</table>

*Note.* Diagonal entries represent the variances of the observed variables. a polyserial correlations b pearson correlations.

The independence model, which tests the hypothesis that the variables are uncorrelated with each other, was rejected $\chi^2(6; N=125) = 35.71, p<.001$. A chi-square difference test indicated that the hypothesized model offered an improved fit to the data relative to the independence model, $\Delta \chi^2(5; N=125) = 32.78, p<.001$. Indeed, support for the hypothesized model was indicated by the chi-square test statistic $[\chi^2(1; N=125) = 2.93, p=0.09]$ as well as standard fit indices: CFI = 0.93, NFI = 0.92, AGFI = 0.94. However, the RMSEA 0.12 (95% Confidence Interval = {0.0, 0.30}) exceeded the acceptable range (i.e., 0.05 to 0.08) and the test of the null hypothesis that RMSEA is less than 0.05 was not rejected, $p=0.14$.

The estimated SEM indicated that COG was strongly predicted by both NEG (Standardized Coefficient = 0.30, $t = 2.92, p<.01$) and POS (Standardized Coefficient = 0.44, $t = 4.23, p<.01$), and that there was a significant change in AUD caused by COG, controlling for NEG and POS (Standardized Coefficient = -0.19, $t = -2.21, p<.05$). However, there was no evidence for a change in AUD status caused by change in NEG or POS either directly or by way of COG. In this model, AUD was predicted only by fewer cognitive symptoms. See Table 13.
Table 13

SEM Standardized Coefficients

<table>
<thead>
<tr>
<th></th>
<th>Direct Effects</th>
<th>Indirect Effects</th>
<th>Total Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NEG</td>
<td>POS</td>
<td>COG</td>
</tr>
<tr>
<td>COG</td>
<td>0.30**</td>
<td>0.44**</td>
<td>--</td>
</tr>
<tr>
<td>AUD</td>
<td>-0.02</td>
<td>0.11</td>
<td>-0.19*</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01

Exploratory Analyses: Missed AUD Diagnoses

With consideration of the aforementioned literature questioning the reliable diagnoses of AUD among staff overseeing inpatient populations, it was of some interest to determine (a) the extent to which DSM-IV diagnoses of Alcohol Abuse/Dependence and/or Polysubstance with Alcohol, as assigned by FSH personnel, mapped onto the current identification of AUD using the CRS and (b) what PANSS factors, if any, predicted missed AUD diagnoses.

A statistically comparable proportion of residents with CRS Abuse (Level 3) and Dependence (Level 4) had an AUD noted in their chart records (62.5% and 40%, respectively), $\chi^2(1) = 1.73$, n.s. Likewise, no differences in proportions of AUD diagnoses were evident between the non-problematic CRS groups, $\chi^2(1) = 0.77$, n.s. Although the proportions of AUD diagnosis differed among those assigned to CRS-Abuse relative to abstainers (21.6%; $\chi^2(1) = 14.12$, $p<.001$) and non-problematic drinkers (13.3%; $\chi^2(1) = 18.15$, $p<.001$), residents identified with Dependence (Level 4) were not significantly differentiated from the non-problematic CRS groups (Levels 1 and 2), perhaps because of the small number of residents assigned to this category, [$\chi^2(1) = 1.40$, n.s. and 3.33, $p<.07$, respectively]. However, relative to the CRS-NP group, the CRS-P group was significantly more likely to have been assigned a DSM-IV AUD Diagnosis (17.9% vs. 59.3%), $\chi^2(1) = 11.26$, $p<.001$.

Although these findings tend to lend support to the current use of a dichotomous, problem vs. non-problem, CRS classification, there was certainly not perfect correspondence between CRS ratings and FSH diagnoses of an alcohol-related disorder. Indeed, of the 58 residents identified as having recent (past year) alcohol problems on the CRS, 24 (41.4%) were not so documented in the chart records. Among those identified with either a recent or past AUD via CRS chart ratings (n=81), 44.4% were missed by hospital personnel.
In order to determine if a resident’s symptom presentation influenced whether a diagnosis of AUD was missed by hospital clinicians, a Hierarchical Logistic Regression analysis using the PANSS factors as independent variables and mismatch of lifetime AUD as the dichotomous dependent variable (Consistent Classification = 0, FSH Missed Diagnosis = 1) was conducted. GAF was entered in the first step of the equation to control for the effects of overall symptom severity. Results suggested that correspondence between AUD Diagnoses and CRS groups could be improved with consideration of the PANSS factors, \( \Delta \chi^2(5) = 10.92, p=0.053 \), particularly ANX/DEP (feelings and/or physical signs of anxiety; depressive outlook; guilt/shame; physical preoccupation). Each unit increase in ANX/DEP was associated with a more than three times greater likelihood that an alcohol problem would go undiagnosed. Missed AUD was not significantly predicted by any interactions among PANSS components. See Table 14.

### Table 14

**Prediction of Missed Lifetime AUD Diagnoses Using the PANSS Components**

<table>
<thead>
<tr>
<th></th>
<th>B (S.E.)</th>
<th>Exp(B)</th>
<th>Wald</th>
</tr>
</thead>
<tbody>
<tr>
<td>GAF</td>
<td>-0.05 (0.03)</td>
<td>0.95 {0.94, 1.03}</td>
<td>2.63</td>
</tr>
<tr>
<td>NEG</td>
<td>0.13 (0.3)</td>
<td>1.13 {0.76, 2.20}</td>
<td>0.14</td>
</tr>
<tr>
<td>COG</td>
<td>-0.88 (0.5)</td>
<td>0.42 {0.19, 0.93}</td>
<td>3.36, ( p=.07 )</td>
</tr>
<tr>
<td>POS</td>
<td>0.39 (0.4)</td>
<td>1.48 {0.86, 3.04}</td>
<td>1.21</td>
</tr>
<tr>
<td>ANX/DEP</td>
<td>1.11 (0.4)</td>
<td>3.03 {1.22, 4.42}</td>
<td>6.68, ( p&lt;.01 )</td>
</tr>
<tr>
<td>EXC/HOS</td>
<td>-0.37 (0.4)</td>
<td>0.69 {0.39, 1.46}</td>
<td>0.80</td>
</tr>
</tbody>
</table>

Overall \( \chi^2(6) = 12.89, p<.05 \)
DISCUSSION

The current study generated fresh insights into the nature of the relationship between schizophrenia and alcohol problems by using factor analytically derived PANSS dimensions to examine relationships between AUD and the core positive and negative symptoms of schizophrenia as a function of variations in cognitive symptomatology. The results pointed to a somewhat unexpected association of SZC cognitive symptomatology with AUD in this sample of inpatients with primary diagnoses of schizophrenia or schizoaffective disorder. Contrary to predictions, symptomatic deficits in cognitive functioning did not serve to place persons with schizophrenia at risk for making maladaptive decisions regarding the initiation and curtailment of alcohol use, and hence AUD. Rather, the lowest rates of alcohol problems were found among those who exhibited the worst cognitive symptoms, an effect that depended upon the severity of positive symptomatology. It is suggested that the current findings reflect a floor effect of cognitive functioning, potentially predating the onset of schizophrenia, that in the context of poor reality-testing could limit these SZCs' efforts to try to compensate for dysfunction and/or resultant distress by drinking. Clinical implications of these findings and directions for future research are explored in the following discussion.

Relationship of Schizophrenia Symptoms and AUD

Evidence that a simple positive-negative dichotomy may be insufficient to explain the observed heterogeneity of SZC symptomatology was supported by the current analysis of PANSS data collected from a large inpatient sample. Furthermore, as one of the first to apply oblique rotation to the PANSS, I have presented data to show the moderate degree of common variance among the five components, thus corroborating the notion that SZC psychopathology is comprised of discrete, but co-existing symptom domains (Lindenmeyer et al., 1994). Five symptom components were identified, including the expected NEG, POS, and COG dimensions, as well as additional components tapping disturbances of an affective and behavioral nature (i.e., ANX/DEP and EXC/HOS). The relative significance of the negative syndrome in
schizophrenia was underscored by the fact that NEG emerged first in the analysis and was the strongest, most stable component (Hwu et al., 2002). It was captured by seven items representing affective/social withdrawal, lack of interest and involvement in life, halted communication, and psychomotor retardation. POS appeared to represent distortions in thought content and perception separable from behavioral disorganization (Lindenmeyer et al., 1995), encapsulating the key positive symptoms of delusions and hallucinations, except those of a somatic nature. Finally, and most importantly to the current study aims, eight PANSS items were linked to a distinct cognitive component, a finding that supports the separable and fundamental nature of cognitive symptomatology to schizophrenia disorders (Bell et al., 1994; Lancon et al., 2000). In this study, COG tapped symptoms of formal thought disorder as well as disturbances in abstract thinking, orientation, attention, judgment, volition, and mannerisms.  

Although the current five-component solution was generally consistent with previous research in this area, some differences in the attachment of specific PANSS items to the five factors were evident (see Appendix D-1). Variations in the application of statistical procedures, such as the type of factor analysis applied, the method of factor extraction/rotation, and criteria for item retention, may partially explain the observed discrepancies. It is noteworthy that the present PCA solution showed the highest concordance with two studies that examined large samples of predominantly chronic SZC inpatients similar to the current sample (i.e., Bell et al., 1994 re-analysis of a Kay & Sevy, 1990; Fredrickson et al., 1997). For the most part, however, the rules applied for inclusion/exclusion of sample participants constituted a notable methodological difference across studies. For example, the proportion of females included have varied widely from the current study (22%), averaging 28% in the studies cited in Appendix D-1 but ranging from a low of 5% (Bell et al., 1994a) to a high of 54.3% (Dollfus et al., 1991). This is perhaps in part because males tend to be over-represented in inpatient populations (Mueser et al., 1990) such that studies that also considered outpatients probably involved more female SZCs. Furthermore, whereas some researchers have excluded those persons with SZA disorders and/or SZCs presenting with certain comorbid conditions, such as substance use disorders, organic dysfunction, mental retardation, and/or somatic illness (e.g., Lancon et al., 2000, Lindenmeyer et al., 1994, Mass et al., 2000), I did not. Indeed, in order that results be representative of the full range of SZC psychopathology, not only were each of subtypes of schizophrenia and SZA represented in the factor analysis sample, but also no restrictions on the medication status, severity, or duration (acute vs. chronic) of illness were imposed. Although the majority of the current sample had been referred to treatment by legal authorities, each
required present treatment for their psychotic symptoms and most had required previous psychiatric hospitalizations.

Given the potential impact of variable sample composition on analyses of the PANSS, the consistency of the current factor analysis results with former investigations is really quite remarkable. Although the exact location of each of the PANSS items on the five factors can not be conclusively delineated on the basis of the current findings, when considered together with previous research, the present utilization of a more inclusive sample may suggest that the five-factor model may be applied more broadly than it has been previously. Specifically, it seems to describe the heterogeneity of SZC symptomatology regardless of gender composition, treatment status, phase of illness, or the presence of any mood disturbance or other comorbid conditions.38 Thus, current use of a more inclusive sample not only is justifiable, but also extends previous research in this area, which to date has either involved select purified samples that limit generalizability to dual-diagnosis populations (e.g., Lançon et al., 2000; Lindenmeyer et al., 1995), or has involved similarly heterogeneous samples but without attention to how symptom variations may relate to, or perhaps explain, the AUD-SZC connection.

Indeed, the current findings point to some specificity in the relationship between the disorders of schizophrenia and alcohol use. Although a large majority (72%) of the current inpatient sample had engaged in alcohol use in the year prior to their admission, the likelihood of alcohol-related problems during this time period was related to the severity and interdependence of COG and POS, but not NEG, symptomatology. Specifically, I found that whereas the degree of cognitive impairment was not associated with AUD in the context of fewer positive symptoms, worse cognitive deficits were associated with a nearly four-times reduced likelihood of AUD among those who suffered more severe hallucinations and delusions. The current findings do replicate research indicating NEG symptoms do not vary with dual-diagnosis status (Zisook et al., 1992; Sevy et al., 1990), but they clearly contrast with research showing that worse functional outcomes are predicted largely by deficits in neurocognitive functioning (e.g., Green 1996; Green et al., 2000), with the contribution of other SZC symptoms largely attenuated by the introduction of neurocognitive variables that carry more of the variance (e.g., Dickensen & Coursey, 2002; Velligan et al., 1997). Rather, the current results underscore the complexity of the independent and interactive relationships among the symptoms of schizophrenia and relevant clinical outcomes, such as AUD. As variance associated with other drug use disorders was statistically controlled, these symptom associations can be interpreted with respect to AUDs specifically, rather than substance disorders more generally.
Yet, in light of my assertion that the cognitive deficit symptoms of schizophrenia, which in some ways resemble acute cognitive impairment associated with moderate alcohol intoxication, render certain cognitively-compromised SZCs particularly susceptible to maladaptive decisions regarding alcohol use as well as the experience of adverse alcohol-related outcomes with less use, finding that intact cognitive functioning was associated with a greater likelihood of AUD among those with worse positive symptoms was unexpected. Moreover, I had presented research evidence suggesting a link between certain positive symptoms and specific cognitive errors in order illustrate how bivariate relationships between positive symptoms and AUD could be attributed to, i.e., mediated by, the primary contribution of cognitive deficit symptomatology to AUD development. Hence, I am faced with the task of explaining how that same link could decrease the risk for AUD in a manner consistent with the present results. The present findings cannot be easily attributed to the demographic characteristics, IQ, other pathology, severity of non-SZC symptoms, medication status, and/or chronic institutionalization, as the effects of these variables were either statistically controlled or evaluated and found to be of negligible relevance. Furthermore, the observation that worse cognitive symptoms were associated with a reduced likelihood of recent problematic alcohol use renders it unlikely that the cognitive or other symptom differences were due simply to the direct effects of substance use. Although based on evidence from the extant alcohol, schizophrenia, and dual-diagnosis literatures, the rationale for my initial predictions obviously requires revision in light of the obtained pattern of findings.

For a variety of practical and ethical reasons, the present study was not designed to examine the effects of acute alcohol exposure on SZCs’ functioning, which would be necessary to examine directly whether cognitively-compromised SZCs experience more of alcohol’s deleterious effects on their functioning with lower doses than do non-SZC drinkers. In addition to the fact that a substantial minority of non-problematic drinkers was identified, this proposed mechanism was rendered further unlikely by the finding that cognitively-compromised SZCs were least likely to experience alcohol problems. Nonetheless, given that the present study did successfully identify SZCs who previously drank problematically, it can perhaps be assumed that those so-identified at some point “decided” to drink and subsequently encountered negative alcohol-related outcomes. Thus, perhaps the high rate of AUD in schizophrenia is not attributable to SZCs’ failure to make the connection between alcohol use and consequent outcomes, but rather reflects how cognitive deficits serve to limit the repertoire and ability to implement coping strategies, including maladaptive ones like alcohol use. Thus, better
understanding of the current findings may be gained through closer examination of the factors potentially involved in SZCs’ drinking decisions, which were less likely to lead to alcohol problems among those with severe positive and cognitive symptoms.

**Alcohol and Self-Medication: Revisited**

Relevant research suggests that SZCs who drink alcohol report that they do so not only to remedy their psychotic symptoms, but also for many of the same reasons as non-SZCs do (e.g., Bellack & Gearon, 1996; Carey & Correia, 1998; Carey et al., 1999; Fowler et al., 1998; Mueser et al., 1995b; Spencer et al., 2002). These include the desires to enhance pleasurable engagement, cope with negative emotions, and achieve social goals (e.g., Arndt et al., 1992; Bergman & Harris, 1985; Brunette et al., 1997; Cooper, 1995; Cooper et al., 1992; Drake & Mueser, 1996; Farber, Khavari, & Douglass, 1980; Mueser et al., 1992a; Phillips & Johnson, 2001; Test et al., 1989). Yet, despite these speculations and SZCs’ self-reported attributions, the idea that SZCs use alcohol to address their symptoms is without much empirical basis (e.g., Blanchard et al., 2000; Brunette et al., 1997; Mueser et al., 1998).

However, there are indications that non-SZCs who report drinking for “coping” reasons tend to drink heavily and score high on indices of trait anxiety (Cutter & O’Farrell, 1984; Parry et al., 1974; Reardon, Patrick, & Lang, 2002; Stewart, Loughlin, & Rhyno, 2001; Stewart and Zeitlin, 1995). Furthermore, it has been suggested that the expectation for alcohol’s anxiolytic effects may account for the frequent comorbidity of anxiety disorders and AUD more generally (Brady & Lydiard, 1993; Kushner, Abrams, & Borchardt, 2000) and research pointing to higher rates of depressive syndromes and anxiety disorders among dually-diagnosed SZCs (Bartels et al., 1992; Cassano et al., 1998; Goodwin et al., 2003; Heilä, et al., 1997; Krausz et al., 1996; Stratowski et al., 1994) has been taken by some to suggest a similar mechanism might underlie the AUD-SZC link (Blanchard et al., 2000). Indeed, one study found that among persons with schizophrenia or SZA, those with high trait Negative Affectivity were especially likely to report that they tend to cope with stress via alcohol or drug use rather than utilize more adaptive, direct coping strategies. Further, those SZCs with high Negative Affectivity who drank for coping reasons seemed to encounter more problems as a result of their alcohol use (Blanchard et al., 1999) much like in research with non-SZCs who drink to cope (e.g., Cooper et al., 1992).

Of course, the coping process itself requires the abilities to perceive an event as threatening and/or stressful and to bring to mind a potential coping response (Blanchard et al., 2000). Thus, the notion that SZCs drink alcohol to regulate negative emotions presumes these cognitive processing skills. To break the sequence down further, drinking to cope may require:
(a) a basic level of insight and awareness of their symptoms, (b) that the elements of a situation, including symptoms, are processed in such a way as to feel distressed or threatened, (c) the identification and then selection of alcohol from a variety of response alternatives, and (d) execution of the response (drinking). Although the current study results cannot address the role or certain personality traits in coping responses (viz. Blanchard et al., 2000), it would appear that individual differences in cognitive functioning could also explain the link between drinking to cope and its adverse consequences among SZCs, albeit not in the initially predicted direction. In the following section, I speculate about how POS and COG symptomatology may interactively impact the underlying processes of self-intervention and their outcomes.

Ignorance is Bliss…or at Least Indifference

The mechanisms underlying SZCs’ insight into their mental illness are pertinent to the present discussion because the ability to formulate such insights has been linked to efforts to cope in relevant populations (Lysaker et al., 2002; Potkin et al., 2002). Although some research indicates no significant associations between insight and SZC symptoms exists (Brüne, 2003; David et al., 1995), others have supported an inverse relationship between insight and positive symptomatology (Vaz, Bejar, & Casado, 2002). For example, one examination of persons with recent-onset psychosis revealed that better insight, defined as “awareness” of the mental illness, was significantly predicted by fewer positive symptoms, even after controlling for the significant contribution of depressive symptoms. Variance in negative symptomatology and performance on a task measuring abstract thinking ability were also accounted for in this study, and were not found to contribute meaningfully (Collins et al., 1997). Other studies that have pointed only to the neurocognitive underpinnings of poor insight, particularly when executive cognitive functions were evaluated (e.g., Lysaker et al., 1994; Lysaker et al, 2003; Young, Davila, & Scher, 1993), have generally not accounted for variance attributable to other SZC symptoms, and thus may have overlooked the complex relationships among them.

In the present study, scores on the PANSS item tapping “insight” (GP12) partially depended on assessment of the resident’s awareness and understanding of mental illness and life circumstances, as evidenced by identification of past and current symptoms and recognition of need for treatment. Although this item was most closely attached to COG, it also showed a relatively high loading on POS as compared to the other symptom components. Considering these connections, it could be that SZCs who receive enhanced stimulus input from their external and internal environment (i.e., severe hallucinations, delusions) but lack the capacity to cognitively process the incoming information, may be essentially unaware of the distorted nature
of their experiences and/or its adverse impact on their overall functioning. Given that a large majority of SZCs can identify the symptoms of their mental illness (Talley, 1999), perhaps due to repeated contact with the mental health treatment system, it may be that the combination of high POS/high COG is particularly limiting in complex situations, such as social situations, the most common context for alcohol use. Indeed, relevant research has shown that although most SZCs accurately identify the sights and sounds of interpersonal situations (simple factors), they typically make incorrect inferences regarding the rules, goals, and affect (complex factors) that govern social interactions (Corrigan & Nelson, 1998). Of course, SZCs with AUD also may show limited awareness into their mental health problems (e.g., Osher et al., 1994), but it could be that persons with a combination of poor reality testing and cognitive deficits may be blissfully -- or at least indifferently -- ignorant of the impact of their mental health problems on their psychosocial functioning. This would seem to obviate any need to alleviate distress arising from them. It follows that worse COG and more severe POS could potentially serve to minimize the appeal and use of alcohol as a coping mechanism, thereby reducing risk for AUD in a manner consistent with the present findings. By contrast, SZCs with positive symptoms who are more in tune with their distorted experience and recognize its deteriorative impact on their functioning may engage in active efforts to offset them, perhaps through alcohol use. Of course, such interpretations assume that those SZCs who processed the elements of a situation -- even if in a distorted way -- had an emotional reaction (e.g., perceived threat, distress) from it.

Although ostensibly affective symptomatology (i.e., ANX/DEP, EXC/HOS) was statistically controlled, the present study was not designed to examine the role of such symptoms in AUD development among SZCs. However, relevant theory suggests that in essence, emotional reactions are initially little more than physiological responses produced out of awareness, but are experienced consciously as affectively-charged because the process of learning and memory assigns some emotional valence to previously neutral stimuli. In other words, the derivation of affective meaning is a cognitive process (LeDoux, 1989). Thus, many emotions can be regarded as a subset of cognitive processes, and thus may be subject to the impact of cognitive limitations associated with the SZC symptomatology. In the case of schizophrenia, the breakdown of this connection is evidenced by grossly inappropriate affect (e.g., giggling for no apparent reason), a diagnostic symptom of schizophrenia indicative of disorganization (APA, 1994), but one that is not directly assessed by the PANSS.

There also is evidence to suggest that cognitive symptoms such as impaired attention, which loaded on COG in this study, and stereotypical thinking, adversely impact SZCs’ ability
to recognize affect (e.g., Poole, Corwin, & Vinogradov, 2000). Additional inferences about SZCs’ interpretation of emotion can be gleaned from research examining the impact of affective stimuli on language production. Specifically, the available evidence suggests that SZCs tend to exhibit more “reference disturbances,” that is, words or phrases that obscure meaning of communication, when discussing negative as compared to positive or neutral topics. Interestingly, such errors were found to be most prominent among the subset of SZCs who scored high on the PANSS items identified with the COG factor as defined in the present study (see Burbridge & Barch, 2002). Thus, if cognitively-compromised SZCs’ are unable to adequately process affective information in general, or negatively-valenced in particular, this would seem to obviate any inclination to drink for coping reasons and thus could produce results consistent with those observed in the present study. Certainly, deficits in the experience of emotion also have the potential to make social contact less rewarding (Sayers et. al., 1996), an effect that could motivate consumption for the purpose of coping specifically with social isolation and unmet needs for affiliation (Arndt et al., 1992; Bergman & Harris, 1985). However, as discussed above, poor reality testing coupled with cognitive deficits implies limited awareness of overall functioning, which would include social functioning, meaning that individuals with this symptom presentation may be unlikely to compensate for social shortcomings via drinking.

Recent research suggests that specific cognitive deficits (verbal memory, executive cognitive functions) may be associated with a more restricted range and limited use of adaptive coping mechanisms by persons with schizophrenia, thereby limiting the flexibility by which SZCs select and employ a variety of coping skills to address their problems (Wilder-Willis et al., 2002). I had speculated that SZCs with similar cognitive deficits might rely more heavily on maladaptive coping strategies like alcohol use, with a narrow focus on its immediate positive impact and without due attention to, or at least a failure to make the connection with, its potentially adverse ramifications. In retrospect, however, this interpretation would seem to presume that alcohol use was within the repertoire of available coping mechanisms in the first place. In other words, the assumption that the cognitive deficit lies in the inability to self-regulate presumes that certain coping response alternatives were accessible and could be “brought to mind” (Blanchard et al., 2000). However, the availability of coping strategies may be largely dependent on experience with, or exposure to, coping options even prior to illness onset (viz. Arndt et al., 1992).

**The Role of Premorbid Social (Mis-) Connection**

As noted in the introduction, few would question whether dually-diagnosed SZCs experience worse outcomes than their non-addicted counterparts following illness onset.
However, there is some evidence to suggest that SZCs who use substances have a history of better pre-morbid adjustment relative to those who abstain (Arndt et al., 1992; Dixon et al., 1991; D'Mello et al., 1995; Kirkpatrick et al., 1996; Krausz et al., 1996; Sanguineti & Samuel, 1993). In their “Stage Model,” Arndt and colleagues (1992) specifically implicate the role of premorbid functioning in the development of substance disorders, like AUD. Intact premorbid functioning, they contend, promotes contact with substances, which, in turn, increases the risk for later development of substance disorders. Specifically, in the first stage, persons with better overall premorbid functioning may have more exposure to those who use substances, experience more social pressure, and maintain more confidence in their (substance-using) peers, relative to those with poorer premorbid adaptation (Arndt et al., 1992). Further, associations with substance-using peers has been shown to mediate expectancies for the stress-dampening effects of alcohol (Blanchard et al., 1999; Wills & Dishion, 2004) which may contribute to “drinking to cope” which has been linked to adverse outcomes (e.g., Cooper et al., 1992). Consequently, individuals exposed to substances by virtue of their better premorbid psychosocial adjustment may be more likely to regard substance use as a coping option, which potentially accelerates use to clinical levels as the adverse consequences arising from the onset of psychoses – particularly positive symptomatology -- mount in the second stage (Arndt et al., 1992). In contrast, persons with impaired premorbid adjustment, with its associated lack of social experiences and/or interest in social contact (Sayers et al., 1996), would have fewer opportunities to be introduced to substance use as a potential option, thereby reducing the likelihood of use both before and after illness onset (Arndt et al., 1992; Mueser et al, 1998).

Moreover, relevant research suggests a significant association between premorbid adjustment and the degree of neurocognitive impairment evidenced after schizophrenia onset (Carpenter et al., 1999b; Silverstein, Mavroleftheros, & Close, 2002). In one study, for example, interview and archival review procedures were used to examine various premorbid adjustment factors, such as school adaptation and the nature of peer relationships, in a sample of outpatients with schizophrenia. Participants were also subjected to a battery of neuropsychological tests. Results indicated that the psychosocial aspect of school adaptation, rather than academic performance, predicted later cognitive dysfunction. In particular, the quality and extent of peer relationships in early childhood predicted performance on measures of attention and executive cognitive functions, such that those with worse cognitive functioning in adulthood had a history of social adjustment problems predating their illness. Interestingly, however, premorbid psychosocial factors such as social withdrawal and aversion to social
interaction -- characteristics akin to negative symptomatology -- were unrelated to neurocognitive performance (Silverstein et al., 2002). Thus, the role of psychosocial adjustment in later cognitive functioning may have more to do with impairment, rather than disinterest, in peer relationships. Similarly, the present analyses also did not support either an interactive or mediational relationship between NEG and COG as they relate to alcohol problems.

Considered together, research on premorbid adjustment and neurocognitive performance seems consistent with the current observation of greater alcohol problems among those with better cognitive functioning. That is, those SZCs with intact cognitive functions (low COG) would be more likely to be among those with better, more extensive peer groups prior to illness onset, conditions that could promote their exposure to, and probable use of, alcohol. Indeed, relevant research has found that whereas a majority of the dually-diagnosed experienced their first psychotic episode following a period of social dysfunction, this sequence was less frequently observed among SZCs without AUD (Cleghorn et al, 1991).

It is possible that unfavorable premorbid social adjustment instigates a sequence of events and experiences that impact subsequent socialization and school performance, which in turn, hinder the development and maturation of cognitive processes (Silverstein et al., 2002). In contrast, observed differences in social functioning might be best considered a consequence, rather than a cause, of cognitive deficits (Holthausen et al., 2002) – even at the premorbid level. As noted above, social problem solving involves not only awareness (orientation), but also complex and controlled cognitive processes, such as processing critical elements of a situation, identifying and selecting from response alternatives, and performing appropriate behavioral routines (Bellack et al., 1994). It has been suggested that from an early age, certain SZCs may ineffectively attend to the more subtle, contextual cues when determining the relative salience of information in their environment, which, in turn, hinders their navigation of situations where contextual cues are complex and numerous, such as social interactions (Smith et al., 2002). Their functioning likely is further impaired with illness onset, as incoming sensory information from the environment presumably becomes more disturbed (Corrigan & Nelson, 1998; Smith et al., 2002), particularly for those suffering from positive symptomatology.

Moreover, positive symptoms, such as hallucinations and delusions, could serve to provide faulty information concerning one’s environmental context. If it is assumed that at least a rudimentary level of cognitive ability is needed to procure alcohol in the first place (Addington & Addington, 1996; Dixon et al., 1991), then such misinformation, coupled with an inability to navigate the environment due to severe cognitive limitations, could limit access to alcohol. In
other words, those with a combination of High POS and High COG may be unable to carry out the sequence of behaviors required to effectively execute the response of drinking on their own due to their cognitive limitations. Given their aforementioned restricted or even absent social networks, SZCs with worse COG also are probably less likely to have others obtain alcohol for them (viz. Kirkpatrick et al., 1996).

Considered together, findings from the current multivariate analysis of SZC symptoms as they relate to AUD potentially suggest not abandonment, but rather a modification of self-medication comorbidity theories. Traditional self-intervention via drinking models may not be applicable to all SZCs, or even to those with severe positive symptoms. If they were, a bivariate association of POS and alcohol problems would have been observed in this study, but it was not. The present results could suggest that it is the subset of SZCs with preserved cognitive functions that may be most prone to self-medicate with alcohol in the face of acute positive symptomatology (i.e., High POS, Low COG), mainly because they are the ones who are capable of carrying out the various cognitive processes implied by “drinking to cope.” In contrast, cognitively-compromised SZCs may be less aware and/or less bothered by their symptoms and/or their impact on functioning, meaning that they would be less inclined to drink to alleviate distress arising from them. Moreover, SZCs with severe cognitive symptomatology are among those who may be less likely to have been exposed to the use of alcohol as a coping option premorbidly, so that even if confronted with distressing positive symptomatology they may not “know” to turn to alcohol. In the case of severe cognitive limitations, the basic skills required to procure alcohol also may be lacking.

In sum, a case can be made that differences in inclinations to use alcohol to cope may be best explained as a consequence of chronic, underlying cognitive impairment and its associated premorbid correlates. Although there were insufficient data concerning differences in premorbid cognitive functioning to evaluate these speculations directly, the current findings seem to correspond closely with the limited research that is available on variance in AUD rates across subgroups of SZCs with known premorbid and cognitive correlates.

Relevance to AUD Rates among Identified Subgroups of SZCs

As noted in the Introduction, some research has pointed to higher rates of AUD among the DSM Paranoid Subtype relative to other groups (Alterman et al., 1981; Soyka et al., 1993) and the current findings are consistent with that observation. That is, the combination of symptoms associated with a lower risk for AUD (high POS/high COG) would be less characteristic of the Paranoid Subtype, which, by definition, is probably best represented by
severe delusions and hallucinations (high POS) with preserved cognitive functions (low COG). Furthermore, Paranoid Type SZCs show better premorbid functioning, later onset of psychosis, and relatively preserved cognitive functioning (APA, 1994; Cardno & Farmer, 1995; Kremen et al., 2000), all of which either independently or interactively might facilitate greater peer involvement, at least prior to illness onset, relative to other subtypes, thereby promoting their exposure to, and probable use of, alcohol (viz. Arndt et al., 1992). Future investigations examining the correspondence of the PANSS factors to SZC subtypes should not only allow for more direct comparisons of the present study results with research on variance in AUD rates across the DSM categories, but also have the potential to inform current diagnostic conceptualizations of schizophrenia.

Indeed, there is continued debate among schizophrenia researchers about how best to define the heterogeneity observed in SZC symptomatology (e.g., Berman et al., 1997; Cardno & Farmer, 1995; Herbener & Harrow, 2001; Garver, 1997; Goldberg & Weinberger, 1995; Lenzenweger, 1999; Peralta & Cuesta, 1994; Vorgunti et al., 1997; Seaton et al., 2001; Goldberg & Weinberger, 1995). Although some have favored dimensional approaches similar to that applied in the present study, others have pushed for the identification and empirical isolation of subgroups sharing in some major psychopathological component of the schizophrenia syndrome. For example, there has been much research on the clinical, neurological, and etiological correlates of a homogeneous subgroup of SZCs defined by the presence of trait negative symptomatology and known collectively as Deficit Syndrome. Interestingly, the clinical indicators of the Deficit Syndrome include poor insight, impaired sensory integration, restricted affective reactivity, and poor premorbid social development (see Carpenter, Heinrichs, & Wagman, 1988; Carpenter et al., 1999a/b), processes which were identified in this discussion as potentially critical to efforts at self-intervention via drinking. Indeed, consistent with these speculations, those identified as Deficit Syndrome show lower rates of alcohol and drug problems compared to non-Deficit SZCs, a group that likely subsumes the DSM Paranoid Subtype (Kirkpatrick et al., 1996).

However, the present application of the PANSS as the sole measure of SZC symptomatology could limit efforts to classify the current sample into Deficit and Non-Deficit groups, meaning I can only speculate about the correspondence of these findings with the available literature on correlates of the Deficit Syndrome. Specifically, the trait negative symptoms that define the Deficit Syndrome must be primary, arising directly from the schizophrenia illness, not secondary to other conditions (e.g., depressive anhedonia, paranoid
avoidance) or medications. Yet, the PANSS does not distinguish whether or not symptoms are primary manifestations of schizophrenia (Carpenter et al., 1999a/b; Kirkpatrick et al., 2000; Kirkpatrick et al., 2001), a distinction that is probably difficult to determine via clinical inference alone (Sayers et al., 1996). It has been argued that when negative symptomatology is defined broadly, as on the PANSS, its relationship to other variables may be attenuated by the presence of transient, “secondary” negative symptoms (e.g., Johnson-Selfridge & Zalewski, 2001). Thus, it is possible that previous research demonstrating the association of PANSS-derived NEG and cognitive functions (Herbener & Harrow, 2001; Johnson-Selfridge & Zalewski, 2001; Lindenmeyer et al., 1994; Lancon et al., 2000) may have been confounded by the simultaneous measurement of primary and secondary negative symptoms, perhaps in partial explanation of the lack of association between NEG, COG, and AUD in the present study.

Nonetheless, research on five-factor solutions of the PANSS has supported the stability of the NEG factor despite medication washout, indicating that NEG may be a primary component unconfounded by any secondary impact of medication side-effects (Lindenmeyer et al., 1995). Additional concerns about whether symptoms attached to NEG were secondary to depression (anhedonia), anxious avoidance, or paranoid social withdrawal may be further lessened by the identification of a mood component (i.e., ANX/DEP) and separable component loading for P6 – Suspiciousness/Persecution in the present study. However, others have questioned whether ANX/DEP components derived from the PANSS constitute a valid proxy for depression in schizophrenia (Lancon et al., 2000). In a recent study, standardized measures of depression were found to correlate not only with the ANX/DEP component derived from factor analysis of the PANSS, but also with NEG and an excitability component (ElYazaji et al., 2002). Thus, it would appear that secondary depressive symptoms may be captured by other PANSS components, such that the simple separation of ANX/DEP from NEG may be insufficient to suggest that the NEG component derived in this study reflects primary SZC pathology alone.

Of course, only about 20% of SZCs are identified with the Deficit Syndrome (Carpenter et al., 1999a/b) and hence probably constitute only a subset of SZCs who may be at a reduced risk for AUD. Moreover, reminiscent of the problems introduced by a Paranoid/Non-Paranoid classification schemes (Cardno & Farmer, 1995), the Deficit Syndrome is defined by specific criteria, making them a more homogeneous group than non-Deficit SZCs, which includes a larger composite of all other SZCs. Thus, observed differences in dual-diagnosis could emerge as an artifact of this differential symptom variability within each classification (viz. Sorensen et al., 1988). Indeed, the heterogeneity of symptoms characteristic of the remaining non-Deficit
group makes it impossible to determine whether it is the absence of deficit symptoms that makes the entire group susceptible to alcohol problems or perhaps the presence of other symptoms placing only certain members of this group at risk. Indeed, the present results suggest that the presence of POS symptomatology may be important in determining risk for AUD among the subset of presumably Non-deficits with fewer cognitive deficits.

In general, questions could be raised about whether PANSS ratings adequately assessed the “primary” nature of any of the Schizophrenia symptom dimensions. However, support for the clinical importance of identified dimensions in accounting for SZC heterogeneity, particularly NEG, POS, and COG, can be gleaned from the fact that similar patterns of results have been obtained when factor analytic procedures are applied to other measures of the SZC symptomatology, such as the SAPS/SANS (Andreasen, 1982) and the Brief Psychiatric Rating Scale (e.g., Grube et al., 1998; Mueser et al., 1997; Peralta & Cuesta, 1999). Future research could be directed at clarifying those demographic or clinical variables that vary systematically with item loadings, such that items of the PANSS, perhaps in combination with other SZC symptom measures, may be identified more precisely with the five symptom dimensions. Such research would allow for more valid comparisons of resultant factor scores and their relationships to relevant clinical variables, such as AUD, across SZC samples and subgroups.

Although I have illustrated how the present study findings may be understood by, and fit with, theory and research from the schizophrenia and dual diagnosis literatures, the observed role of COG in AUD development was clearly at odds with initial predictions and begs for replication and further explanation. In an effort to account for the discrepancy, a discussion of the potential methodological and theoretical limitations of the present study follows next, identifying areas of future research which perhaps better integrate insights from relevant alcohol research into current understanding of the AUD-SZC connection to inform clinical intervention.

Limitations and Future Directions

Questionable Construct Validity of COG

The application of COG as a practical means for evaluation of cognitive impairment associated with the SZC symptomatology was supported by a body of research pointing to the connection between PANSS-derived COG and performance deficits on several different tests of executive cognitive processes by persons with schizophrenia (e.g., Bell et al., 1994b; Kay & Sevy, 1994a). As noted previously, the present factor analysis results were remarkably similar to those observed in previous investigations of the PANSS (see Appendix D-1). Moreover, the present index of COG corresponded closely to models emphasizing a comprehensive set of
cognitive dysfunctions in addition to formal thought disorder, such as attention deficits, rigid/barren thinking, and failures in illness-awareness. Such inclusive definitions of COG are closely aligned with the sorts of performance deficits SZCs show on neurocognitive tests, both in theory and in research (Bryson et al., 1999). Confidence in the validity of the construct assessed by the present cognitive index was further increased by present efforts to statistically evaluate and/or control for those variables which may otherwise confound its association with neuropsychological test measures (e.g., age, number of hospitalizations, anxiety and mood symptoms, medication status). Nonetheless, the present model constituted a relatively poor fit to the study data, a finding not inconsistent with other confirmatory factor analyses of the PANSS (e.g., Fitzgerald et al, 2003; Mueser et al., 1997; Nakaya et al., 1999), but one that potentially limits definitive conclusions about what truly caused variance on COG.

Currently, there is little consensus about the precise nature of cognitive deficits in schizophrenia or which neurocognitive functions impair which areas of life (Green, 1996; Rund, 1998), due in part to failures to equate true score variance when comparing SZCs’ deficits on tasks of varying difficulty (Chapman & Chapman, 1973; Chapman & Chapman, 1978; Chang & Lenzenweger, 2001; Knight & Silverstein, 2001). Certainly, the degree to which cognitive performance deficits in schizophrenia may be unreliable impacts the validity of its purported associations with PANSS-derived COG in previous research. Moreover, much of the research demonstrating a relationship between PANSS-derived COG and performance on neuropsychological tests has been conducted with non-acute, medication-stabilized SZCs without comorbid organic or substance disorders (e.g., Bell et al. 1994b; Berman et al., 1999; Mass et al., 2000). In contrast, very few participants were excluded from the present study on the basis of cognitive or clinical factors and for obvious reasons persons with substance disorders were included. Furthermore, although most of the study participants were medicated at the time of admission, the majority underwent subsequent medication changes, perhaps suggesting the inadequacy of their former treatment regimen in addressing their symptoms. Thus, the PANSS data utilized in the present study probably represented symptoms of an acute episode, undiluted by psychotropic intervention, even among those chronically afflicted. Such methodological differences could account for the greater variance in the present index of COG relative to that derived from previous research supporting its use as a proxy for neurocognitive impairment. Indeed, a recent study observed that variance in certain functional outcomes, like work performance, was attributable to performance on a cognitive battery of tests, but not COG “abilities” assessed by the PANSS (Bell & Bryson, 2001).
Thus, to the extent that COG taps variance in constructs other than the cognitive deficit symptoms associated with performance on neuropsychological tests (Carpenter et al., 1999a), it may be less effective for evaluating the sorts of deficits manifested with the acute effects of alcohol or by persons with non-comorbid AUD initially predicted to underlie the AUD-SZC connection. Future research aimed at integration of the alcohol research literature to explain the high rates of AUD among SZCs may benefit from direct assessment of the “parallel” skill deficits, such as decision-making, memory, or attention, in predicting AUD. Certainly, a comprehensive neuropsychological test battery tapping all relevant abilities would be unsuitable, particularly given the compromised attentional capacities of many SZCs. However, a series of studies utilizing neurocognitive measures that reliably differentiate SZCs from non-SZCs, may be an appropriate start for this purpose (Johson-Selfridge & Zalewski, 2001). Given the relationship of premorbid functioning with neurocognitive deficits (viz. Sliverstein et al., 2002) and its potential impact on available coping strategies, specific skills in social cognition may prove important to the development of AUD in this population. Investigations along these lines could not only elaborate upon the role of cognitive deficits in AUD, but also might have the potential to elucidate the sorts of cognitive capacities actually tapped by COG derived from the PANSS if administered concurrently with objective measures.

In this connection, a recent study found that higher scores on a cognitive dimension (i.e., formal thought disorder, mental confusion, distractibility, concrete reasoning) was associated with SZCs’ self-reported feelings of “information overload” when evaluated on indices of executive functions, but neither the factor nor subjective reports were indicative of actual performance (Holthausen et al., 1999). On the face of it, the subjective sense of “information overload” appears rather similar to the difficulties SZCs tend to experience when dealing with enhanced stimulus input from the environment (i.e., high POS) discussed above with regard to the processes involved in coping as they relate to the present findings. In further support of this possibility, an investigation of non-SZCs with alcohol disorders revealed that self-ratings of cognitive impairment were unrelated to actual performance deficits, but were instead correlated with depression and vulnerability to stress (Horner, Harvey, & Denier, 1999a). Thus, future studies may benefit from an evaluation of subjective measures of cognitive ability in the relationship between PANSS-derived COG and AUD, perhaps pointing to difficulties SZCs encounter in their daily lives that are not assessed by objective measures (Holthausen et al., 1999), but that relate more closely to self-regulation and hence AUD.
Of course, it is also possible that slightly different dimensions of SZC symptomatology might have been obtained had another measure of SZC symptoms been applied in the present study (Bryson et al., 1999). For example, factor analyses of the Schedule of Negative Symptoms (SANS; Andreason et al., 1984) have yielded three negative symptom dimensions, including “Diminished Expression,” “Inattention-Alogia,” and “Social Amotivation” (Sayers et al., 1996), a refinement of NEG not possible in the current study. Thus, other symptoms, such as affective dysregulation or “information overload,” may constitute indicators of the cognitive deficit symptom construct relevant to AUD, but simply were not assessed.

In sum, the current evaluation of the role of cognitive symptomatology in AUD was restricted to cognitive symptoms included on the PANSS and thus potentially vulnerable to unexplained variance in this dimension. Furthermore, the present data collection procedures may have introduced additional problems not only for resolving this variance, but also for making any directional interpretation of the study findings.

**Cross-Sectional Data Limitations**

Given that PANSS data were collected upon admission, perhaps at the point of an acute exacerbation, the long-term stability of the resultant symptom components, and hence their relationship to AUD, cannot directly be ascertained. Although previous research has suggested a limited impact of neuroleptic medication on cognitive abilities, anticholinergic medications have the potential to disrupt memory functioning (Johnson-Selfridge & Zalewski, 2001; Spohn & Strauss, 1989). The stability of the construct tapped by COG is assured by evidence of its robustness to differences in age, duration of illness, education, or antipsychotic treatment (Amminger et al., 2002; Daneluzzo et al., 2002; Lindenmeyer et al., 1995). However, positive symptomatology is often effectively ameliorated with antipsychotic intervention (Barch et al., 2002; Johnson-Selfridge & Zalewski, 2001) and has been suggested to reflect transient pathology (Blanchard et al., 1999; Soyka et al., 2001). Although some research supports the stability of POS despite medication (Goldman et al., 1991), the dependent relationship of POS to COG with AUD risk observed in the present study may not prove consistent and/or perhaps could have limited generalizability to medication-stabilized SZCs. Assuming that certain SZC symptoms can and often do vary across acute episodes within the same individual, longitudinal research will be needed to determine whether these relationships hold over time.

Indeed, inferences concerning the temporal sequence of schizophrenia symptoms and AUD remain entirely speculative in the absence of longitudinal study (Blanchard et al., 2002; Herbener & Harrow, 2001; Mueser et al., 1998). Although Structural Equation Modeling (SEM)
allows for some causal inference about relationships among variables, the current data were insufficient to test these relationships adequately. Indeed, the “predictor” symptom dimensions were assessed at a point in time subsequent to the emergence of any alcohol-related symptoms. Thus, although it was speculated that POS and COG independently and interactively impact the process of coping, which in turn impacts AUD development, the possibility of an opposite temporal sequence, a bidirectional association, and/or the contribution of another variable (e.g., personality traits) in this relationship, cannot be ruled out. One potentially useful follow-up investigation could evaluate whether variance in POS and COG, or the interaction thereof, predicts which of the SZCs non-problematic drinkers and/or abstainers develop AUD at some later point of time. Based on the present findings, I would predict that non-problematic drinkers/abstainers with worse positive symptomatology and poor cognitive functioning would be least likely to develop an AUD.

Additionally, the emphasis on recent-year alcohol use/problems in the present study may have introduced some unexplained “noise” with regard to the non-problem group. For example, lifelong abstainers could not be differentiated from former non-problematic drinkers who abstained in the year prior to their admission. However, this distinction may be important with regard to the present results, as some research with non-SZCs suggests that lifelong abstainers may be more rigid in their thinking and suffer greater psychosocial disadvantages than those who drink alcohol moderately (e.g., Brodsky & Peele, 1999; French & Zarkin, 1995; Lipton, 1994; Neff & Husaini, 1982; Nezlek, Pilkington, & Bilbro, 1994). Accordingly, closer examination of the longitudinal symptom profile of the SZC lifelong “teetotaler” relative to the “quitter” may have the potential to address whether chronic, underlying cognitive impairment and its associated premorbid correlates impact drinking decisions in the manner proposed to account for the present study findings. Moreover, a substantial minority of the non-problematic drinkers and abstainers met CRS criteria for problematic alcohol use at some point in their lives, perhaps in partial explanation for the greater variance in COG in this group relative to the problem drinking group (see Appendix C-2). Consequently, it is also possible that observed differences in cognitive pathology reflect remnants of a prior, perhaps more severe course of AUD among members of the non-problematic group relative to current problem drinkers, who perhaps had not yet reached this level of AUD severity. Thus, although it was speculated that COG and POS interacted to prevent alcohol use and hence AUD in this group, it is possible that a subset of the former problem drinkers discontinued use prior to the onset of these SZC symptoms. Future studies will need to disentangle whether the onset of positive and cognitive symptomatology
interacts to curb problematic use of alcohol or if perhaps other factors operate to explain recovery from AUD in this population.

Although the symptom factors involved in abstinence, non-problematic alcohol use, and recovery from AUD may be improved by longitudinal study and/or examination of past alcohol involvement, results are dependent on efforts to ensure that reliable and valid estimation of both past and present AUD. Suggestions in this regard are offered next via examination of the assets and liabilities of the current AUD assessment procedure.

**Clinician-Rating Scale and AUD Rates**

The CRS was selected for use in the present study not only because of questions raised about the validity of chart diagnoses in inpatient psychiatric populations (e.g., Anath et al., 1989; Barbee et al., 1989; Blanchard et al., 2000; Goethe & Ahmadi, 1991; Kirchner et al., 1998) but also because it allowed for a refined assessment of alcohol use and problems spanning the full range of severity. Despite some minor methodological variations across raters, reliability estimates were excellent for each of the CRS categories of AUD. However, when category assignments were compared against residents’ self-reported alcohol use/problems on the AUDIT only distinctions between non-problem and problem groups demonstrated valid for the study purpose. Thus, the advantages of broader range using this alternate methodology over a simple dichotomous AUD variable were not realized. Of course, ratings of alcohol use/problems and other potentially relevant data were dependent on the volume and quality of information provided in chart records, which for some coded variables were rather scant (e.g., quantity/frequency of consumption, premorbid functioning, etc.).

In addition to problems with diagnosis of AUD by personnel overseeing inpatient SZC populations, clinicians’ assessment of simple alcohol consumption has also been questioned in some research (Carey et al., 1996; Dawes, Frank, & Rost, 1993). Thus, it could be that in the absence of any documented history of AUD or obvious alcohol-related problems, information regarding alcohol use was simply not elicited from patients or collateral sources, perhaps in part because psychotic symptoms or legal questions constituted the major focus of clinical attention. Alternately, if alcohol use history was acknowledged, but deemed non-problematic, information concerning simple use patterns may be considered superfluous for inclusion in the chart. In either case, such data limitations complicate distinctions between Abstinence and Use Without Impairment on the CRS. Although these categories were reliably coded, this distinction was not consistent with resident self-reports, perhaps partially because data concerning simple use patterns were not recorded in the chart records of those without alcohol problems. Apart from
the methodological limitations introduced by this potential oversight, inquiry and documentation of simple use patterns seems especially important to prevention efforts insofar as any alcohol use by this population may introduce risk of future alcohol problems (e.g., Bartels et al., 1992; Drake et al., 1989). The AUDIT may prove to be a useful and practical screening measure in this regard in that it specifically taps hazardous use patterns, is simple and takes less than 10 minutes to administer, and showed a relatively high correspondence rate (71%) across all CRS ratings in the present sample.

Nonetheless, examinations of the discrepancies between self-reported alcohol problems with chart review indicated that a sizeable minority of the problem drinkers in the present study were inaccurate reporters of their alcohol use and consequent problems. Although some research suggests that self-report may be better than medical exams, laboratory tests, and collateral reports in correctly identifying substance problems among the seriously mentally ill, exclusive reliance on this methodology risks neglect of a substantial proportion of actual problem users (Wolford et al., 1999). Indeed, one study found that a majority of SZCs classified as having AUD using the subtle, less face-valid items of a self-report measure identified themselves as “normal drinkers” when queried directly (Smith & Pristach, 1990). Of course, efforts to dissimulate, minimize, or deny alcohol problems are quite commonplace among persons with primary AUD (Selzer et al, 1975). Among persons with schizophrenia or SZA, the failure to accurately disclose alcohol problems also has been attributed to their relatively poor ability to connect drinking to their symptoms (Bellack & Gearon, 1998; Carey & Correira, 1998; Drake et al., 1990; Drake et al., 1996; Goldfinger et al., 1996; Rosenberg et al., 1998; Test et al., 1989). Thus, the present estimates of the concurrent validity of the CRS may have suffered from these potential liabilities of self-reported alcohol use/problems. Although perhaps impractical for the purposes of the present study, distinctions between the CRS categories may have been supported had other sources of information been available for comparison.

Of course, the strengths of the present study’s multi-source, multi-method approach to AUD assessment should not be understated. Although the prevalence of current AUD among SZCs has been cited as 25-35% (D’Mello et al., 1995; Drake et al., 1989; Drake & Wallach, 1989; Osher et al., 1994), higher and potentially more reliable estimates may be obtained when more comprehensive methodologies are applied (e.g., Regier et al., 1990; Turner & Tsuang, 1990). Nearly half (46.4%) of the present sample was identified as having significant alcohol-related problems, a figure that corresponds closely with estimates obtained from research employing similar assessment methodologies (Fowler et al., 1998; Mueser et al., 1990; Mueser
et al., 1995; Mueser et al., 2000). The finding that CRS ratings did not distinguish among the levels of AUD severity may be due, in part, to the relatively small number of individuals meeting the criteria for the severe Dependence classifications, which reduces statistical power for detecting these differences. Nonetheless, observed rates were consistent with research evidence that SZCs with AUD are less likely to be assigned Alcohol Dependence diagnoses that typically require higher levels of use, and are more likely to experience consequence-based Alcohol Abuse diagnoses compared to non-SZC populations (Mueser et al., 1998; Noordsey et al., 1994). Thus, despite its potential shortcomings, the present results tend to support the use of CRS via chart ratings as a viable option for research with SZCs, among whom reliable AUD diagnoses are often difficult to obtain.

Certainly, several factors can impact estimates of AUD rates and should be considered in the interpretation of the clinical correlates of dual disorders (Mueser et al., 1992b; Mueser et al., 1998). In general, figures obtained from clinical samples may not denote the true frequency of AUD among the SZC population as a whole, as the additive effects of treatment utilization for each disorder can boost estimates of comorbidity (Berkson, 1946). In particular at FSH, the majority of the SZC population is committed for forensic reasons with the number of civil units diminishing as such treatment is increasing relegated to outpatient mental health centers. Additionally, although gender proportions are close to equivalent on civil units, the forensic units are predominantly male (90%). The present use of a disproportionately male sample may have exaggerated estimates of AUD relative to the entire SZC population as rates of this disorder are generally higher among men (APA, 1994; Kandel, 1997). AUD rates may have been further inflated by the high degree of co-morbidity between antisocial behavior and alcohol use problems found among men and especially males in forensic settings (e.g., APA, 1994; Collins, Schlenger, & Jordan, 1988; Lewis, Cloninger, & Pais, 1983; Smith & Newman, 1990). Additionally, failures of random selection yielded a sample slightly younger than the FSH population of SZCs, which is relevant in that substance misuse has been found to be associated with younger age in general (e.g., Cuffel & Chase, 1994; Drake & Wallach, 1993; Mueser et al., 1990; Mueser et al., 1992), and in SZC forensic samples in particular (Wheatley, 1998). Lastly, the current sample was largely comprised of Caucasians and African-Americans, groups whose rates of AUD may differ from those of other ethnic groups (Kandel et al., 1997). Thus, although the present sample was representative of FSH and more inclusive than previous research in this area, the results may apply less well to explain the AUD-SZC connection in ethnically-diverse, female, outpatient, civilly-committed, and/or untreated SZC populations. Of course, variance in
AUD attributable to many of these potentially confounding sample characteristics were either statistically controlled or tested and found to be of negligible relevance to the model, thereby increasing confidence in attributing variance in AUD to the key SZC symptom components.

It is noteworthy that despite the high rates of AUD observed, very few of the participants had a history of specialized substance disorder treatment, a finding perhaps reflective of the dissociation of alcohol and mental health treatment resources and administration (Calloway & Morrissey, 1998; Lett, 1988). Relative to non-problematic alcohol users, the problem users presented with higher rates of previous hospitalizations, but no differences in the total duration of previous hospital stays. This finding replicates previous research suggesting a relative lack of contact with community providers and a reliance on more frequent, brief hospital stays for stabilization among dually-diagnosed SZCs (Jackson et al., 2001). Thus, although psychiatric hospitals represent the setting where evaluation and treatment of AUD may be most critical, such treatment can be overlooked when other symptoms appear more pressing and in greater need of clinical intervention (Carey & Correia, 1998; Drake et al., 1996; Goethe & Ahmadi, 1991 Hambrecht & Hafner, 1996; Lehman et al., 1996). Indeed, approximately two out of every five problematic drinkers identified in this study were not diagnosed with an AUD by hospital personnel. Particularly neglected were those whose SZC presentation was characterized by significant anxious or depressive symptomatology. To the extent that above speculations about the processes involved in “drinking to cope” suffice to explain the current findings, obviously these “distressed” individuals may be most in need of AUD intervention. Future research could benefit from more careful examination of the role of anxious and affective symptomatology, perhaps as they relate to cognition and the processes involved in coping, in the development of AUD among SZCs.

Conclusion

This initial investigation applied multi-method assessment procedures and sophisticated analyses to elucidate the contribution of cognitive symptoms to the elevated rates of AUD observed in SZC populations, yielding some interesting, albeit unexpected information about the association. Despite the apparent parallels between the schizophrenia-related cognitive deficits and the effects of acute alcohol intoxication, in addition to the sorts of impairments displayed by addicted normals, worse cognitive functioning was associated with lower rates of alcohol problems among SZCs with severe positive symptoms. Although the current findings may suggest the need for some elaboration of the cognitive constructs tapped by COG, particularly among psychiatrically heterogeneous samples of SZCs, the present results can be understood
in the context of theory and research from the schizophrenia and dual diagnosis literatures. Specifically, it was speculated that severe cognitive deficiencies in the context of poor reality-testing may serve to limit alcohol use, both in general and as a coping mechanism in particular. In other words, those SZCs at greatest risk for AUD may be those who are functioning at a sufficiently high level to consider drinking to be a part of their available coping repertoire. This insight might be by virtue of a better premorbid adaptation, which left them capable of carrying out the various cognitive processes implied by “drinking to cope” in the face of, and perhaps in spite of, debilitating positive symptomatology. Notwithstanding this obvious disadvantage, SZCs with good cognitive functioning may be more receptive to educational interventions aimed at developing more adaptive alternative coping skills as well broadening sober social support networks. Although replication and future research is warranted, such interventions may hold the greatest promise for the prevention and remediation of alcohol problems in populations with SZC symptomatology. The present findings should help to generate further systematic research that will consider the social, affective, and ideally the developmental factors relevant to the cognitive processes of self-regulation as they relate to AUD. Toward this end, I have presented data to support a method by which problem alcohol users may be reliably identified in an important segment of the institutionalized mentally ill and have pointed to some potential clinical indicators of missed AUD diagnoses. Certainly, the high prevalence of AUD in this inpatient sample underscores the urgent need for continued investigation of the SZC-AUD connection.
1 Unless otherwise specified, both Alcohol Abuse and Alcohol Dependence disorders are referenced collectively as “Alcohol Use Disorders” or “AUD”.

2 Although “dual diagnosis” or “dually diagnosed” could be used to signify the co-occurrence of any two psychiatric disorders, these terms are employed exclusively to denote the comorbidity of schizophrenia disorders and AUD.

3 Some investigators have not observed higher rates of hospital recidivism among SZCs with AUD (e.g., Fowler et al., 1998; Gerding et al., 1998; Mueser et al., 1992a).

4 There is at least a 50% lifetime prevalence of co-occurring psychiatric disorders among persons with schizophrenia (Bermanzohn et al., 2000; Fenton, 2001; Green et al., 2003). In general, psychiatric comorbidity is associated with poorer prognosis in SZCs (Fenton, 2001; Green et al., 2003) and indeed, many of the negative correlates cited in connection with AUD -- the most frequent comorbid condition (Drake & Mueser, 1996) -- also are observed when other disorders co-occur with schizophrenia, the most common being drug disorders, panic disorder, obsessive compulsive disorder, social anxiety, and simple phobia (Cassano et al., 1998). For example, some correlates of comorbid Social Anxiety Disorder include higher rates of suicide attempts/completions as well as poorer interpersonal and overall adjustment (Pallanti, Quercioli, & Hollander, 2004). Obsessive Compulsive Disorder is associated with greater social isolation, more prolonged hospital stays, and poorer response to treatment (Green et al., 2003). Depressive symptomatology appears to be associated with greater risk of psychotic relapse and suicidality (Fenton, 2001; Green et al., 2003). Such observations could be taken to suggest that the negative correlates associated with AUD simply reflect the additive effects of any two disorders (i.e., the more disordered the individual, the more problems) rather than something unique to AUD. Certainly, the higher rates of depression and anxiety among dually-diagnosed SZCs relative those without AUD (Bartels et al., 1992; Goodwin et al., 2003; Heilä et al., 1997; Krausz et al., 1996; Pallanti et al., 2004; Stratowski et al., 1994) complicates efforts to disentangle the functional impact of a specific comorbid disorder and few researchers in this area have accounted for the impact of SZC heterogeneity let alone the potential contribution of other comorbid conditions. Importantly, in the present study, the effects of non-SZC symptomatology were statistically controlled and the impact of other disorders that may theoretically relate to AUD or cognition on the present study findings was examined empirically.

5 Although the research cited here appears to have been conducted with DSM-IV subtypes, some paranoid vs. non-paranoid conceptualizations of schizophrenia qualify the DSM criteria by a later onset (after 25 years) such that individuals identified as non-paranoid are marked by an earlier onset (before 25 years) and are predominantly comprised of a combination of the disorganized and catatonic SZCs (Cardno & Farmer, 1995). This additional age of onset...
requirement could account for some prognostic differences observed in paranoid as compared to non-paranoid SZCs (APA, 1994; Pishkin & Bourne, 1981), reflective of differences in premorbid functioning that are unrelated to true clinical heterogeneity. Indeed, some individuals with paranoid delusions could be classified as non-paranoid simply because they were compromised cognitively. Thus, observed prognostic differences between paranoid and non-paranoid schizophrenics probably stem from variations in cognitive ability, rather than paranoia per se. Alternatively, because the paranoid subtype is defined by specific and well-defined criteria, whereas all other SZCs are considered together as non-paranoid, the paranoid classification simply may be more homogenous than the non-paranoid group. Consequently, differences might emerge as an artifact of this differential symptom variability within these classifications (Sorensen, Gordon, & Mariotti, 1988).

In the present study, I opted to include persons with diagnoses of SZA but not other disorders involving psychotic symptoms (e.g., Delusional Disorder, Psychotic Disorder NOS) for several reasons. First, per the *DSM-IV* (APA, 1994) a diagnosis of Psychotic Disorder, NOS may be assigned in cases when psychotic symptoms are of uncertain origin, such as a medical condition or substance intoxication/withdrawal. Accordingly, cognitive deficit symptoms observed in persons with this diagnosis may not be tied to SZC symptomatology per se, thus confounding interpretation of any relationship of cognitive symptoms with AUD. Furthermore, at least one study has observed differences in certain cognitive abilities between SZCs and those diagnosed with “other” psychotic disorders (e.g., Major Depression with Psychotic Features, Psychotic Disorder, NOS, and Delusional Disorder), but not those with SZA. Whereas deficits in context processing were evident for all inpatients in that study presenting with psychotic symptoms in the acute phase of illness, these cognitive symptoms were evident only among those with primary schizophrenia and SZA at 4-week follow-up (Barch et al., 2003). Thus, it would appear that similar cognitive deficits may be shared among persons with schizophrenia and SZA, but not with other psychotic disorders. Second, although specific schizophrenia disorders may be reliably diagnosable in theory, they may not be in practice. Political or institutional guidelines can impact which diagnoses are ultimately recorded in chart records. For example, persons with primary schizophrenia may not be treated with both an antipsychotic and a mood stabilizer unless they have a recorded diagnosis of SZA -- regardless of whether the mood stabilizer was administered to treat a true diagnosable affective episode, reduce aggressive behaviors, or for other therapeutic reasons. In such cases, the recorded diagnosis of SZA and the various subtypes of schizophrenia may not distinctively represent actual clinical presentation. Of course, diagnostic distinctions are of less concern in the present study as relationships with AUD were analyzed with respect to SZC symptom dimensions assessed by a standardized measure. Indeed, a consistent, five-factor solution of the PANSS has been obtained in research with SZCs alone or when considered together with persons diagnosed with SZA (e.g., Hayashi et al., 2002; Lançon et al., 2000; Lindemayer et al., 1994; Lykourous et al., 2000; Von Knorring & Lindstroem, 1995). This research suggests that in addition to the core SZC symptomatology (i.e., POS, NEG, COG) affective and behavioral symptoms apply not only to SZA, but also to explain heterogeneity observed among the various schizophrenia subtypes. Thus, the symptom dimensions, rather than diagnoses, may better capture the range of clinical presentations common to these disorders. Lastly, the available research indicates no differences in the rates or correlates of Alcohol Abuse between SZCs and SZAs (Drake & Wallach, 1989; Mueser et al., 1992b).

Thus, when considered together, the available research supports the present inclusion of persons with SZA together with SZCs as they do not appear to vary along the key constructs of interest. Any remaining concerns about increased sample heterogeneity introduced by the inclusion of SZAs were addressed statistically. The present study examined association of AUD
to three symptom dimensions (POS, NEG, COG) ostensibly shared to a similar degree among SZCs and SZAs, while also statistically accounting the impact of mood/behavioral disturbances (i.e., ANX/DEP, EXC/HOS) which might, by definition, otherwise differentiate SZA from the other subtypes of schizophrenia. We further assessed differences in the results of the present study as a function of primary diagnosis (i.e., schizophrenia vs. SZA) as well as presence of certain comorbid disorders (e.g., Axis II personality disorders, other substance use disorders, etc.) which could confound interpretations of the relationship of schizophrenia symptoms and AUD.

7 At FSH, the determination of competency to consent to treatment and/or medication is based primarily on a clinical interview conducted by the Service Team Psychiatrist. Any person identified as incompetent requires intervention from a guardian advocate or the Court to make decisions impacting their treatment. Thus, by extension, these individuals are considered incompetent to provide voluntary informed consent to participation in a research study.

8 For more information concerning hospital admission criteria, see www5.myflorida.com/cf_web/myflorida2/healthhuman/substanceabusementalhealth/amhfacilities.html.

9 The term “residents” rather than “inpatients” is employed here as it is the preferred nomenclature at FSH.

10 Average after three univariate outliers were reined in by median-interquartile method. Original Median = 41.5 (75\textsuperscript{th} % = 47, and 25\textsuperscript{th} % = 34)

11 Average GAF after one univariate outlier was reined in by median-interquartile method. Original Median = 40 (75\textsuperscript{th} % = 45, and 25\textsuperscript{th} % = 30). In terms of the range of GAF scores observed in the sample, a GAF of 15 corresponds to “Some danger of hurting self or others…OR occasionally fails to maintain personal hygiene…OR gross impairment in communication” and a GAF of 70 is indicated by “Some mild symptoms…OR some difficulty in social, occupational, or school functioning…but generally functioning pretty well, has some meaningful interpersonal relationships” (APA, 1994, p.32).

12 The most recent or current episode of SZA (i.e., bipolar, depressed, or mixed) is not recorded in the FSH database; hence, these data were available only in the phase two sample.

13 It should be noted that the frequency of secondary diagnoses reported in this table indicate only single instances of the disorder and do not take into account their co-occurrence within the same individual. Moreover, these figures probably underestimate the actual comorbidity of certain disorders in the sample, because the schizophrenia diagnosis, which usually precipitated the admission, constitutes the primary focus of clinical attention. Time pressures and staff limitations may hinder a thorough evaluation of comorbid diagnoses beyond the primary disorder. Additionally, the frequency provided for “Any Substance Disorder” excludes six residents who were diagnosed with Nicotine Dependence. The actual frequency of this disorder is probably much higher given the relatively high prevalence of Nicotine dependence among similar inpatient samples (e.g., Green et al., 2003; Hughes et al., 1986; McCloughen, 2003).

14 It should be noted that among the 436 cases included in the first phase of the investigation, 54 PANSS ratings were completed exclusively on the basis of behavioral observations and information obtained from collateral sources, presumably because these
Residents were unable/unwilling to complete the semi-structured interview. Differences in PANSS scores as a function of administration were evaluated and are reported below.

One team of raters was assigned greater proportions of civilly-committed (12 vs. zero; $\chi^2(1) = 13.26, p<.001$) as well as female residents (21 vs. zero; $\chi^2(1) = 25.20, p<.001$), who were overall slightly older ($M = 41.82, SD = 9.1$ vs. $M=37.36, SD = 9.4, t(124) = 2.71, p<.01$) than those assigned to the other team for chart review. Importantly, however, the residents assigned to one team did not differ significantly from those assigned to the other team with regard to their overall functioning (GAF) or their scores on the five PANSS factors, all $p$'s >0.20. Moreover, teams were comparable in their assignment of the various CRS-AUD categories and ratings of problematic use among the charts assigned to them, $\chi^2(3) = 3.41, p=.33$ and $\chi^2(1) = 0.29, p=.59$, suggesting that assignment of cases by resident unit/ward (i.e., non-randomly) did not result in disproportionate numbers of problematic drinkers assigned to any one team.

As a naturalistic investigation, pharmacological treatment protocols were not controlled. Computation of dose-equivalents on medications on admission was hindered by missing data, poly-pharmacotherapy, and unknown compliance. Furthermore, medication changes were not uncommon following admission, suggesting that previous treatments may have been inadequate. Therefore, initially proposed efforts to transform medication doses into chlorpromazine/benzotropine equivalents were not undertaken. Instead, I examined the impact, if any, of medication type following admission -- presumably the more appropriate treatment protocol -- on the relationship of AUD with the observed PANSS factors by evaluating this variable for inclusion as a covariate.

Length of Stay at Consent was computed by taking the absolute difference between the date of hospital admission and the date informed consent procedures were completed.

It should be noted that of the 126 cases included in the final study sample, there were 60 cases in which the resident age at time of admission as recorded from the charts were discrepant from those recorded in the FSH-maintained database (46 were off by 1 yr, 3 by 2 yrs, and 11 by 3 yrs or more). Self-reported birth date was available in 125 cases, of which consistent age data were found in 66 chart ratings, and 86 FSH database entries. To address these discrepancies, an age variable was computed using data from these three sources (i.e., chart, database, and self-report). For 308 cases, only database entries for age were available. Age data were consistent and retained for two cases in which only self-report and the FSH database were available, and for two others in which only chart-recorded age and FSH database were available. Consistent age data were found across all three sources for 49 cases and accordingly, these age data were retained. For cases in which self-reported date of birth confirmed the age recorded in either the FSH database or the chart, this value was retained ($n=55$). For 18 cases in which the chart and database were consistent but discrepant from self-report, the former age value was retained. If all three sources differed ($n=8$), the average of the two most congruent age recordings was computed and this value was retained as indicative of the resident’s age (chart and database = 1, chart and self-report = 1, database and self-report = 5, equidistant across sources = 1).

The following number of errors in data entry were identified and corrected: Length of Stay = 3, Ethnicity = 1, Age = 1, Education = 2, Axis III diagnosis = 1.

The average difference in loadings between PCA with and without multivariate outliers included was -0.001 ($SD = 0.02$; Median = -0.003, Range: -0.06 to -0.001)
Item N7, which was dropped from the oblique solution, also showed inconsistent loadings in the orthogonal PCA and PAF solutions, lending further support to its deletion. PCA with Varimax (orthogonal) rotation yielded the following N7 loadings on each of the five extracted factors: 0.35, 0.41, 0.30, 0.39, and 0.20. PAF with orthogonal rotation yielded an identical factor pattern. The rotated percent variance for the components using all 30 PANSS Items were: NEG = 17.1%, POS = 12.8%, COG = 12.7%, EXC/HOS = 10.9% and ANX/DEP = 10.5%. When N7 was dropped, there were only minor differences in the variance explained: NEG = 17.3%, POS = 12.6%, COG = 13.0%, EXC/HOS = 10.6% and ANX/DEP = 10.8%.

In order to further evaluate the validity of the current PCA solution, additional factor analyses forcing three, four, and six factors were conducted. In the three-factor model, the first component contained all the original PANSS Negative Subscale items except N7 and corresponded closely with the first component extracted in the PCA presented above, but contained some COG items as well. The second component corresponded closely with the PANSS Positive Subscale, but also included various General Psychopathology Subscale items that made little theoretical sense to group together. The third component was highly consistent with ANX/DEP obtained in the initial PCA solution. This model was rejected, however, not only because it yielded no interpretable COG component, but also because it was found to be too conservative for the Cattell Scree Test and Kaiser Criterion (Eigen 3rd = 2.21). Examination of the residual matrix also suggested the presence of another component, as several moderate residuals (>0.05) and large residuals (>0.10) were evident.

Forcing four components, only the EXC/HOS and ANX/DEP components from the original five-factor solution emerged. Although separate NEG and POS components were evident, each contained several of the COG items (four on NEG and one on POS). There was no interpretable COG; rather, a seeming “disorganized behavior” factor emerged that contained all the EXC/HOS items along with COG items tapping attention, preoccupation, and insight/judgment skills. In addition to this model’s lack of a clear COG component, it was also considered too conservative for the Cattell Scree Test and Kaiser Criterion (Eigen 4th = 1.48). Examination of the residual matrix further suggested the presence of an additional component.

With six factors forced, the same first five components emerged as in the unrestricted EFA. The sixth component accounted for about 3% of the variance and was comprised of just two items (GP4 and GP15) which seemingly pulled an “agitated anxiety” element from the original ANX/DEP and COG dimensions. However, the rotated loadings of these items were inconsistent across PCA and PAF, suggesting the emergence of this component may be due, in part, to measurement error. Moreover, unless the constituent items were highly correlated only with each other (>0.70) and uncorrelated with other items – not the case in the present study (see Appendix C-1) -- components defined by two variables are generally considered to be unstable and uninterpretable (McDonald, 1985; Sayers, Curran, & Mueser, 1996; Tabachnik & Fidell, 2001). Thus, although this model yielded interpretable NEG, POS, and COG, it was rejected because it was less parsimonious than the five-factor model, failed the Scree and Kaiser Criterion tests (Eigen 6th = 0.92), and the sixth component was unstable.

Additionally, based on a comparison of their incremental fit indices derived from CFA procedures, the current five-factor model seemed to provide an improved fit to the observed data than the models suggested by the forced three \( \chi^2(402) = 2817.97, p<.001; \text{RMSEA} = 0.118 \) (90% Confidence Interval = [0.11, 0.12]); CFI = 0.92, NNFI = 0.92, AGFI = 0.65], four \( \chi^2(399) = 2498.92, p<.001, \text{RMSEA} = 0.11 \) (90% Confidence Interval = [0.11, 0.11]), CFI = 0.93, NNFI = 0.92, AGFI = 0.68], and six factor \( \chi^2(390) = 1996.90, p<.001; \text{RMSEA} = 0.097 \) (90% Confidence Interval = [0.093, 0.10]); CFI = 0.94, NNFI = 0.93, AGFI = 0.72] analyses.

Considered together, these results provide support for the current five-factor solution obtained via PCA with oblique (PROMAX) rotation.
Modest positive relationships were found between the number of days to PANSS administration \((Med = 7, M=8.31, SD=3.2)\) and scores on COG \((r=.10, p<.05)\), ANX/DEP \((r=.10, p<.05)\), and EXC \((r=.11, p<.05)\), suggesting that more severe symptoms within these dimensions tended to delay the completion of the PANSS by hospital personnel. Four univariate outliers on EXC/HOS and two on ANX/DEP were reined in by median-interquartile method.

With regard to method of administration, higher NEG scores were found among PANSS ratings completed on the basis of observation alone \((n=54, M=0.34, SD = 1.0)\) compared to those who were able to interview \((n=375, M=-0.06, SD = 1.0)\), \(t(427) = 2.82, p<.01\), suggesting that worse NEG symptoms may have interfered with PANSS interview procedures. There were seven missing data on method of administration.

Lastly, PANSS scores in the entire sample \((n=436)\) appeared to depend partly on the staff position of the individual completing the rating: \(F(4, 431) = 10.67, p<.001\) on NEG, \(10.30, p<.001\) on COG, \(8.02, p<.001\) on POS, \(22.05, p<.001\) on ANX/DEP, and \(4.00, p<.01\) on EXC/HOS. Follow-up simple effects comparisons indicated that PANSS rated by graduate-level psychology trainees \((n=24)\) yielded significantly higher factor scores than those completed by Ph.D. psychologists \((n=188)\), Master’s level psychology specialists \((n=142)\), post-doctoral psychology residents \((n=54)\), and pre-doctoral psychology interns \((n=28)\). However, in the absence of inter-rater reliability data on the PANSS, it cannot be determined which staff were more or less accurate in their ratings.

As can be seen from Appendix A-3, raters coded several additional alcohol-related variables, including Past AUD (present vs. absent, level of severity), Quantity-Frequency of Consumption, and Age of AUD Onset. An acceptable level of agreement was achieved in raters’ identification of a past history of AUD \((86.7\% \text{ agreement, Kappa } = 0.68)\) and the severity thereof \((93.75\% \text{ agreement; Kappa } = 0.76)\). Unfortunately, information concerning alcohol consumption was limited in most chart records. Consequently, these data were coded by the raters in too few cases to be reliably evaluated in the present study. Age of AUD Onset was recorded in 29 cases and an acceptable level of inter-rater reliability was achieved, Pearson \(r=0.98, p<.001\). Though not explored in the current investigation, collection of similar alcohol-related data may be useful in future studies of the AUD-SZC relationship.

There was one missing data point on date of rating.

There were three missing data points in time to complete ratings.

This outlying case also emerged in supplemental multivariate outlier analyses, in which regressions were carried out separately for each of the CRS categories. This male forensic resident was the only one with a primary diagnosis of Schizophrenia-Catatonic Type in the study, thus exclusion of this resident from further analyses appeared warranted. His comorbid diagnoses included Alcohol and Cannabis Abuse.

Demographic statistics follow correction for univariate outliers identified and reined in by the median-interquartile method: Age = 3 outliers, original median = 41.5 \((75^{th} \% = 47, 25^{th} \% = 34)\); Education = 15 outliers, original median = 11.5 \((75^{th} \% = 12, 25^{th} \% = 10)\).

Statistics presented in this treatment history section follow correction for univariate outliers identified and reined in by the median-interquartile method: Age at first admission, zero outliers; Number of Previous Admissions, eight outliers, original median = 5.38 \((75^{th} \% = 8.25,\)
Information concerning past treatment was noted to be somewhat unreliable by chart raters, with most estimated on the basis of limited data in chart records. However, inter-rater agreement for Number of Previous Hospitalizations was an acceptable 0.81 as was Duration of Previous Hospitalizations (ICC = 0.92), and thus data from each rater were averaged to optimize stability. Complete ratings of premorbid functioning (see Code Sheet, Appendix A-3) were available in just 72 cases, and agreement was poor Kappa = 0.35.

As this was a naturalistic study, all residents were treated according to standard clinical practice and thus taking medication at the time of the AUDIT interview. Additionally, 102 residents were medicated at the time of admission to FSH, among whom 16 required some medication injections. Of these, a substantial majority (87.2%) subsequently underwent medication changes, suggesting, perhaps, the inadequacy of their previous treatment regimen. Specifically, dosages of medication were increased in 50 cases and decreased in 10 others; the remainder stayed on the same dose of their original medication but had another added. There were no significant differences between CRS-P and CRS-NP groups with regard to medication status at the time of admission, nor with regard to subsequent medication changes.

Of the 23 residents admitted without medication, each was subsequently prescribed either one or some combination of the following psychotropic treatments: typical antipsychotic (n=10), atypical antipsychotics (n=15), side effect medications (n=7), mood stabilizers (n=9), antidepressants (n=2) and/or anxiolytics (n=4). Importantly, a substantial majority (97.6%) of the current sample were ultimately prescribed either one or a combination of typical (n=55) and atypical antipsychotics (n=100), supporting their psychotic disorder diagnosis. Other psychotropic interventions included either one or a combination of: side effect medications (47.2%), mood stabilizers (46.4%), antidepressants (12.8%), and/or anxiolytics (28.0%). In addition, 30 residents were prescribed one or more medications for various medical conditions. A statistically smaller proportion of the CRS-NP group (36.7%) were prescribed side effect medications as compared to those in the CRS-P group (56.6%), χ²(1) = 4.50, p<.05.

For crime classification, raters agreed in a significant, albeit not perfect, proportion of the cases (81.1% with 3 missing data). For cases with discrepant ratings, the offense was classified as both violent and non-violent.

It should be noted that Primary Axis I diagnoses were modified in 19 cases from the time of their admission to chart review. On admission, the diagnostic distribution was as follows: 41.3% SZA (N=52, n= 14 female), 38.8% Paranoid Type (N=49, n=6 female), 14.3% Undifferentiated Type (N=18, n=1 female), 4.0% Disorganized Type (N=5), and <1% Residual Type (N=2) and Catatonic Type (N=1). Eleven residents originally identified as Paranoid Type and two others with Undifferentiated Type Schizophrenia were later diagnosed as having SZA Disorder. Two other cases were changed from SZA to Paranoid Type and another from Undifferentiated Type to Residual Type. Finally, three residents later received Undifferentiated Type diagnoses, two of whom were initially diagnosed as SZA and the other as Paranoid Type.

In addition, 94 residents (74.6%) were diagnosed with one or more medical conditions on Axis III. The most common of these were Hypertension (n=26), Seizure Disorder (n=16), High Cholesterol (n=13), History of Head Injury (n=10), Diabetes (n=9), Hepatitis (n=9), and extrapyramidal side-effects/Tardive Dyskinesia (n=7). Some other documented conditions included anemia, cancer, gastrointestinal problems, heart-related illnesses, HIV/AIDS, obesity, respiratory problems (asthma, bronchitis, COPD), and various surgical procedures.
Figure excludes AUD diagnosed concurrently with polysubstance disorder with alcohol identified as a substance of abuse (n=2).

In addition to the sample characteristics which varied as a function of CRS group membership, several demographic and clinical variables were found to differ systematically with the PANSS factors (see Appendix C-3). In selection of covariates, including those specified a priori, I attended to their common shared variance. Examination of tolerance statistics suggested the statistical independence of the six covariates included in final model: EXC/HOS = 0.70, GAF = 0.80, ANX/DEP = 0.82, Gender = 0.90, drug disorder = 0.91, and age = 0.94.

Other potential covariates were identified via analysis of bivariate relationships with key variables (i.e., NEG, POS, COG, and CRS-AUD) and significant stepwise entry into the planned hierarchical equation. These included: (a) total number of comorbid diagnoses, and (b) the number of previous hospitalizations.

I opted not to adjust statistically for the total number of comorbid Axis I and Axis II diagnoses, because this variable can be expected to relate to CRS-P versus CRS-NP structurally, rather than incidentally. However, the impact of specific comorbid disorders that may theoretically relate to AUD or COG (e.g., Antisocial Personality Disorder, History of Traumatic Brain Injury, Mental Retardation, etc.) on the present study findings was tested. There were too few anxiety disorders represented in the sample for such analyses, and evaluating the impact of the presence/absence of mood disorders (e.g., bipolar vs. depressive) was essentially similar to analyses of the SZA subtypes, as discussed below. Apart from a comorbid diagnosis of “other drug disorder,” however, no other diagnostic classification entered as a significant covariate in the present analyses. My a priori decision to include overall severity (i.e., GAF) and the additional factor scores (EXC/HOS and ANX/DEP) in the model aimed to account for any “noise” introduced by the presence of these various comorbid conditions. The decision to include the presence/absence of a drug disorder stemmed not only from its significant entry as a covariate in the analyses, but also because it had the potential to speak to the specificity of results to AUD, as opposed to SUDs more generally.

It is plausible that rates of AUD comorbidity and the underlying mechanisms thereof may vary along several lines, including perhaps the subtype of schizophrenia and/or SZA disorder (i.e., Bipolar – Manic, Bipolar – Depressed, Depressed, Mixed), a diagnostic determination that is determined by the resident’s most recent clinical presentation of psychotic and mood symptoms. Although differences in rates of CRS problem use between residents with primary diagnoses of schizophrenia and SZA (39.1% vs. 54.1%, respectively) approached significance, χ²(1) =2.84, p=.092, this distinction did not enter as a significant covariate in the planned analyses, nor did it significantly impact the present pattern of results. Too few residents were assigned primary diagnoses of SZA - Depressed Type or types of schizophrenia other than Paranoid Type to allow for a more fine-grained analysis of these DSM-IV diagnostic categories. Although the total number of previous hospitalizations varied with CRS group membership (i.e., CRS-P > CRS-NP) this variable was not statistically controlled because it was considered more likely a consequence, rather than predictor, of AUD. As can be seen from Table 17, more previous hospitalizations were also associated with higher scores on POS and EXC/HOS in this sample. Again, however, this shared variance with POS and EXC/HOS may be more of a consequence, rather than predictor, of these symptoms which by their nature may tend to draw increased attention from legal or community authorities. In any case, a similar pattern of results was obtained regardless of whether this variable was included in the model.

As with the Phase One sample, factor scores varied systematically with the position of the FSH staff completing the PANSS in the subset of residents evaluated for AUD. Specifically, psychology trainees tended to rate NEG [F(4, 120) = 4.58, p<.01], COG [F(4, 120) = 8.63,
p<.001], and ANX/DEP \[F(4, 120) = 10.20, p<.001\], but not POS \[F(4, 120) = 2.06, n.s.\] or EXC/HOS \[F(4, 120) = 2.03, n.s.\] as more severe than staff holding other positions at FSH. However, group sizes for this analysis were quite disparate as only six psychology trainees were compared with 62 Ph.D. psychologists, 39 Masters level psychology specialists, 10 post-doctoral psychology residents, and eight pre-doctoral psychology interns. Nonetheless, a similar pattern of results was obtained when the six cases of PANSS data originally completed by psychology trainees were dropped, although the \(p\)-values for the effects of interest were slightly elevated due to loss in power \(p\)-value for POS x COG interaction was 0.11. As noted previously, there were insufficient data to determine whether certain of the staff were more or less accurate in their ratings and hence I cannot support simply dropping these cases.

A similar pattern of results was obtained when the planned regression was repeated using a restricted sample that excluded persons with documented Mental Retardation or Borderline IQ. A significant POS X COG interaction was observed when entered in step four of the equation, \(\Delta \chi^2(1) = 7.65, p<.01\). Follow-up analyses, applying a median-split of POS scores, indicate that there was no association between COG and AUD for residents with fewer POS symptoms \(\Delta \chi^2(1) = 0.80, n.s.\), but each unit increase in the severity of COG symptoms was associated with a 3.2 times decreased likelihood of problematic use for persons with higher POS symptoms, Wald = 3.93, \(p<.05\), \(\Delta \chi^2(1) = 5.19, p<.05\). Likewise, although there was no difference in any PANSS factor score as a function of a cognitive-related comorbid diagnosis (e.g., history of brain injury, dementia, etc.), I re-analyzed the data excluding these participants. A similar pattern of results was obtained when persons with cognitive related diagnoses were excluded \((n=11)\) in that a significant POS X COG interaction was observed, \(\Delta \chi^2(1) = 4.69, p<.05\). Follow-up analyses, applying a median-split of POS scores, indicate that there was no association between COG and AUD for those non-cognitively disordered residents with fewer POS symptoms \(\Delta \chi^2(1) = 0.01, n.s.\), but each unit increase in the severity of schizophrenia-related cognitive symptoms (COG) was associated with a 3.5 times decreased likelihood of problematic use for persons with higher POS symptoms, Wald = 4.46, \(p<.05\), \(\Delta \chi^2(1) = 6.28, p<.05\). Considered together, these results suggest that the apparently protective effects of COG cannot be attributable to general deficits in intellectual functioning or the impact of some identifiable organic or degenerative condition, but probably depend on the degree of cognitive impairment associated with SZC symptomatology.

Based on previous factor analyses of the PANSS with similar samples, I expected COG to be comprised of P2 - Conceptual Disorganization, N5 – Difficulty in Abstract Thinking, GP10 - Disorientation, and GP11 – Poor Attention. Indeed, all four were represented on COG with three of them showing the highest loadings. Differences in item retention criteria, which are to some degree arbitrarily defined by the researchers, might explain some of the discrepancies in the items comprising COG across studies (see Appendix D-1). As a case in point, G5 and G13 were found to load on NEG in the Kay & Sevy (1990) study, but subsequent re-analyses and reinterpretation of these data were taken to support their attachment to COG (Bell et al., 1994). Though the results of this latter study may serve to increase confidence in the present study’s similar attachment of these two items, the general consistency of current findings with previous research should not be interpreted as a firm resolution of the aforementioned lack of consensus in the literature on precisely which items should be identified with COG.

Indeed, other items located on COG in this study are less often similarly attached in other research, including those tapping problems in the thoughtful control of movement (G5, G13), insight and judgment, and preoccupation with internal thoughts. For example, G13 was found to load relatively highly on COG in the current sample, but this item has shown similar
attachment to COG in just two other analyses (Bell et al., 1994b re-analysis of Kay & Sevy, 1990; Dollfus et al., 1991). More often it either has not been retained in the final solution or alternatively attached to NEG (Bell et al., 1994a; Kay & Sevy, 1990; Peralta & Cuesta, 1994). The remaining items (i.e., G5, G12, and G15) were attached to COG with slightly lower loadings, and thus it is perhaps unsurprising that these items have demonstrated inconsistent or non-existent factor attachments in other analyses of the PANSS. Despite mixed evidence for their retention on COG in the literature, these items were found not only to load more highly on COG than any of the other four components in this study, but also they were of theoretical relevance to the construct of cognitive deficit symptoms of schizophrenia and partly retained for that reason. For example, G12 maintained the lowest loading of the eight items located on COG meeting my minimal communality requirement. However, retention of this item was considered appropriate because it was designed to measure a lack of acknowledgement or awareness of psychiatric symptoms and their implications, making it highly consistent with my initial conceptualization of the type of cognitive deficits thought to impact drinking decisions.

38 In the present sample, SZCs differed from those with SZA not in the symptom dimensions tapping emotional/behavioral disturbance (i.e., ANX/DEP, EXC/HOS) as one might expect, but rather only in NEG symptomatology. Some have argued that because these “additional” factors are not useful for identifying SZC subgroups, their emergence probably reflects the broader assessment of instruments like the PANSS, rather than the existence of actual added SZC symptom dimensions that explain the heterogeneity of this complex disorder (Hwu et al., 2002). It could also be effectively argued that the emergence of these factors underscores the relevance of symptom dimensions, rather than diagnoses, in describing the range of presentations common to schizophrenia spectrum disorders.

39 I was able to locate 29 of the 30 items on the NEG, COG, POS, ANX/DEP, and EXC/HOS; however, N7 could not be attached to any of these five components, a finding common to all but three of the 11 factor analytic studies evaluating similar samples (see Appendix D-1). Moreover, among those studies that retained N7 in their factor solutions, there has been no consistent pattern of affiliation on any one factor: EXC/HOS in a combined inpatient and outpatient sample of SZCs (Dollfus et al., 1991), COG in a sample of inpatients with schizophrenia (Bell et al., 1994a re-analysis of Kay & Sevy, 1990) and POS in another sample of SZC inpatients (Lindenmeyer et al., 1995). This item was designed to measure spontaneity and flexibility of thought processes, but questions could be raised about the definition criteria and validity of the construct tapped by this item on the basis of these conflicting data. In the present study, there was no evidence of multicolinearity that would suggest variance in N7 was completely accounted for by one or some combination of PANSS items. Thus, it remains plausible that this item is tapping a separate construct altogether, one for which the other PANSS items provide insufficient coverage.

40 Evidence pointing to an earlier onset of SUD following a period of good premorbid functioning has been taken to implicate substance use in the development of schizophrenia (Allen & Frances, 1986). Although there is general agreement that alcohol does not directly cause schizophrenia, some have speculated that drinking indirectly contributes to schizophrenia by increasing the likelihood of its expression in those already vulnerable to its development (Dixon et al., 1991). However, such notions are generally thought to be inapplicable to the special case of AUD and schizophrenia (Mueser et al., 1998), though they may be relevant to other psychiatric conditions or to associations with other drugs (e.g., cannabis, cocaine).
In accordance with FSH Research guidelines, ineligible participants included persons who were incompetent to consent to treatment and those for whom participation was deemed clinically contraindicated. Competence to consent to treatment is a legal determination involving the capacity to make treatment decisions in one's own best interest. Research comparing persons competent and not competent to consent to treatment suggests that such classifications are closely linked to severity of cognitive deficits (e.g. Holzer et al., 1997; Rosenfeld & Turkheimer, 1995). In the present sample, incompetent residents were more likely to be civilly committed, exhibit more severe symptoms overall (GAF), and worse NEG and COG relative to legally competent individuals. Thus it is plausible that the few individuals with the worst NEG and COG were systematically excluded from the present study. Similarly, persons excluded on the basis of whether participation was considered clinically inappropriate showed worse COG and EXC/HOS, again suggesting that the present sample may have been representative of less severe symptomatology in these domains.

It is perhaps noteworthy that although P2-Conceptual Disorganization is closely aligned with the COG dimension in some research (See Appendix D-1), it seemed to load less distinctly in the present study, maintaining a moderate attachment to POS. Similar cross-affiliation of this item has been observed when PANSS data are collected from SZCs in an unmedicated, acute phase of their illness (Lancon et al., 2000).

In the present study, variance attributable to Antisocial Personality Disorder was minimized when FSH_DUD was entered into the model.
### Instructions:

“Now I am going to ask you some questions about your use of alcoholic beverages in the year prior to your admission to Florida State Hospital. By alcoholic beverages, I mean your use of a glass of wine, a can of beer, or a shot of liquor. Please try to be as honest and accurate as you can be. Let’s start.”

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How often did you have a drink containing alcohol during the year prior to your admission?</td>
<td>(0) Never [Skip to Qs 9-10] (1) Monthly or less (2) 2 to 4 times a month (3) 2 to 3 times a week (4) 4 or more times a week</td>
</tr>
<tr>
<td>2. How many drinks containing alcohol did you have on a typical day when you were drinking during the year prior to your admission?</td>
<td>(0) 1 or 2 (1) 3 or 4 (2) 5 or 6 (3) 7, 8, or 9 (4) 10 or more</td>
</tr>
<tr>
<td>3. How often did you have six or more drinks on one occasion in the year prior to your admission?</td>
<td>(0) Never (1) Less than monthly (2) Monthly (3) Weekly (4) Daily or almost daily</td>
</tr>
<tr>
<td>4. How often during the year prior to your admission did you find that you were not able to stop drinking once you had started?</td>
<td>(0) Never (1) Less than monthly (2) Monthly (3) Weekly (4) Daily or almost daily</td>
</tr>
<tr>
<td>5. How often during the year prior to your admission did you need a first drink in the morning to get yourself going after a heavy drinking session?</td>
<td>(0) Never (1) Less than monthly (2) Monthly (3) Weekly (4) Daily or almost daily</td>
</tr>
<tr>
<td>6. How often during the year prior to your admission did you have a feeling of guilt or remorse after drinking?</td>
<td>(0) 1 or 2 (1) 3 or 4 (2) 5 or 6 (3) 7, 8, or 9 (4) 10 or more</td>
</tr>
<tr>
<td>7. How often during the year prior to your admission were you unable to remember what had happened the night before because you had been drinking?</td>
<td>(0) Never (1) Less than monthly (2) Monthly (3) Weekly (4) Daily or almost daily</td>
</tr>
<tr>
<td>8. How often during the year prior to your admission did you find that you were not able to stop drinking once you had started?</td>
<td>(0) Never (1) Less than monthly (2) Monthly (3) Weekly (4) Daily or almost daily</td>
</tr>
<tr>
<td>9. Have you or someone else ever been injured as a result of your drinking?</td>
<td>(0) No (1) Yes, but not in year prior to admission (2) Yes, during the year prior to admission</td>
</tr>
</tbody>
</table>

Skip to Qs 9-10 if total score for 2 & 3 = 0
AUDIT – Continued

<table>
<thead>
<tr>
<th>5.</th>
<th>How often during the year prior to your admission did you fail to do what was normally expected from you because of drinking?</th>
<th>10</th>
<th>Has a relative or friend or doctor or another health worker been concerned about your drinking or suggested you cut down?</th>
</tr>
</thead>
<tbody>
<tr>
<td>(0)</td>
<td>Never</td>
<td>(0)</td>
<td>No</td>
</tr>
<tr>
<td>(1)</td>
<td>Less than monthly</td>
<td>(1)</td>
<td>Yes, but not in year prior to admission</td>
</tr>
<tr>
<td>(2)</td>
<td>Monthly</td>
<td>(2)</td>
<td>Yes, during the year prior to admission</td>
</tr>
<tr>
<td>(3)</td>
<td>Weekly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4)</td>
<td>Daily or almost daily</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hazardous Alcohol Use (1 – 3)  
Dependence Symptoms (4 – 6)  
Harmful Alcohol Use (7 – 10)  
Total
Clinicor Rating Scale – Alcohol Use Disorder

Participant ID#: ___________________ Date of Rating: ___________________

Instructions: Rate the resident’s use of alcohol over the twelve months prior to institutionalization. You should weigh evidence from self-report, laboratory tests (e.g., urine screens), chart diagnoses, and collateral reports (referral information, legal, family reports) as noted in the chart progress notes, treatment plan, and discharge summary. Take care to ensure that you are weighing evidence from the worst period in the 12-month interval prior to admission to FSH.

____ 1. ABSTINENCE – There is no evidence that the resident has used alcohol during this period.

____ 2. USE WITHOUT IMPAIRMENT – resident has used alcohol during this time interval, but there is no evidence of persistent or recurrent social, occupational, psychological, or physical problems related to use and no evidence of recurrent dangerous use.

____ 3. ABUSE – resident has used alcohol during this time interval and there is evidence of social, occupational, psychological (other than just cognitive impairment), or physical problems related to use, or evidence of dangerous use (see Table 4). Problems have persisted for at least one month during this interval. Do not rate if cognitive impairment is the only consequence identified. An arrest for drunk & disorderly conduct would have at least this rating.

____ 4. DEPENDENCE – there is evidence for at least three of the following:
   1. Tolerance (high consumption with little effect on behavior)
   2. Withdrawal (blackouts, nausea, hangovers, etc.) OR alcohol taken to relieve or avoid withdrawal
   3. Drink in larger amounts or for longer intervals than intended
   4. Repeated unsuccessful efforts to quit or control use of alcohol (e.g., patient has been in treatment several times for substance use but apparently drank in year prior to admission)
   5. Spends a lot of time trying to obtain or use substance,
   6. Frequent intoxication or withdrawal interferes with activities, or important activities given up because of alcohol use,
   7. Continued use despite recurrent abuse-level problems (see Table 4). For example, drinking binges and preoccupation with drinking have caused resident to drop out of job training and non-drinking social activities.

____ 5. SEVERE DEPENDENCE – alcohol-related problems are so severe that they make non-institutional living difficult. For example, constant drinking leads to disruptive behavior and inability to pay rent so that client is frequently reported to police and seeking hospitalization. Rate if alcohol is the primary reason for FSH readmission.

If 1 or 2, rate past history of AUD:   Yes   No (if yes, level _____)

From Toolkit for Evaluating Substance Abuse in Persons with Severe Mental Illness by Mueser et al., 1995b. Modified from the 9/28/00 version by the NH-Dartmouth Psychiatric Research Center.
APPENDIX A-3

DATA RECORD SHEET

Rater ID: 
PARTICIPANT ID: _____ Date of Chart Rating: _____ Time to Complete (in mins): ______

| DEMOGRAPHICS/ADMISSION CRITERIA |
|---------------------------------
| Commitment Type (circle one): Civil - Competent  Forensic – Competent  Both |
| Specify Type of Crime (if forensic): Non-Violent  Violent  Both  Education (in yrs): |
| Age at time of admission: |

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<td>Axis V:_______</td>
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<th>TREATMENT HISTORY</th>
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<td>Age at first psychiatric hospitalization:</td>
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<td>Level of Pre-morbid Functioning (if noted, circle one): Poor  Fair  Good/Normal  Very Good</td>
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<tr>
<td>Number of Previous Hospitalizations:  (No. Psychiatric, No. Substance)</td>
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<td>Total Duration of Previous Hospitalizations (in months):</td>
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<td>NOTE: Do NOT include the current (or most recent, if discharged) admission, in this figure – see example</td>
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<tr>
<td>Length of Current Hospitalization (in months) (See Date Conversion Table)</td>
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<td>Medicated at time of admission (Circle One): Yes  No</td>
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<td>(if yes, specify medications and doses):</td>
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<tr>
<td>Current Medications: specify medications and doses:</td>
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<tr>
<td>Intelligence Tests Available? (circle one) Yes  No (Specify Test:________)</td>
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<td>[Verbal IQ:_______ Performance IQ:_______ Full Scale IQ:_______]</td>
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<td>Summarize if WAIS-III</td>
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<td>Average QUANTITY of standard drinks per episode in year prior to admit:</td>
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<td>Average FREQUENCY of consumption (Code: 0, 1, 2, 3, 4):</td>
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<tr>
<td>Approximate age of AUD onset</td>
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</table>

CLINICIAN RATING SCALE (1 - 5): ____ (see CRS for instructions and scale) |
| Degree of confidence in CRS Overall Rating (1- no confidence to 10- high confidence) |
| If CRS = 1 or 2, rate past history of AUD: Yes  No  (if yes, CRS level _____) |
| If CRS = 3 (Abuse), record applicable problem code(s): |
| If CRS = 4 (Dependence), record applicable symptoms (1-7): |
| Record any other comments you may have regarding alcohol or other substance use (use reverse if needed) |

Reardon Dissertation Study - FSH
INFORMED CONSENT FORM

Hello ____________________________, my name is ____________________________.

I am a student from the Florida State University. I would like your help in a research study that I’m doing. We are trying to find out why some people getting treatment in hospitals like this one use alcohol in the community and others do not.

To do this, we will look at your hospital chart. We will also ask about your experiences with alcohol. This will probably take about 20 minutes. I’m going to be asking some of the other residents here at the hospital to review their charts and to help me answer the same questions.

Some of these questions may seem a little personal or private and might be hard to answer. If you feel uncomfortable and don’t want to answer any more questions, you just let me know and we can stop at any time. I want you to feel comfortable talking with me, so tell me if you don’t like it. Your participation is voluntary, meaning that it is up to you whether you do the study or not. You will not get into any trouble if you say you don’t want to do it, or if you want to stop once we start.

You will not be given any compensation (e.g., credits for the canteen) for taking part in this study. You will also not benefit directly from taking part in the study. If you do decide to take part in this study, it may make it easier for the hospital to help residents with their problems, so we’d really like your help.

We will only mark your answer sheet with numbers. We will not use your name in the study. Your answers will not be put anywhere in your chart. Information about your participation in this study will be private and protected to the full extent of the law.

If you have any questions about the study, you can contact Maureen Reardon or Dr. Lang at The Florida State University by calling (850) 644-3034 or by writing to the address below (noted *).

If you have any questions or concerns about your rights about taking part in this study or if you need to report a research related injury, you can call the Florida State University Institutional Review Board for Human Subjects at (850) 644-9633 or the Review Council for Human Subjects at (850) 245-4585 or toll free at 1-866-433-2775.

Now, please tell me in your own words what I’ve just told you about this study. Name two things you will be asked to do during the study. What would you do if you didn’t want to take part in the study? What would you do if you felt upset or uncomfortable during the study? Do you have any questions? (Interviewer: please ensure resident understands and clarify any misconceptions. Complete the RCHS Evaluation to Sign a Consent Form).

Your signature below means that you have been allowed to ask questions about the study and that you got answers to them. You may keep a copy of this form.

_____________________________ Date

Signature of Legal Guardian

_____________________________ Date of Birth

Signature of Resident

Printed Name

The Florida State University
Department of Psychology
Tallahassee, FL 32308

RCHS IRB

APPROVED

AUG 20 2003

EXPIRES

RCHS IRB

99
Review Council for Human Subjects
Evaluation to Sign a Consent Form for Research

Subject Identifier: _____________________  Date of Evaluation:_______________

Date of Birth:_________________________

1. Is the subject alert and able to communicate with the examiner?   ____ Yes   ____ No

2. Ask the subject to tell you the purpose of the study.

_______________________________________________________________________

_______________________________________________________________________

3. Ask the subject to name at least two things that he/she will be expected to do during the study.

_______________________________________________________________________

_______________________________________________________________________

4. Ask the subject to explain what he/she would do if he/she no longer wanted to participate in the study.

_______________________________________________________________________

_______________________________________________________________________

5. Ask the subject to explain what he/she would do if he/she experienced distress or discomfort during the study.

_______________________________________________________________________

_______________________________________________________________________

Examiner’s signature that this subject was alert, able to communicate, and gave acceptable answers to the questions asked.

_____________________________________________
APPENDIX B-3

Award Certificate

CERTIFICATE OF APPRECIATION

THIS CERTIFICATE IS PRESENTED TO:

______________________________

FOR TAKING PART IN A FLORIDA STATE UNIVERSITY STUDY

Signature of Person Doing Study   Date

Florida State University
Table 15

*Intercorrelations and Descriptive Statistics for the PANSS Items*

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** Correlation significant at the 0.01 level, one-tailed; *Correlation significant at the 0.05 level, one-tailed.
Table 15 - continued

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|   | SD |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
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|   | 1.43 | 1.41 | 1.13 | 1.37 | 1.40 | 1.24 | 1.10 | 1.09 | 1.07 | 1.07 | 1.47 | 1.47 | 1.26 | 1.13 | 1.06 | 0.78 |   |

** Correlation significant at the 0.01 level, one-tailed; *Correlation significant at the 0.05 level, one-tailed.
Table 15 - continued

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** Correlation significant at the 0.01 level, one-tailed; *Correlation significant at the 0.05 level, one-tailed.
## APPENDIX C-2

Table 16

*Mean Comparisons of PANSS Factor Scores Between CRS Groups.*

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<th>CRS-P</th>
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<td>NEG</td>
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<td>COG(^a)</td>
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<td>EXC/HOS</td>
<td>-.012 (1.0)</td>
<td>-0.03 (1.0)</td>
<td>-.049</td>
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\(^a\) Levine’s Test for the Equality of Variances revealed significant differences in the variance of COG scores within the CRS-P and CRS-NP groups, \(F = 6.48, p<.05\). Accordingly, the \(t\)-test corrected for un-equal variances is reported.
### APPENDIX C-3

Table 17

Demographic and Clinical Correlates of the PANSS Factors

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<th>COG</th>
<th>POS</th>
<th>ANX/DEP&lt;sup&gt;c&lt;/sup&gt;</th>
<th>EXC/HOS&lt;sup&gt;c&lt;/sup&gt;</th>
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<td>Age (on admission)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-.002&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>0.05(1.1)</td>
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p<.05 level, ** p<.01, ***p<.001; <sup>a</sup>Pearson r; <sup>b</sup>Mean (SD); <sup>c</sup>planned covariates
## APPENDIX D-1

### Table 18

*Synopsis of Selected Factor Analytic Research on the PANSS*

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\(^a\) re-analysis of data from Kay & Sevy (1990) study in COG items were originally identified as P2, N5, GP11
APPENDIX E-1

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SENT VIA FAX

April 30, 2004

Florida State University
Department of Psychology
Tallahassee, FL 32306

Attention: Maureen Lyons Reardon

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17. This Agreement and Schedules attached hereto constitutes the entire agreement between the Licensee and MHS. This Agreement may be amended or modified only by express written consent by both parties under this Agreement.

18. Faxed and photostatic copies of this Agreement shall be considered valid legal documents.

MULTI-HEALTH SYSTEMS INC.:

[Signature]
Authorized Signing Representative

May 11, 2004
Date

cc: Rina Chadda, Legal Counsel

The Licensee hereby agrees to the above terms and conditions on this 30th day of 4, 2004.

MAUREEN LYONS REARDON:

By: [Signature]
Authorized Signing Representative

Name: Maureen Lyons Reardon
(Print)

Title: ___________________________
(Print)
SCHEDULE “A”

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MAUREEN LYONS REARDON:

Date: 4/30/04

By: [Signature]

Print Name: Maureen Lyons Reardon

Title: [Title]

Address: 202 Folsom Ln

[City, State, Zip]

Page 5 of 5
Text Description.

The PANSS by Stanley R. Kay, Ph.D., Lewis A. Opler, M.D., Ph.D., and Abraham Fiszbein, Ph.D., is a 30-item symptom rating scale that was designed to measure core positive and negative symptoms of schizophrenia and related psychopathology (e.g., anxiety, depression). Symptoms are rated along a seven-point continuum of severity on the basis of a semi-structured interview with the patient as well as observations of the patient's behavior during the past seven days as reported by hospital staff, family, or other collateral informants. The PANSS appears to be a psychometrically sound instrument in the measure of the SZCs' overall clinical profile (Kay, Opler, & Lindenmayer, 1988; Kay, Opler, & Lindenmayer, 1989; www.mhs.com). See Table 1.

<p>| Table 1 |
| Positive and Negative Syndrome Scale by Stanley R. Kay, Ph.D., Lewis A. Opler, M.D., Ph.D., and Abraham Fiszbein, M.D. FSH psychology personnel rate the following items on the basis of structured interview (SCI-PANSS; Kay, 1991), behavioral observations, and reports from staff, family, or referral sources. |</p>
<table>
<thead>
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<th>Positive Scale (POS)</th>
<th>Negative Scale (NEG)</th>
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<tr>
<td>1 Absent</td>
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<tr>
<td>2 Minimal</td>
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<td>3 Mild</td>
<td>3 Mild</td>
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<tr>
<td>4 Moderate</td>
<td>4 Moderate</td>
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<tr>
<td>5 Moderate- Severe</td>
<td>5 Severe</td>
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<tr>
<td>6 Severe</td>
<td>6 Severe</td>
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<tr>
<td>7 Extreme</td>
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</table>

- **P2 Conceptual disorganization**: Impairments in thought processes as evidenced by an inability for goal-directed communication, e.g., tangential or circumstantial speech, loose associations, illogical speech, or thought blocking.
- **N5 Difficulty in abstract thinking**: Difficulty in classifying objects or concepts, generalizations and similarities. Problems are resolved using only concrete or egocentric thought processes.
- **GP10 Disorientation**: Unawareness or confusion about whom one is (name, birth date), where they are, or passage of time (may be due to withdrawal).
- **GP11 Poor attention**: Attention deficits evident through easy distractibility, or difficulty sustaining attention or re-adjusting attention to new stimuli.
- **GP13 Disturbance of volition**: Impairment in the voluntary initiation, maintenance, and control of one's thoughts and behaviors.
- **GP15 Preoccupation**: Fixed focus on thoughts, feelings, or other internal experiences that interfere with reality-testing and behavior.


PANSS by Stanley R. Kay, Ph.D., Lewis A. Opler, M.D., Ph.D., and Abraham Fiszbein, M.D.
APPENDIX E-2

Florida State University Human Subjects Approval

The forms that you submitted to this office in regard to the use of human subjects in the proposal referenced above have been reviewed by the Human Subjects Committee at its meeting on September 11, 2002. Your project was approved by the Committee.

The Human Subjects Committee has not evaluated your proposal for scientific merit, except to weigh the risk to the human participants and the aspects of the proposal related to potential risk and benefit. This approval does not replace any departmental or other approvals which may be required.

If the project has not been completed by September 10, 2003, you must request renewed approval for continuation of the project.

You are advised that any change in protocol in this project must be approved by resubmission of the project to the Committee for approval. Also, the principal investigator must promptly report, in writing, any unexpected problems causing risks to research subjects or others.

By copy of this memorandum, the chairman of your department and/or your major professor is reminded that he/she is responsible for being informed concerning research projects involving human subjects in the department, and should review protocols of such investigations as often as needed to insure that the project is being conducted in compliance with our institution and with DHHS regulations.

This institution has an Assurance on file with the Office for Protection from Research Risks. The Assurance Number is IRB00000446.

APPLICATION NO. 02.298
Cc: A. Lang (Psychology)
September 12, 2002

Maureen Lyons Reardon, M.S.
Florida State University
Department of Psychology
Tallahassee, FL 32306

RE: RCHS 1153 “Prediction of Comorbid Alcohol Use Disorders Using Factors Derived from the Positive and Negative Syndrome Scale in an Inpatient Hospital Population”

Dear Ms. Reardon:

I am happy to inform you that the Florida Department of Health, Review Council for Human Subjects (M1350-01) has approved the changes listed in the letter dated September 3, 2002.

Council members agreed that this is a minimal risk study, therefore, this will need to be reviewed annually. The first progress report will be due before August 20, 2003. Please feel free to call the RCHS administrative office at (850) 245-4585, should you have any questions.

Sincerely,

[Signature]

Paul Arons, M.D.
Chair, Review Council for Human Subjects
APPENDIX E-4

Florida State Hospital Research Approval

Ms. Maureen Lyon Reardon, M.S., is a graduate student in the Department of Psychology at Florida State University. She has been employed as a contract graduate student at Florida State Hospital. For her doctoral research named above, she has requested to collect some data here. If approved at this site, the data collection here could be expected to be completed before July 2003.

A copy of the proposal and material for the Review Council for Human Subjects, Application for Study Review, are attached. The proposed research would study the relationship between schizophrenia-related cognitive deficits and comorbidity alcohol use disorders. The primary measures would be the Positive and Negative Syndrome Scale and the Alcohol Use Disorder Rating.

The proposed research has been reviewed by the Florida State Hospital Research Committee as appropriate with regard to research design and resident safety. At the suggestion of the Research Committee, Ms. Reardon made minor changes in the proposal.

The proposal was reviewed by members of the Florida Local Advocacy Council at the June meeting. Questions regarding the informed consent form and permission to review records were raised. The informed consent form has been amended; the record review and interview procedures have received approval at a subsequent hospital meeting. It is recommended that hospital approval be granted for the collection of data as outlined in the proposal, and that it be forwarded in the review process.

Attachment
cc(memo): Dr. Annis, Dr. Resch, Ms. Kilpatrick, Ms. Murray

Florida State Hospital • P. O. Box 1000 • Chattahoochee, FL 32324-1000
Working in partnership with local communities to help people be self-sufficient and live in stable families and communities
REFERENCES


Name: Maureen Y. Lyons Reardon
Birth Date: March 3, 1975
Birth Place: Danbury, Connecticut

Education:
2003 – Present Clinical Psychology Internship (APA Approved)
Department of Psychiatry, University of North Carolina, Chapel Hill, NC
Federal Correctional Complex, Butner, NC
2001 – Present Doctoral Candidate, Clinical Psychology
Florida State University, Tallahassee, FL
   Major Research Interests: substance use in special populations (e.g.,
correctional institutions, homeless, suicidal, schizophrenic), forensic
evaluation, psychotherapy outcome, and acute alcohol tolerance.
   Professor Directing Dissertation: Alan R. Lang, Ph.D.
1997-2000 Master of Science, Psychology
Florida State University, Tallahassee, FL
   Thesis Title: The influence of extraversion on the role of operant
learning in the augmentation of acute alcohol tolerance.
   Professor Directing Thesis: Alan R. Lang, Ph.D.
1993 – 1996 Bachelor of Science, Psychology, Summa Cum Laude
University of Connecticut, Storrs, CT,
   Honors Thesis: Effects of thioridazine on lever pressing and tacrine-
induced vacuous jaw movements: Clinical implications for the treatment
of schizophrenia.
   Professor Directing Thesis: John D. Salamone, Ph.D.

Honors and Awards:
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Department of Psychology, Florida State University
2002 Dissertation Research Grant
Office of Graduate Studies, Florida State University
1998, 2002 Student Merit Award, Research Society on Alcoholism
2001 Kennedy-Shaeffer Graduate Award of Excellence in Clinical Training
Department of Psychology, Florida State University
1998-99, 2000 University Fellowship, Florida State University
1997-1998 College Teaching Fellowship, Florida State University
1996-Present  
*Phi Kappa Phi National Honor Society*

1993-1996  
*Thomas J. Watson Memorial Scholarship, I.B.M.*

1993-1996  
*Presidential Academic Scholarship, University of Connecticut*

**Publications:**


