COMORBIDITY OF SEASONAL AFFECTIVE DISORDER WITH SCHIZOPHRENIA IN THE EXTREME NORTH

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SCHIZOPHRENIA IN THE EXTREME NORTH

A
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Abstract

The purpose of this study was to provide estimates of the prevalence of seasonal affective disorder (SAD) among Alaska residents with schizophrenia and to evaluate the relation of SAD symptoms to symptoms of general depression, negative schizophrenia, and alcohol abuse. Nine (33%) of the subjects exceeded cut-off criteria for SAD. Assessment of depression supported the diagnostic classification of respondents who met cut-off criteria for SAD as depressed, but also supported conceptualizations of SAD as a syndrome separate from unipolar depression. Evaluation of negative symptoms of schizophrenia validated the divergence of SAD and depression symptoms from negative symptoms. Implications of this study are discussed in terms of a potential heightened vulnerability to SAD among people with schizophrenia. SAD is prevalent among general population residents in the northern latitudes. Therefore, a heightened risk for comorbid SAD is a potential issue for clinical management among people with schizophrenia in the North.
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Seasonal Affective Disorder Comorbidity with Schizophrenia in the Extreme North

Introduction

Drastic climate changes in hours of daylight and temperature exert an influence on those who dwell in the far north. Fairbanks, Alaska, lies at the latitude of 64 degrees north, just below the Arctic Circle and Anchorage, Alaska lies at 60 degrees north. Roughly thirty percent of the residents of Fairbanks suffer from a severe or mild form of seasonal affective disorder (SAD; Booker & Hellekson, 1992). What effect does extreme climate change have on the thoughts, feelings, and behaviors of a person with schizophrenia? Are the SAD rates equally high among Alaska residents with and without schizophrenia?

The aim of the present proposal is to assess the prevalence of comorbid SAD among northern latitude residents diagnosed with schizophrenia to that of a published control data set. The research idea is presented in four sections. First is a definition of terms and review of relevant literature. In the rationale section, the hypotheses are stated and justified. The third section describes the methods of data collection. The analysis portion presents the various statistical analyses used.

People living in high latitudes are more likely to suffer from SAD than those in lower latitudes (Rosen et al., 1990; Rosenthal, 1993). The relationship between
schizophrenia and depression has been well documented (Bernard, Lancon, Auquier, Reine, & Addington, 1998; Harrow, Yohan, Sands, & Marengo, 1994; Siris, 1991; Tollefson, Sanger, Lu, & Thieme, 1998). Seasonal variations in relapse rate, prolactin levels, and serotonin levels among people suffering from schizophrenia have been documented (Abe, Suzuki, & Egashira, 1992; Brown et al., 1988; Jakovljevic et al., 1997). However, the relationship between schizophrenia, high latitude, and seasonal mood variations has not yet been examined.

Seasonal Affective Disorder

Description

SAD, or "major depressive disorder with seasonal pattern" (American Psychiatric Association, 1994), is characterized by seasonal fluctuation in sleep patterns, eating patterns, weight, mood, libido, and energy level, with a craving for carbohydrates during the depressive cycle. Females are more susceptible to SAD than men (Kasper, Rogers, et al., 1989; Rosen et al., 1990; Rosenthal, 1993), experiencing it in a ratio of about 4:1 (Hellekson, 1989). The prevalence of SAD also appears to increase as degrees of latitude increase (Rosen et al., 1990; Potkin, Zetin, Stamenkovic, Kripke, & Bunney, 1986).

A person diagnosed with winter SAD has a history of at least one major depressive episode along with regularly occurring autumn-winter depressions (at least 2 occurring during consecutive winters) alternating with nondepressed periods during spring and summer. No psychosocial variables account for these seasonal mood changes.
Subsyndromal SAD (S-SAD) is a less severe form of the disorder. Those who suffer from this mild winter dysfunction have physiological changes similar to those in SAD but have never had an episode of major depression (Rosen et al., 1990).

Winter depression is the most common manifestation of SAD. It tends to recur annually in October-December and remit in the spring, usually in March. Hypomania often follows this remission in late spring or summer. Winter depression is characterized by hypersomnia, overeating, and carbohydrate craving, whereas insomnia and loss of appetite are features of major depressive disorder (Rosen et al., 1990).

In a minority of people with SAD, the depression occurs in the spring or summer and fall or winter brings hypomania. For instance, Lingjaerde, Bratlid, and Hansen (1985) documented a phenomenon of midwinter insomnia among Norway residents. Other scientists concur that this reverse pattern is common in many regions north of the Arctic Circle (Boyce & Parker, 1988; Rosenthal, Sack et al., 1984; Wehr, Sack, & Rosenthal, 1987).

The diagnosis of SAD has predictive and construct validity. In a longitudinal study of 59 participants with SAD, Schwartz, Brown, Wehr, and Rosenthal (1996) found that the timing of depressive episodes within individuals was consistent. Among these 59 participants, relapse symptomatology, duration, and seasonality remained constant (Schwartz et al., 1996). They concluded that the diagnostic criteria of winter SAD (Rosenthal et al., 1984b) has predictive diagnostic validity. Another study by Sakamoto, Nakadaira, Kamo, Kamo, and Takahashi (1995) tracked the longitudinal course of 41 participants...
participants who met diagnostic criteria for SAD at baseline. After a mean of 10 years, 22% of the participants still met SAD criteria. These results affirm the validity of SAD as a distinct subtype of recurrent affective illness.

**Pathophysiology**

The antidepressant effects of phototherapy suggest biologic mechanisms play a role in SAD. About 80% of SAD patients who undergo phototherapy have at least temporary alleviation of their depressive symptoms (Rosenthal, Genhart, Jacobsen, Skwerer, & Wehr, 1987). Phototherapy reduces symptoms in either of two ways: by increasing the intensity of light, or by extending the photoperiod (the period of time a person is exposed to light). In a pilot study, Rosenthal et al. (1984b) discovered that extending the photoperiod by means of bright white artificial light might have significant anti-depressant effects. These findings have since been replicated (Wehr et al., 1986; Hellekson, Kline, & Rosenthal, 1986; Terman et al., 1989; Kasper et al., 1989a; Schwartz et al., 1996).

The efficacy of phototherapy is causing scientists to take a closer look at the biological effects of bright light on the human organism. As we discover significant physiological differences between people who suffer from SAD and people who do not, we come closer to uncovering the disease entity, thus finding optimal treatment. For instance, people who suffer from SAD may have disorders in melatonin secretion, circadian rhythm, light sensitivity, or serotonin transmission. These four hypotheses, based on the success of phototherapy, have received vast attention in the literature.
Disorders in melatonin secretion. Both melatonin and light seem to regulate a person’s daily sleep/wake cycle, or circadian rhythm (Lewy, Sack, & Singer, 1985). Melatonin, a hormone produced in the brain’s pineal gland, is secreted to other parts of the brain that contain melatonin receptors. The suprachiasmatic nucleus (SCN) has many such receptors. Their activation regulates circadian rhythms. During the day, the SCN sends a rhythmic inhibitory signal to the pineal gland, resulting in low level melatonin production. At night, a rhythmic excitatory signal from the SCN results in high-level melatonin release, causing a person to feel drowsy. Melatonin levels are the highest during heavy sleep. Therefore, the hypothesis is that if levels of melatonin secretion relate to a person’s circadian cycle, and phototherapy restores a person’s disturbed circadian cycle, then a baseline disturbance of melatonin levels will be found among people suffering from SAD.

Many studies have described this hypothesis (Lewy & Sack, 1997; Lewy, Sack & Singer, 1985; Wehr, 1991). In a study comparing levels of melatonin secretion and symptoms of depression, Wehr et al. (1986) found that phototherapy, while reducing symptoms of winter depression, had no effect on levels of melatonin secretion. In a double-blind, crossover design study comparing the effects of oral melatonin and placebo, Rosenthal et al. (1986) found no changes in the Hamilton Rating Scale for Depression (Hamilton, 1960). Scores were significantly higher in the melatonin condition on ratings of overeating, oversleeping, carbohydrate craving, and weight gain (core features of SAD). Using another double-blind, crossover design, Rosenthal et al.
(1988) administered placebo or the beta-blocker atenolol, a melatonin inhibitor, to participants with SAD. By measuring plasma melatonin profiles, they found that although melatonin secretion stopped, SAD symptoms were not alleviated. In another study, Waldhauser, Ehrhart, and Forster (1993) could not substantiate alterations in melatonin levels among participants suffering from SAD or major depressive syndrome. Further research is necessary to determine the role of melatonin secretion in SAD.

**Disorders in circadian rhythm.** The rhythm of physiological responses such as sleep, eating, and body temperature is regulated by an internal biological “clock” located in the SCN. Daily light/dark cycles influence the regulation of this internal clock. Photoperiodic messages are sent to the retina. The optic nerve transmits these messages from the retina directly to the SCN. People suffering from SAD experience disturbances in their sleep cycles, eating habits, and body temperatures. Therefore, there may be a relationship between SAD, disturbances in circadian rhythm, and photoperiod. This hypothesis is further explained in several studies (Rosenthal & Wehr, 1992; Rosenthal et al., 1988; Jacobsen, Sack, Wehr, Rogers, & Rosenthal, 1987).

Lewy, Sack, and Singer (1985) and Dahl et al. (1993) found that the timing of phototherapy was integral to its efficacy. They hypothesized that early morning light administration, like early morning sun, adjusts disordered circadian rhythms. However, some studies have shown that the intensity of the light predicts favorable outcome in phototherapy (Hellekson et al., 1986; Rosenthal et al., 1984b; James, Wehr, Sack, Parry, & Rosenthal, 1985). As further research resolves the discrepancy between timing and
intensity in phototherapy, we will more clearly understand the role of circadian rhythms in SAD.

Disorders in light sensitivity. Symptom reduction due to phototherapy has led to investigation of the structural conduit and chemical reactions of light perception. Most research has investigated visual photoreception. In the visual sensory system, the retina contains two types of photoreceptor cells: rods and cones. When light contacts these molecules, they send chemically charged information along the optic nerve to the SCN. Phototherapy may exert its ameliorative influence by stimulating photoreceptor cells. Therefore, the photoreceptor cells of a person suffering from SAD may have reduced light sensitivity. Research by Wirz-Justice et al. (1993) lends support to this hypothesis.

Disorders in serotonin transmission and synthesis. Serotonin may have a role in the pathophysiology of SAD and the mechanism of action of phototherapy (Rosenthal et al., 1984b; Rosenthal & Wehr, 1992). People who suffer from SAD may have a deficiency in serotonin.

Levitan, Rector, & Bagby (1998b) and Schwartz et al. (1997) describe five findings that lend credence to the serotonin hypothesis. First, environmental light regulates serotonin (Cagampang & Inouye, 1994) and phototherapy assuages symptoms of winter depression. Second, carbohydrate-rich meals elevate serotonin levels by increasing tryptophan uptake in the brain (tryptophan is the metabolic precursor to serotonin). Such meals energize participants with SAD but sedate normal controls (Rosenthal et al., 1987). Third, those symptoms which distinguish SAD from major
depressive disorder (e.g., hypersomnia, increased appetite, and carbohydrate craving) are regulated by serotonin. Fourth, medications that enhance serotonin transmission (e.g., tryptophan, fenfluramine and fluoxetine) effectively reduce symptoms of winter depression (Levitan, Kaplan et al., 1998). Fifth, serotonin metabolism changes seasonally, decreasing in the winter (Carlsson, Svennerholm, & Winblad, 1980).

In short, favorable outcomes from phototherapy suggest a biologic pathogenesis of SAD. Disorders in melatonin secretion, circadian rhythm, light sensitivity, and serotonin have been implicated.

Prevalence

Rosenthal (1993) reports that an estimated 10 million United States residents meet diagnostic criteria for SAD, and another 25 million suffer from the less severe S-SAD. Prevalence rates increase as degrees of latitude increase. Winter SAD afflicts 1.4 percent of the population of Florida and 3.6 percent of that of California, Arizona, Texas and Georgia. In Oregon, Pennsylvania, New York, and Vermont, the rates increase to 8 percent, with an additional 17 percent suffering from S-SAD (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989b). Farther north still, more than 10 percent of residents of Minnesota, North Dakota and Maine meet criteria for SAD and 20 percent for S-SAD (Rosenthal, 1993).

In a study of prevalence in four locations, Rosen et al. (1990) also found that in the more northern latitudes, rates of winter SAD and S-SAD were significantly higher and no correlation between summer SAD and latitude was found. Rosen et al. (1990).
Also, Potkin et al. (1986) found a correlation of $r = .85$ between latitude and SAD prevalence. Lingjaerde, Bratlid, Hansen, & Grotestam (1986) found a higher prevalence of winter depression in the northern counties of Norway than in the southern counties. Among a stratified representative community sample of 283 residents of Fairbanks, Alaska, Booker and Hellekson (1992) found the prevalence rate of SAD was 9.2%, and the rate of the less severe S-SAD was 19.1%.

**Depression, SAD and alcohol abuse**

There are two forms of alcohol use disorders referred to in this study: alcohol dependence and the less severe alcohol abuse. In the United States, a diagnosis of alcohol dependence is made when a person experiences three or more of the following during a single 12-month period: (1) tolerance of alcohol; (2) withdrawal symptoms upon the discontinuation of alcohol use; (3) amount or duration of use is often greater than intended; (4) the person repeatedly tries without success to control or reduce alcohol use; (5) the person spends much time using alcohol, recovering from its effects, or trying to obtain it; (6) the person reduces or abandons important work, social, or leisure activities because of alcohol use; or (7) the person continues to use alcohol despite knowing that it has probably caused ongoing physical or psychological problems (American Psychiatric Association, 1994).

A less severe diagnosis of the less severe alcohol abuse is made when a person experiences one or more of the following during a single 12-month period: (1) because of repeated use, the person fails to carry out major obligations at home, work, or school; (2)
the person repeatedly uses alcohol even when it is physically dangerous to do so; (3) the person repeatedly has legal problems resulting from alcohol use; or (4) despite knowing that it has caused or worsened social or interpersonal problems, the person continues to use alcohol (American Psychiatric Association, 1994).

Alcohol use disorders among people suffering from major depression are pervasive in the general population. Specifically, Grant and Harford (1995) found a greater association between alcohol dependence and major depression than between alcohol abuse and major depression. Among 123 alcohol rehabilitation patients, Hasin, Grant, and Endicott (1988) found a high lifetime prevalence of comorbid major depressive disorder. Among 127 participants with both alcohol abuse and major depressive disorders, Hasin et al. (1988) found that sustained alcoholism remission strongly increased the chances of sustained depression remissions over a period of five years.

A few investigators have studied the relationship of SAD and alcohol abuse. In a study by McGrath and Yahia (1993), a number of Veterans Administration inpatients reported a fall-winter pattern of alcohol use. Of this number, 6 met criteria for seasonal alcohol dependence (based on DSM-III-R criteria for seasonal depression). In a comparison of 34 patients with SAD and 34 patients with nonseasonal mood disorders, Allen, Lam, Remick, and Sadovnick (1993) found that alcoholism was found more frequently in the relatives of the SAD patients. In conclusion, alcohol dependence and
abuse are prevalent among people suffering from major depression, and investigators are taking a closer look at the relationship of alcohol use and SAD.

Factors in extreme north latitudes

Specific attributes of far-northern climates may account for increased SAD prevalence rates. Drastic changes in light and temperature correspond with changes in mood among people with SAD and the less severe S-SAD (Booker & Hellekson, 1992). At 60 and 64 degrees north respectively, Anchorage and Fairbanks, Alaska, have extreme seasonal contrasts. In Fairbanks, winter temperatures are often colder than 20 degrees below zero Fahrenheit for months at a time, while the summer can bring temperatures above the eighties.

Before describing the drastic changes in light in the extreme north, it is necessary to discuss “civil twilight.” Civil twilight is the period of time when the sun is from the horizon to 6 degrees below the horizon (Leibowitz & Owens, 1991). This is the limit at which twilight illumination is sufficient for a person to clearly distinguish objects. The brightest stars are visible and the horizon is clearly defined in the morning before the beginning of civil twilight and in the evening after the end of civil twilight. Table 1 offers the amount of available daylight in Fairbanks and Anchorage during the darkest and lightest days of the year, excluding and including civil twilight.
<table>
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<th>Hours of possible sunshine:</th>
<th>Hours of possible sunshine including civil twilight:</th>
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<tr>
<td><strong>Fairbanks:</strong></td>
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<td><strong>Anchorage:</strong></td>
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<td>June 21\textsuperscript{st}</td>
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<td>June 21\textsuperscript{st} 24:00</td>
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Edward Plumb, Meteorological Technician, National Weather Service Fairbanks, emphasizes the importance of considering civil twilight in the study of SAD:

With civil twilight included in a day, the actual amount of "useable" daylight is greatly increased. In December, it adds almost an hour and a half of light before and after sunrise/sunset. Then in the summer, Fairbanks experiences civil twilight every night while the sun is below the horizon from mid May through the end of July. Just looking at the possible sunshine hours can be misleading. In December, the sun is above the horizon for 3 hours and 42 minutes, but it is light much longer than that. Then in the summer, it is light all night (for about 2 months) even though the sun sets for a few hours each night. Twilight is something that should be considered when studying SAD at high latitudes because it really contributes to our total daily amount of light (Edward Plumb, personal communication, April 14, 1999).

Including civil twilight, the total daylight in mid-December in Fairbanks and Anchorage is similar to that of populous cities such as Vancouver, B.C., Seattle, and Tacoma, Washington, Minneapolis, Minnesota, Boston, Massachusetts, and many other municipalities. These dramatic conditions of daylight and temperature provided the backdrop for the present proposal to assess the prevalence of SAD among northern dwellers diagnosed with schizophrenia: a subject yet to be addressed in the literature.
Schizophrenia

Description

Schizophrenia separates a person from her capacity to experience emotions, express feelings, and think clearly. The symptoms of schizophrenia involve disturbances in perception, thought, speech and language, emotion, social interactions and behavior, volition, creativity, and the capacity to think abstractly (Andreasen, 1997b). People who suffer from this disorder often have strange and terrifying internal perceptions and experiences, feelings of estrangement, and the sense that personal autonomy is being violated or completely usurped (Andreasen, 1994).

In the United States, a diagnosis of schizophrenia is made when a person experiences two or more of the following for at least 6 months: delusions, hallucinations, disorganized speech (e.g., difficulty staying on the subject or incoherence), grossly disorganized or catatonic behavior, or negative symptoms (American Psychiatric Association, 1994). Symptoms such as affective flattening (lack of emotional expression), alogia (poverty of thought and speech) and avolition (inability to formulate and execute plans) are called “negative” because they represent a diminution of normal behavior. Symptoms such as delusions, hallucinations, disorganized speech and disorganized or catatonic behavior are characterized as “positive” because they represent an exaggeration of normal behavior (Andreasen, 1997a).

Most delusions and hallucinations stem from over-acuteness of the senses and the brain’s inability to filter, interpret, and respond to incoming stimuli (Torrey, 1995). One
of Torrey’s (1995) patients describes the difficulty to comprehend visual stimuli: “I have to put things together in my head. If I look at my watch I see the watchstrap, watch, face, hands and so on, then I have got to put them together to get it into one piece” (p. 29).

Some people with schizophrenia may think the television is sending special messages just to them. They may believe that others’ thoughts are being inserted into their mind, or that their own thoughts are being suddenly withdrawn with no reason or antecedent. While a person can experience hallucinations of any of the five senses, auditory hallucinations are the most common in schizophrenia. Murphy (1997) describes her “voices”:

The demons mocked and scorned me and sounded menacing. Even though the voices told me to do things, I never did what they said. Sometimes the voices came from machines. A running vacuum cleaner called me filthy names. Laundry machines, air conditioners, cars, and motorcycles all taunted me. The flame on the gas stove also spoke (p. 542).

Hallucinations and delusions can be frightening and disturbing, both to the person experiencing them and to nearby loved ones and strangers.

The course of schizophrenia is characterized by alternating periods of relapse and remission. During a relapse, hallucinations and delusions may become so severe as to prevent a person from eating, sleeping, interacting with others, and executing daily life functions. Pharmacological and psychosocial treatment in an inpatient medical center ordinarily attenuates these symptoms. A person is generally discharged from the hospital
as soon as these positive symptoms remit. Upon return home, a person may be unable to
work or enjoy life due to persistent negative symptoms. Common manifestations of
negative symptoms are social and emotional withdrawal, avolition, flat or blunted affect
(lack of emotional expression), anhedonia (loss of the ability to feel pleasure), narrowing
of ideation (loss of the ability to concentrate or focus attention), and alogia (Andreasen &
Olsen, 1982a; Andreasen, 1985; Andreasen, Swayze, Flaum, O’Leary, & Alliger, 1994;
Buchanan & Gold, 1996).

Family members of a person with schizophrenia find that coping with the negative
symptoms of their loved one is especially difficult. In a study of caregiver burden in the
relatives of people with schizophrenia, Provencher and Mueser (1997) found that
negative symptoms may have a greater impact on daily role functioning than positive
symptoms, leading to greater burden. Family members find the emotional burden very
heavy as well. Willick (1994) writes:

I feel the loss of a son that I once had, because in many ways he is quite
different...he has lost that gleam in his eye, that joyous good humor, that zest for
life which he once showed. Today, it is hard for him to feel things strongly, or to
enjoy his music, sports, or being with the family...Some of the expressions that
came into my mind [to describe this difference] were these: ‘He has become a
shell of a person, there is no one there, he has lost his self, he looks different, his
face has changed, he’s a lost soul’ (pp. 8-9).
In short, although they do not command the immediate attention that positive symptoms do, negative symptoms are in many ways the more debilitating features of schizophrenia.

Hughlings-Jackson first distinguished positive from negative symptoms in 1931 (Taylor, 1931). He wrote that negative symptoms represent a pure loss of function, while positive symptoms represent an exaggeration of normal function. This symptom distinction was revived in 1980 when Crow proposed that schizophrenia could be divided into Type I and Type II syndromes (Crow, 1980; 1985). He wrote that people who suffer from Type I schizophrenia have a good response to conventional neuroleptics (e.g. haloperidol, fluphenazine, chlorpromazine), a favorable prognosis, an acute (sudden) onset, and normal brain structure. Type I schizophrenia is characterized by positive symptoms including cognitive deficits (disorganized speech, formal thought disorder, or loose associations). He explained that people with Type II schizophrenia do not respond to conventional neuroleptics, have a poor prognosis, an insidious (slow) onset, and anatomical brain differences. Type II schizophrenia is characterized by negative symptoms. Neuropsychological comparisons of positive (Type I) and negative (Type II) schizophrenia (Zakzanis, 1998) lend further support to this dichotomy. Crow (1985) highlighted that this distinction oversimplifies a complex disease process. He clarified that because both Type I and Type II syndromes often occur in the same person, they may have the same pathogenesis (Crow, 1985).

Factor analytic studies of symptoms have revealed two potential subcategories of positive symptoms: psychosis and disorganization (Andreasen, 1997b; Arndt, Andreasen,
The psychosis category is composed of delusions and hallucinations, while the disorganization category consists of disorganized speech, bizarre behavior, and inappropriate affect (Andreasen, 1997b).

Carpenter and colleagues first compartmentalized negative symptoms into primary and secondary categories (Carpenter, Buchanan, & Kirkpatrick, 1991; Carpenter, Heinrichs, & Alphs, 1985; Carpenter, Heinrichs, & Wagman, 1988). Primary negative symptoms are core features of schizophrenia. They are due to a core deficit in the brain. Atypical antipsychotics (e.g. clozapine, risperidone, olanzapine, experimental medications) have been used to treat primary negative symptoms with limited success (Breier, 1996).

Secondary negative symptoms are due to an external force, and can be alleviated to the extent that the external force can be manipulated or removed. There are four external forces that cause secondary negative symptoms (Andreasen, 1997b; Andreasen, Nopoulos, et al., 1994; Earnst & Kring, 1997). First, antipsychotic medication can cause extra-pyramidal side effects such as sedation and affective blunting (Van Putten, May, & Marder, 1984). Second, depression or demoralization can be the root of negative symptoms. Third, an understimulating environment (such as that of a mental health inpatient ward) can lead to avolition, withdrawal, and anhedonia. Fourth, positive symptoms may cause negative ones. For instance, withdrawal may be secondary to a core persecutory delusion. Alternately, an individual may withdraw or remain silent in order to integrate incoming sensory stimuli. A patient of Torrey (1995) describes this
phenomenon: “Everything is all right when I stop. If I move, everything I see keeps changing, everything I’m looking at gets broken up and I stop to put it together again” (p. 61).

A “syndrome” is a set of symptoms occurring together. The members of a group of syndromes are phenomenologically similar but pathophysiologically different. Most clinicians agree that what we call “schizophrenia” is actually a group of heterogeneous syndromes. A “pathognomonic feature” is a symptom experienced by every person with a particular disease. Evidence from selective neuroleptic response, brain imaging techniques, and clinical observations affirms that schizophrenia does not have a single pathognomonic feature. Currently, most clinicians and researchers identify two syndromes of schizophrenia: positive and negative.

Although it lends itself to clean diagnostic criteria and treatment options (Andreasen, 1997b), there are three difficulties with the positive-negative syndrome typology (Lindenmeyer, Bernstein-Hyman, Grochowski, & Bark, 1995). First, most people suffering from schizophrenia experience a mixture of positive and negative clinical features (a mixed syndrome). Second, criteria for what constitutes a positive and negative syndrome are variable: there is currently no consensus on diagnostic criteria. Third, it is difficult to distinguish primary from secondary negative symptoms. The inability to definitively identify the syndromes that comprise schizophrenia is due not to a lack of research but rather to the blurred boundaries of these constructs.
Pathophysiology

Researchers differ in their opinions regarding the causes of schizophrenia. Most recent research has investigated biological theories via brain imaging studies and pharmaceutical trials of antipsychotic medications. Kleinman (1988) criticizes an overemphasis of biological factors and a lack of attention to cultural factors in schizophrenia. However, mental health advocacy groups such as the National Alliance for the Mentally Ill emphasize that schizophrenia is a disease of the brain and should be accorded equal status with all other physical diseases (the National Alliance for the Mentally Ill, no date provided). Most biological findings suggest that differences in brain structure, neurotransmission, or neurodevelopment may be implicated in schizophrenia.

Differences in brain structure. Many studies have revealed anatomical differences in the brains of people with schizophrenia such as ventricular enlargement, hypofrontality, frontal and temporal lobe impairments, and cortical atrophy.

Ventricular enlargement: Cerebral ventricles are the three ducts in the brain through which our cerebral spinal fluid flows. The results of numerous brain imaging studies reveals that ventricles of people with schizophrenia are significantly larger than those of controls (Johnstone, Crow, Frith, Husband, & Kree, 1976; Crow, 1980; Andreasen, 1982).

Hypofrontality: The hypo-(diminished) frontality (frontal lobe) explanation of schizophrenia stems from the fact that the frontal lobe is the seat of abstraction and attention, two areas affected by schizophrenia. Positron emission tomography (PET) and
single photon emission computed tomography (SPECT) reveal high or low cerebral blood flow in specific brain regions, reflecting heightened or decreased activity. Many investigators have used these functional neuroimaging techniques while participants are engaged in neuropsychological tests designed to activate specific brain regions. In a summary of 20 years of such studies, Andreasen, Swayze, et al. (1994) concluded that people with schizophrenia consistently show decreased frontal cerebral blood flow both at baseline and while engaged in cognitive tasks requiring the use of this region. Thus, the frontal lobe may be less active in schizophrenia and may be part of its pathogenesis.

Frontal and temporal lobe impairments: Neuropsychological investigations and studies of the effects of head trauma and stroke have identified the frontal lobe as the seat of abstraction and attention (Andreasen, 1994). These studies have also revealed that the temporal lobe is the locus of learning and memory. Some forms of schizophrenia are characterized by deficits in abstraction, attention, learning, and memory. Thus, as Crow (1985) suggests, people suffering from Type II (negative) schizophrenia may have frontal and temporal impairments.

Cortical atrophy: Traditional radiography is not sophisticated enough to illustrate the soft gray and white matter of the cortex. However, magnetic resonance imaging (MRI) has this sensitivity. An MRI scan can create several two-dimensional images of the brain from different angles. Thus, neurologists can conceptually piece the images together to have a three-dimensional idea of brain structures. MRI studies reveal that the mass of the entire cortex may be decreased among people with schizophrenia (Sullivan,
Shear, Lim, Zipursky, & Pfefferbaum, 1996; DeLisi et al., 1995; Harvey, Persaud, Ron, Baker, & Murray, 1994).

**Differences in neurotransmission.** Symptoms of schizophrenia are affected by medications that alter neurotransmission. Dopamine is a neurotransmitter involved in muscle movement, emotions, behavior, prolactin release, and food intake. In the 1950s, Carlsson developed the “dopamine hypothesis” based on the ability of chlorpromazine, the first neuroleptic, to reduce the positive symptoms of schizophrenia through dopamine blockade (Snyder, 1973).

Three observations support this hypothesis. The first comes from similarities between positive symptoms of schizophrenia and symptoms of amphetamine psychosis. Amphetamines cause the release of presynaptic dopamine and norepinephrine. Moreover, neuroleptics attenuate the symptoms of amphetamine psychosis (Snyder, 1973; Angrist, Sathananthan, Wilk, & Gershon, 1974; Bleich, Brown, Kahn, & Van Praag, 1988). The dopamine theory is also strengthened by observing pharmacologic treatment response in Parkinson’s disease, caused by too little dopamine. Parkinson’s patients treated with L-DOPA, the metabolic precursor of dopamine, often develop psychotic symptoms. Lastly, PET studies have shown that individuals with schizophrenia have a higher density of dopamine receptors in a part of the brain called the caudate nuclei (Bleich et al., 1988).

Traditional neuroleptics reduce positive symptoms of schizophrenia. Newer medications, called “atypical antipsychotics” (e.g. clozapine, risperidone, olanzapine,
experimental medications), may be effective at reducing negative symptoms. They exert their influence on other neurotransmitter systems along with dopamine. Clozapine, the parent atypical antipsychotic, reduces negative symptoms in many people. This drug combines serotonin antagonism with dopamine antagonism. Other experimental antipsychotic medications have serotonin reuptake inhibition, cholinergic mechanisms, \( \alpha-2 \) antagonism, NMDA receptor agonism, and neuropeptide antagonism (Ames, Marder, Wirshing, & Van Putten, 1996). These systems, therefore, in addition to dopamine, may be implicated in the pathogenesis of schizophrenia. Results from clinical trials of atypical antipsychotics are leading to a clearer understanding of the role of specific neurotransmitters in schizophrenia.

**Differences in neurodevelopment.** Searching for the cause of schizophrenia, many investigators have turned their attention to brain development. Minor physical anomalies (MPAs) of the head, feet, hands and face have been discovered among people with schizophrenia (Green, Satz, & Christenson, 1994). MPAs reflect abnormalities in fetal neurodevelopment. These anomalies may reflect early, largely extra-genetic stressful events in fetal development. Prominent symptoms of schizophrenia typically appear when individuals reach their late teens or early twenties. Thus, schizophrenia may result from prenatal abnormalities in neural development. It is possible that this abnormality is latent until the affected region matures and is required to function properly. At this time, the symptoms of schizophrenia appear.
Many studies have reported the high prevalence of winter births among people with schizophrenia. Because the second trimester of gestation is a critical period for fetal brain development, one hypothesis is that second trimester influenza leads to abnormal neurodevelopment. Some studies have documented a correlation between prenatal influenza and adult schizophrenia (Mednick, Machon, Huttunen, & Bonnet, 1988; Barr, Mednick, & Munk-Jorgenson, 1990; O'Callaghan, Sham, Takei, Glover, & Murray, 1991) while others have not (Morgan et al., 1997; Selten & Slaets, 1994; Susser, Lin, Brown, Lumey, & Erlenmeyer-Kimling, 1994; Crow & Done, 1992).

In short, research involving brain imaging, antipsychotic response, and neurodevelopment reveal structural, chemical, and developmental differences (respectively) in the brains of people suffering from schizophrenia. Therefore, these differences may have a role in the pathophysiology of schizophrenia.

Prevalence

Schizophrenia afflicts 1% of the population worldwide, including 2.6 million people in the United States (Breier et al., 1996). In the U. S., people with schizophrenia occupy 25% of all hospital beds. Costs associated with lost productivity and treatment are estimated at $30 billion per year. The life expectancy in schizophrenia is 20% shorter than the national average. The suicide rate is an alarming 10% (Breier, 1996; Torrey, 1995). The course of schizophrenia seems to be more favorable in developing than in developed societies (World Health Organization, 1973; Sartorius et al., 1986; Lin & Kleinman, 1988). One reason for this discrepancy is the possibility that diagnosticians in
developed societies are more conservative in their diagnoses of schizophrenia (Edgarton & Cohen, 1994). Another hypothesis is that community and family support ties are stronger in developing societies, thus providing better treatment (Lin & Kleinman, 1988).

There remains some controversy regarding the effects of culture on the course and outcome of schizophrenia. Lin and Kleinman (1988) reviewed approximately fifteen international longitudinal studies that have produced results indicating that the outcome of schizophrenia is better in non-western cultures than in western, industrialized societies. Two of the largest and most important of these studies were the multi-centered International Pilot Study of Schizophrenia (IPSS; World Health Organization, 1973) and the subsequent Determinants of Outcome in Severe Mental Disorders (DOSMD) study (Sartorius et al., 1986), both conducted by the World Health Organization (WHO). In their review, Lin and Kleinman speculated on cultural, social and familial mechanisms that may underlie the better prognoses in non-western societies. These mechanisms are social isolation and social support, family milieu, the nature of employment and the sociopolitical milieu, stigma/self-attribution/sick role, and differential survival of vulnerable individuals.

On the other hand, Edgarton and Cohen (1994) provided a critical evaluation of the WHO's IPSS and DOSMD studies (both of which indicated that the course of schizophrenia is more favorable in developing than in developed societies). Edgarton and Cohen presented summaries of literature demonstrating more favorable outcome in industrialized societies. The investigators addressed methodological shortcomings of the
IPSS and DOSMD such as misdiagnosis, high attrition in certain countries, and choosing an unrepresentative sample of developing societies. While the WHO studies report that cultural factors alone account for this difference in course, Edgerton and Cohen point out that malaria, trypanosomiasis, drug use, diet, and type of onset (acute or insidious) are only sometimes influenced by culture and often affect course.

**Schizophrenia and substance abuse**

Substance abuse among people with schizophrenia is a recognized problem. Estimates of comorbid substance abuse in schizophrenia range from 30% to 57%, and comorbid alcohol abuse from 31% to 47% (Regier et al., 1990; Selzer & Lieberman, 1993; Mueser et al. 1990; Drake et al., 1990; Ziedonis & Fisher, 1994). Results from the Epidemiologic Catchment Area studies indicate that the prevalence of schizophrenia is five times higher among alcoholics than among in nonalcoholics (Helzer & Pryzbeck, 1988).

There are several theories accounting for the high comorbidity of substance abuse with schizophrenia. The stress of the illness could drive some people to abuse drugs. People with schizophrenia often drift downward socially into areas where drug use is endemic (Lieberman & Bowers, 1990). Substances may help a person cope with persistent symptoms and disturbing side effects. Among a sample of 75 rural outpatients with schizophrenia, Osher et al. (1994) found alcohol use disorder was associated with unstable housing, conceptual disorganization, poor insight, and relapse. Selzer and Lieberman (1993) point out that because substance intoxication or withdrawal can cause
psychotic symptoms, achieving diagnostic clarity is difficult. They add that there is a lack of long term treatment settings for the dual diagnosis population. Although no published work so confirms, it is likely that extreme photoperiod and temperature changes in northern latitudes such as Fairbanks and Anchorage contribute to seasonal patterns of substance (particularly alcohol) abuse among people with schizophrenia.

**Schizophrenia and depression**

As early as 1911, Bleuler described two types of depression as features of schizophrenia. He wrote that the first type, resulting from awareness of this debilitating illness (insight), occurs between or after episodes of symptom relapse (Zinkin, 1980). This is referred to as postpsychotic depression (Siris, 1991). The second type is a part of the schizophrenia process itself and occurs throughout the illness, including during the acute phase.

During the last decade, five etiologic theories of comorbid depression in schizophrenia have been widely addressed in the literature: pharmacogenic (drug-induced), akinetic, postpsychotic, psychodynamic, and biologic. In a summary of 28 studies of secondary depression in schizophrenia, Siris (1991) concluded that the differential diagnosis of secondary depression in schizophrenia ought to be included as a diagnostic category in the DSM-IV. Siris (1991) wrote that, according to the 1988 draft of the ICD-10 definition of “Post-Schizophrenia Depression”,

It is uncertain, and immaterial to the diagnosis, to what extent the depressive symptoms have been merely been uncovered by the resolution of earlier psychotic
symptoms rather than being a new development, or to what extent they are an intrinsic part of schizophrenia rather than a psychological reaction to it...It is often difficult to decide which of the patient’s symptoms are due to depression and which to neuroleptic medication or the impaired volition and affective flattening of schizophrenia itself (F20.4).

Investigators are working to tease apart schizophrenia, depression, and negative symptoms. The aim of the present proposal is to detect the prevalence of a separate disease entity, SAD, among high latitude residents suffering from schizophrenia. This is the first important step in addressing a broader question: How do drastic climatic and photoperiod changes affect mood, thoughts, and behaviors in schizophrenia?

**Seasonal variations in schizophrenia**

Although comorbid SAD in schizophrenia has not been addressed, schizophrenia scientists have investigated seasonal variations in relapse rate, prolactin levels and serotonin levels.

Abe et al. (1992) uncovered a seasonal pattern of relapse in 11% of patients diagnosed with schizophreniform psychosis. Data collected from archived admission/discharge data in a psychiatric hospital revealed two opposite patterns of onset, late spring and autumn. The small sample size (n=6) precludes drawing a definite conclusion. Castle, Abel, Takei, and Murray (1995) discovered that females with schizophrenia also had a seasonal pattern of hospital admission. Among females in their
sample, the number of summer admissions was higher than that of winter admissions. These findings are consistent with those of Takei et al. (1992).

Brown et al. (1988) measured seasonal variations in prolactin levels in schizophrenia in Hamilton, Ontario, Canada. In 7 participants, prolactin was significantly higher in spring-summer than in fall-winter. No difference was found among 9 controls. Jakovljevic et al. (1997) discovered seasonal variations in platelet 5HT (serotonin) concentrations among participants in Zagreb, Croatia. In a study comparing patients with schizophrenia, with depression, and participants without a psychiatric diagnosis, spring concentrations of 5HT were significantly higher among those with schizophrenia than the two other groups. Especially in spring, summer and fall, the highest 5HT levels were found in positive schizophrenia patients compared with negative schizophrenia patients and those without a psychiatric diagnosis. This discrepancy clearly separated the positive from the negative types of schizophrenia.

Another interesting finding is that the threshold of high intensity light is higher among participants with schizophrenia than among those with affective disorder (Gerbaldo & Thaker, 1991). Kantor, Browne, Ravindran, and Horn (1991) documented the manic response to phototherapy in one patient diagnosed with schizophrenia. Because the mechanism of action of phototherapy remains controversial, conclusions about the relationship between SAD and schizophrenia cannot be drawn.
Factors in extreme north latitudes

Specific attributes of far-northern climates account for mood variations, represented by increased SAD prevalence rates. Drastic changes in light and temperature correspond to changes in mood among people with SAD and the less severe S-SAD (Booker & Hellekson, 1992). The dramatic seasonal contrasts of Fairbanks and Anchorage, Alaska provide excellent environments in which to study seasonal mood variations among northern dwellers suffering from schizophrenia. Scientists have yet to address this subject. By measuring the prevalence rate of SAD among people suffering from schizophrenia in the far north, the current proposal investigates the relationship between extreme seasonal changes, depression, and schizophrenia.
Rationale

Ten percent of people suffering from schizophrenia commit suicide (Breier, 1996; Torrey, 1995). People with schizophrenia also suffer from comorbid disorders such as depression, substance abuse, and, conceivably, SAD. Positive symptoms of schizophrenia can occlude detection and therapeutic attention to comorbid afflictions because positive symptoms command foremost attention from health care professionals and family members. Nine percent of the northern latitude general population suffers from SAD (Booker & Hellekson, 1992). Thus, nine percent of northern latitude residents with schizophrenia is likely to suffer from SAD or S-SAD as well. Alternatively, given higher rates of depression among people with schizophrenia, SAD rates may in fact be higher. The added symptoms of SAD and the less severe S-SAD may increase the risk for suicide and persistent depressive symptoms in people with schizophrenia.

Decreased daylight in northern locations is associated with higher prevalence rates of SAD and S-SAD (Rosen et al, 1990; Potkin et al, 1986; Lingjearde et al, 1986). Including civil twilight, the total daylight in mid-December in Fairbanks and Anchorage is similar to that of many northern metropolises. If indeed Alaskans with schizophrenia are at greater risk for SAD and S-SAD than the general population, people with schizophrenia living in large cities such as Vancouver, B.C., Seattle, and Tacoma, Washington, Minneapolis, Minnesota, and Boston, Massachusetts may also be at great risk.
The relationship between schizophrenia and depression has been well documented (Siris, 1991; Harrow et al., 1994; Bernard et al., 1998; Tollefson et al., 1998). Seasonal variations in relapse rate (Abe et al., 1992), prolactin levels (Brown et al., 1988), and serotonin levels (Jakovljevic et al., 1997) among people suffering from schizophrenia have been documented. However, the relationship between schizophrenia, high latitude, and seasonal mood variations has yet to be examined.

We know that the symptoms of SAD respond to treatment. Phototherapy assuages some particular affective, vegetative, cognitive and social disturbances that are caused by changes in climate (Rosenthal et al., 1987; Wehr et al., 1986; Hellekson, Kline, & Rosenthal, 1986; Terman et al., 1989; Kasper et al., 1989a; Schwartz et al., 1996). Season-targeted anti-depressant pharmacotherapy successfully treats similar disturbances (Schwartz et al., 1996; Tam, Lam, & Levitt, 1995; Partonen & Lonnqvist, 1995). The symptoms of SAD among people diagnosed with schizophrenia may comprise an exogenous disorder. Alternately, extreme variations in photoperiod or temperature may affect the core features of schizophrenia. In either case, these symptoms can be treated. The symptoms of schizophrenia are frightening to the person experiencing them and to their loved ones. The effects of drastic climate change may compound these disturbing clinical features. People suffering from schizophrenia, their family members, friends and health care providers want relief.
SAD hinders a person from enjoying life. Schizophrenia hampers a person’s ability to experience emotions, express feelings, and think clearly. The combination of these two, probably present in many people in Alaska, would be crippling.

Scientific investigators are working to differentiate between positive schizophrenia symptoms, depression, and negative symptoms. The aim of the present proposal is to detect the prevalence of a separate diagnostic entity, SAD, among high latitude residents suffering from schizophrenia. This question is the first important step in examining the effect of drastic seasonal changes on the mood, thoughts, and behaviors in schizophrenia. Are people who suffer from schizophrenia more sensitive to climate changes than others? Are SAD and schizophrenia separate disease entities? Do climate changes have distinct effects on the specific symptoms of schizophrenia? This study will investigate the prevalence of SAD among Alaska residents diagnosed with schizophrenia.

The hypotheses for this study were: (1) The prevalence of seasonal depression among Alaska residents diagnosed with schizophrenia (ARS) is similar to that among those without this diagnosis; (2) ARS suffer from higher intensity of depression than do those without schizophrenia; (3) There is a positive relationship between severity of negative schizophrenia symptoms and seasonal depression among ARS; (4) There is a positive relationship between severity of negative symptoms and intensity of depression among ARS; (5) There is a positive relationship between alcohol abuse and seasonal depression among ARS; (6) There is a positive relationship between alcohol abuse and intensity of depression among ARS; (7) Among ARS, there is a positive correlation
between intensity of depression measured by a general depression instrument and intensity of depression measured by a depression instrument designed for people with schizophrenia. This correlation will not be a strong one because one instrument measures the construct of general depression and the another instrument measures the construct of depression in schizophrenia (Addington, Addington, & Atkinson, 1996; Collins, Remington, Coulter, & Birkett, 1996); (8) Among ARS, there is a positive relationship between seasonal depression and the severity of four dimensions of negative symptoms: “thought processes,” “cognition,” “volition/motivation,” and “affect/relatedness;” (9) Among ARS, there is a positive relationship between intensity of depression and the severity of four dimensions of negative symptoms: “thought processes,” “cognition,” “volition/motivation,” and “affect/relatedness.”

These two final hypotheses are exploratory in nature and are based on the overlap of some negative symptoms with depressive symptoms (Siris, 1991).
Methods

This section contains descriptions of the methods used in both the general study and the pilot study. The general study addressed the research questions discussed in the two preceding paragraphs while the pilot study assessed interrater reliability on a depression scale.

Participants, general study

Twenty-seven individuals with a DSM-IV diagnosis of schizophrenia as assessed by staff psychiatrists participated in the study. Participants were outpatients in community treatment in Fairbanks, Alaska, or inpatients in the state hospital, based in Anchorage, Alaska. Written informed consent (see Appendix A) was obtained from each participant. One research psychiatrist and/or the primary researcher (a psychology graduate student) verified all diagnoses by chart review and as needed by consultation with their staff psychiatrist. Patients with a history of head injury or schizoaffective disorder were excluded from the study. For the past three consecutive years, all subjects resided at a latitude north of 60 degrees.

The participants were predominantly unemployed, single, outpatient, white male high school graduates with a diagnosis of paranoid schizophrenia. Table 2 presents additional clinical statistics and descriptive characteristics of the sample. The proposal was approved by the University of Alaska Fairbanks Institutional Review Board (IRB),
the Alaska Psychiatric Institute IRB and the management staffs of both the Tanana Chiefs Conference and the Fairbanks Community Mental Health Center.
<table>
<thead>
<tr>
<th>Background Characteristics</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>41 ± 12*</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12 ± 3</td>
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<tr>
<td>Lifetime psychiatric hospitalizations</td>
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<tr>
<td>Age at onset of schizophrenia symptoms</td>
<td>22 ± 7</td>
</tr>
<tr>
<td>Current GAF</td>
<td>48 ± 14</td>
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<tr>
<td>Sex</td>
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<tr>
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<td>5**</td>
</tr>
<tr>
<td>Male</td>
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</tr>
<tr>
<td>African American</td>
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<tr>
<td>White</td>
<td>19</td>
</tr>
<tr>
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<tr>
<td>Schizophrenia</td>
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<td>Paranoid type</td>
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<td>Undifferentiated type</td>
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<tr>
<td>Disorganized type</td>
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<tr>
<td>Residual type</td>
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<tr>
<td>Treatment status</td>
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<tr>
<td>Halfway house</td>
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<tr>
<td>Outpatient</td>
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<td>Separated, divorced or widowed</td>
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<tr>
<td>Employment status</td>
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<tr>
<td>Part time</td>
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</tr>
<tr>
<td>Full time</td>
<td>1</td>
</tr>
</tbody>
</table>

* Mean ± 1 standard deviation.

** Frequency.
Participants, pilot study

Seven individuals with self-reported diagnoses of schizophrenia, bipolar disorder and major depression participated in the interrater reliability study. Participants were members of the Fairbanks chapter of the National Alliance for the Mentally Ill (NAMI-Fairbanks). Written informed consent (see Appendix B) was obtained from each participant. The interrater reliability study was approved by the University of Alaska Fairbanks IRB as an enhancement of the general study.

Instruments, general study

Table 3 lists each of the instruments used in this study and their respective constructs.

SAD and S-SAD. Criteria for SAD and the less severe S-SAD were measured with the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Bradt, & Wehr, 1984). This study used the global seasonality score, comprised of 6 SPAQ questions regarding changes in mood and behavior (See Appendix C). Referred to as the Seasonality Scale Index, these items measure seasonal variations in mood, appetite, weight, sleep, energy, and socialization. Each item is rated as to level on a 5-point scale. The question is worded, ‘To what degree do the following change with the seasons?’ Each of the 5 items is designated by a number (from 0 to 4), and each level is anchored by a verbal label ranging from “no change” to “extremely marked change.” The global
Table 3

Instrument Summary

<table>
<thead>
<tr>
<th>Scale name</th>
<th>Construct measured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seasonal Pattern Assessment Questionnaire</td>
<td>SPAQ</td>
</tr>
<tr>
<td>Center for Epidemiologic Studies Depression Scale</td>
<td>CES-D</td>
</tr>
<tr>
<td>Calgary Depression Scale for Schizophrenia</td>
<td>CDSS</td>
</tr>
<tr>
<td>Negative Symptom Rating Scale</td>
<td>NSRS</td>
</tr>
<tr>
<td>Case Manager Rating Scale for Alcohol Disorder</td>
<td>CMRS</td>
</tr>
</tbody>
</table>

Seasonal affective disorder
General depression
Depression in schizophrenia
Negative symptoms
Alcohol use
seasonality score, a sum of these items, ranges from 0 to 24. In addition, one item on the SPAQ evaluates the degree to which seasonal changes are perceived as a problem, again on a 5-point scale.

For the 6 items of the Seasonality Scale Index, Magnusson, Friis, and Opjordsmoen (1997) found a high internal consistency (α= .82). According to Kasper et al. (1989a), the SSI has convergent validity, correlating well with the Hamilton Rating Scale for Depression. The SPAQ has a positive predictive validity value of 48% and an efficiency of 57% in identifying cases of SAD confirmed by follow-up SAD diagnosis (Raheja, King, & Thompson, 1996). Magnusson (1996) reported that when SAD and S-SAD are combined into a ‘winter problem’ group, the SPAQ’s sensitivity is 94%, its specificity 73%, and its predictive value 45%.

There are limitations to the instrument. For instance, Magnusson (1996) reports that the SPAQ is poor at discriminating SAD from S-SAD, and provides a conservative estimate of both afflictions. Also, the retrospective nature of the SPAQ creates an inherent methodological weakness. Retrospective self-report requires people to remember specific details about their previous illness history. Respondents often cannot accurately remember their age at onset, in which months their symptoms start and end, and the quality of elation during the spring and summer months (Lingjaerde & Reichborn-Kjennerud, 1993).

Previous SAD research has used two different SPAQ cutoffs (Table 4) to assess SAD. Some use a higher threshold (e.g., respondents must score above a certain level to
be categorized as having SAD or S-SAD), while others have used a lower threshold. Using the procedures of Booker and Hellekson (1992), the current investigation uses the conservative (higher) SPAQ cut-off for SAD and S-SAD to err on the side of underestimating the disorders. Specifically, this study assigns a global seasonality score greater than or equal to 11, with perception of the symptoms as a problem (complaints) scored greater than or equal to 2, as a cut-off for SAD. The less severe S-SAD is indicated by either a global seasonality score greater than or equal to 11 with a rating of 0 on complaints or a global seasonality score of 9 or 10 and a rating greater than or equal to 1 on complaints. Some previous SAD research has used these cutoffs while others have used a lower threshold. The higher threshold was chosen to reduce the risk of overestimating SAD and S-SAD. The current study used an interview questionnaire format to administer the SPAQ.

**General Depression.** General depression was rated by the 20-item Center for Epidemiologic Studies Depression Scale (CES-D Scale; Radloff, 1977) which can be found in Appendix D. The CES-D is a composite measure derived from other established measures of depression developed by the Center for Epidemiologic Studies for National Institute of Mental Health research to study depression in the general population. The items on this scale assess the presence and frequency of depressive symptoms (depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness,
Table 4

Two Diagnostic Thresholds of Seasonal Affective Disorder

<table>
<thead>
<tr>
<th></th>
<th>Seasonal affective disorder</th>
<th>Subsyndromal seasonal affective disorder</th>
<th>No seasonal affective disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher threshold*</td>
<td>Global seasonality score ≥ 11 and complaints ≥ 2</td>
<td>Global seasonality score ≥ 11 and complaints rated 0 or 1 or global seasonality score = 9 or 10 and complaints rated 1</td>
<td>Global seasonality score &lt; 9 or global seasonality score = 9 or 10</td>
</tr>
<tr>
<td>Lower threshold</td>
<td>Global seasonality score ≥ 10 and complaints rated ≥ 2</td>
<td>Global seasonality score ≥ 10 and complaints rated 1 or global seasonality score = 8 or 9 and complaints rated 2</td>
<td>Global seasonality score &lt; 8 or global seasonality score = 8 or 9 and complaints rated 1</td>
</tr>
</tbody>
</table>

*The higher threshold was used in this study.

**The global seasonality score was derived from six questions rating sleep length, social activity, weight, appetite, and energy level on a 5-point scale on which 0=no change and 4=extremely marked change. The potential range of scores was 0 to 24.

***The severity of the respondent’s complaints of the combined symptoms was rated on a 5-point scale on which 1=mild, 2=moderate, 3=marked, 4=severe, and 5=disabling.
psychomotor retardation, loss of appetite and sleep disturbance) over the previous week (e. g., ‘I feel that everything I did was an effort’). Each item is rated as to level of severity on a 4 point scale. The instructions are worded, “Below is a list of statements that describe the ways you may have felt. Please indicate how often you have felt this way during the last week.” Each of the 4 statements is assigned a number (from 0 to 3), and the anchors consist of a verbal-chronological label ranging from “rarely or none of the time (less than 1 Day)” to “most or all of the time (5-7 Days).” The CES-D Scale depression score, a sum of the ratings of these items, ranges from 0 to 60.

Psychometric investigations of the CES-D Scale have demonstrated its effectiveness in cross-ethnic studies (Aneshensel & Huba, 1983) and its validity in rural populations (Husaini, Neff, Harrington, Hughes, & Stone, 1980). Comparison with the Hamilton Rating Scale for Depression (Hamilton, 1960) has revealed moderately good convergent validity (Radloff, 1977).

A CES-D Scale score of 16 or more may indicate clinical depression in the general public although the instrument is not generally used for diagnostic purposes. The CES-D has been used as an interview questionnaire and as a self-administered instrument in previous studies (Boyd, Weissman, Thompson, & Myers, 1982). In the current study it was used as an interview questionnaire, similar to the interview procedures of its use by Booker and Hellekson (1992).
Depression in Schizophrenia. Depression in schizophrenia was also assessed with the 9-item Calgary Depression Scale for Schizophrenia (CDSS; Addington, Addington, & Schissel, 1990) which can be found in Appendix E. Especially developed for assessing depression in schizophrenia, the CDSS was designed to address the problem of using depression scales in a population, such as people with schizophrenia, for which they were not designed (Addington, Addington, & Atkinson, 1996). It was originally developed by factor analysis of items from both the Present State Examination (Wing, Cooper, & Sartorius, 1974) and the Hamilton Depression Rating Scale (Hamilton, 1960). A structured interview and rating guide was developed to operationalize the final CDSS instrument items.

This study used the CDSS global depression score, comprised of 8 questions regarding subjective mood and behavior and one question regarding objective observed depression. The 8 subjective items assess the presence and severity of depressive symptoms (depression, hopelessness, self deprecation, guilty ideas of reference, pathological guilt, morning depression, early wakening, and suicide) over the previous 2 weeks. For example, to assess guilty ideas of reference, the interviewer asks, “Do you have the feeling that you are being blamed for something or are even being wrongly accused? (Do not include justifiable blame or accusation. Exclude delusions of guilt.)” Each item is rated as to level on a 4-point scale. Each of the 4 items is designated by a number (from 0 to 3), and each level is anchored by a verbal labels of absent, mild,
moderate, and severe. The global depression score, a sum of these items, ranges from 0 to 27.

For the 9 items of the CDSS, Addington, Addington, Maticka-Tyndale and Joyce (1992) report high internal consistency reliability (\(\alpha = .79\)). The interview and rating scale were found to have good interrater reliability (intraclass correlation coefficient = .89) and validity (Addington et al., 1992). Two findings support the construct and predictive validity of the scale. First, there are significant correlations between CDSS scores and scores on other depression measures (\(r = .82, p < .001; r = .79, p < .001\); Addington et al., 1992). Second, the scale as a whole predicts the presence of a major depressive episode and each scale item contributes to that prediction (Addington et al., 1992). Subsequently the scale was shown to measure depression rather than positive, negative, or extra-pyramidal (side effect) symptoms (Addington, Addington, & Maticka-Tyndale, 1994). Collins, Remington, Coulter, and Birkett (1996) found that the CDSS was far superior to the Hamilton Depression Scale (Hamilton, 1960) in distinguishing between depression and negative symptoms among a sample of individuals with schizophrenia.

**Negative Symptoms.** Negative symptoms were measured using the Negative Symptom Rating Scale (NSRS; Iager, Kirch, & Wyatt, 1985), which can be found in Appendix F. This study used the NSRS summary score which is comprised of items grouped into 4 conceptually related subscales: thought processes (for speech and judgement/decision-making), cognition (for memory, attention and orientation), volition (for grooming, motivation and motion), and affect/relatedness (for emotional response
and expressive relatedness). Each cluster is represented by at least 2 discrete items. Each item is rated as to level of severity on a 6-point scale. Each of the 6 levels of severity is designated by a number (from 0 to -6), and is anchored by a clear description. The global negative symptom score, a sum of these items, ranges from 0 to -60; hence, a low global score would indicate a high level of deficit symptoms. Due to the potential confusion of this negative scoring system, this study used the absolute value of the scores when reporting means and entering scores into correlation and multiple regression formulas.

Grouped into the 4 subscale scores, Iager, Kirch, and Wyatt (1985) found that item scores were highly correlated (.78-.98, p<.001). They also reported that the interrater reliability for total scores was very high (Intraclass correlation coefficient = .96, p<.0001). Construct validity was established by the high correlation (Spearman r = .85, p<.001) between the NSRS and the widely-used Scale for the Assessment of Negative Symptoms (Andreasen, 1983). Discriminant validity was confirmed by the lack of correlation between the NSRS and the Hamilton Rating Scale for Depression (Hamilton, 1960).

In the current study, the NSRS was administered to each participant as a semi-structured interview using the items as a guideline. Collateral information was gathered from case managers, social workers, licensed nurse practitioners, halfway house staff, family members, and patient charts.

Current Alcohol Use. Current alcohol abuse was assessed with the Case Manager Rating Scale for Alcohol Disorder (CMRS; Drake et al., 1990) which can be found in
Appendix G. The CMRS allows case managers to rate the extent of alcohol-related problems among their clients over the past year. Alcohol problems are rated as to level on a 4-point scale, designated by a number from 1 to 5. Each level is anchored by verbal labels of “none, mild, moderate, marked, or severe.” Beside each verbal label is a paragraph describing the corresponding severity of alcohol-related problems. A rating of none=1 indicates no drinking in the past year; mild=2 connotes nonproblematic drinking; moderate=3 indicates problematic drinking and corresponds to DSM-IV alcohol abuse; and severe=4 and extremely severe=5, which connotes a serious drinking problem, correspond to DSM-IV alcohol dependence. Drake, Osher, and Wallach (1989) and Drake and Wallach (1989) found high interrater reliability and concurrent validity.

Because it is based on a case manager’s ongoing examination of patients and routine interactions with their treatment team and family members, this instrument provides a more accurate estimate of alcohol usage than self-report measures. In the current study, the CMRS was completed by each participant’s case manager or social worker. Collateral information was gathered as necessary.

**Instruments, pilot study**

The CDSS, described above, was used in the interrater reliability study.

**Procedure, general study**

**Recruitment.** Thirty participants were recruited from two community mental health centers in Fairbanks and the Alaska Psychiatric Institute, an inpatient setting in
Anchorage. The researcher explained the purpose and procedure of the study at a meeting of the Fairbanks Community Mental Health Center (FCMHC) Clubhouse Executive Committee which is made up of FCMHC consumers and staff. The researcher also presented the study to members of NAMI-Fairbanks and to administrators of the Tanana Chiefs Conference (TCC) outpatient center.

Interview setting and format. Five assessments were administered in a private office located in the outpatient (18 Fairbanks subjects) or inpatient (7 Anchorage subjects) mental health facility or in the assisted living residential center (2 Fairbanks subjects). The researcher used an individual interview format to administer the questionnaires in order to ensure comprehension of all the questions. Interviews and chart reviews provided information for the demographic survey (See Appendix H). Interview sessions did not exceed 50 minutes in length. In most cases, the five assessments were completed in one session, while in other instances two sessions were required. The interviews occurred between November 16, 1998 and February 12, 1999.

Informed consent. Before participating in this study, each volunteer gave written, informed consent (see Appendix A). The researcher explained each section of the consent form verbally, using the written form as a guide to assure that the participant understood all points. In the event that a second day of assessment was necessary, visit two began with a verbal review of important aspects of the informed consent (e.g., voluntary nature, confidentiality, withdrawal with no negative consequences, conflict of interest, procedure). The informed consent procedure took approximately five minutes.
A copy of the signed consent form was kept by the participant and the original was kept with other confidential documents at the interview site.

**Instructions.** The following verbal instructions preceded each visit:

This study will look how people’s moods change with the seasons. For the next 45 minutes, I will be asking you many questions about your moods, thoughts, and activities. I want you to answer them to the best that you can. There are no wrong answers. All of your answers will remain confidential. I really appreciate your volunteering for this interview.

**Honoraria.** To compensate for missed lunch and the cost of transportation, a $15.00 gift certificate to a local grocery store was given to each outpatient participant upon completion of all assessments. If two sessions were required, a $7.50 honorarium was given at the end of each session. Inpatient volunteers received $10 cash.

**Potential health risks.** Because some assessments used contain personal questions about moods, thoughts, and feelings, there was slight possibility that a participant may have become agitated, despondent, or otherwise psychologically injured during the interview session. Although such a scenario did not occur, the investigator took specific precautions to respond to a potential crisis. Had such an event transpired, the researcher planned to immediately contact on-site mental health professionals. The researcher, a community psychology graduate student with therapy and practice training, was also prepared to provide temporary, immediate counseling until staff arrived.
Confidentiality. The three mental health facilities from which participants were recruited (Fairbanks Community Mental Health Center, Tanana Chief's Conference, and Alaska Psychiatric Institute) are committed to the welfare of their consumers. Part of this welfare is each consumer's right to anonymous and confidential care from competent professionals. The researcher and the Community Psychology Program of the University of Alaska Fairbanks used specific safeguards to preserve the anonymity and confidentiality of all research participants. Signed informed consent forms of outpatients were kept with other confidential materials at the FCMHC or TCC. These materials are locked at night and have restricted access during the workday. Inpatient consent forms were kept on-site in a locked cabinet with other confidential materials in the locked, restricted office of a research psychiatrist (J. B.). Collected raw data were coded with 3-digit numbers and kept in a locked cabinet within a secure, locked office in the Psychology Department at the University of Alaska Fairbanks. Names of participants were not kept at the University.

Procedure, pilot study

Recruitment. Seven participants were recruited from NAMI-Fairbanks. The researcher explained the purpose and procedure of the study at a monthly NAMI-Fairbanks meeting made up of mental health consumers and their family members.

Interview setting and format. The CDSS assessment was administered and videotaped in a private room either at the participant's workplace, in the participant's home, or in the private office of the researcher. The researcher used the semi-structured
interview format to administer the questionnaire. Interview sessions did not exceed 20 minutes in length. The interviews were videotaped between October 1, 1998 and November 1, 1998.

Informed consent. Before participating in this study, each volunteer gave written, informed consent (see Appendix B). The researcher explained each section of the consent form verbally, using the written form as a guide to assure that the participant understood all points. The informed consent procedure took approximately two minutes. A copy of the signed consent form was kept by the participant and the original was kept with other confidential documents in a secure, locked office at the University.

Instructions. The following verbal instructions preceded each visit:

As you may recall from the NAMI-Fairbanks meeting, I am doing a study on SAD and schizophrenia. For the next 20 minutes, I will be asking you many questions about your mood. I want you to answer them to the best that you can. There are no wrong answers. Three other graduate students and I will watch this videotape to compare our ratings with one another. The videotape will be destroyed as soon as we have completed the ratings and your answers will remain confidential. I really appreciate your volunteering for this videotaped interview.

Honoraria. No honoraria were given for participation in the pilot study.

Potential health risks. Because the CDSS contains personal questions about moods, thoughts, and feelings, there was slight possibility that a participant may have become agitated, despondent, or otherwise psychologically injured during the interview
session. Had such an event transpired, the researcher, a community psychology graduate student with therapy and practice training, was prepared to provide temporary, immediate counseling.

Confidentiality. The researcher and the Community Psychology Program of the University of Alaska Fairbanks used specific safeguards to preserve the anonymity and confidentiality of all research participants. Signed informed consent forms and raw data were coded with 2-digit numbers and kept in a locked cabinet within a secure, locked office in the Psychology Department at the University of Alaska Fairbanks. Names of participants were not kept at the University.
Results

Pilot Study

To establish interrater reliability with the CDSS, the primary researcher and three other psychology graduate students independently rated video-taped interviews of 7 volunteers with chronic mental illness. The Pearson $r$ correlations for the CDSS total ranged from $r = .93$ to $r = .99$.

General Study

Table 3 (page 46) lists each of the instruments and the respective constructs tapped by each scale. Table 5 summarizes mean scores, standard deviations and coefficients alpha for each scale. Table 6 presents the intercorrelations of the five scales.

Hypothesis (1): The prevalence of seasonal depression among Alaska residents diagnosed with schizophrenia is similar to that among Alaska residents without this diagnosis. (archival data used with permission from Booker & Hellekson, 1992).

Thirty-three percent ($n = 9$) of this sample meets diagnostic criteria for seasonal affective disorder (SAD), 11.1% ($n = 3$) for subsyndromal seasonal affective disorder (S-SAD), and 55.6% ($n = 15$) do not have SAD (No SAD). Rates among the general Fairbanks population are 9.2% ($n = 26$) with SAD, 19.1% ($n = 54$) with S-SAD, and 71.7% ($n = 203$) with No SAD (Booker & Hellekson, 1992). A chi square test for cell counts was used to test for statistical significance of these different prevalence rates. Fairbanks and Anchorage residents diagnosed with schizophrenia (row one) and the general Fairbanks population (row two) were divided into SAD (column one), S-SAD
### Table 5

**Means, Standard Deviations, and Internal Consistency for Five Scales**

<table>
<thead>
<tr>
<th>Scale</th>
<th>Mean score</th>
<th>Standard deviation</th>
<th>Coefficient alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPAQ</td>
<td>8.48</td>
<td>6.04</td>
<td>.82</td>
</tr>
<tr>
<td>CES-D</td>
<td>21.33</td>
<td>12.29</td>
<td>.86</td>
</tr>
<tr>
<td>CDSS</td>
<td>6.0</td>
<td>4.93</td>
<td>.80</td>
</tr>
<tr>
<td>NSRS</td>
<td>13.52</td>
<td>9.38</td>
<td>.87</td>
</tr>
<tr>
<td>CMRS</td>
<td>1.7</td>
<td>0.67</td>
<td>---*</td>
</tr>
</tbody>
</table>

*one item only*
<table>
<thead>
<tr>
<th></th>
<th>SPAQ</th>
<th>CES-D</th>
<th>CDSS</th>
<th>NSRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D</td>
<td>.27</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDSS</td>
<td>.36</td>
<td>.92*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NSRS</td>
<td>.21</td>
<td>.38**</td>
<td>.33</td>
<td></td>
</tr>
<tr>
<td>CMRS</td>
<td>.33</td>
<td>.20</td>
<td>.21</td>
<td>-.13</td>
</tr>
</tbody>
</table>

* p < .005
** p < .05
(column two), and No SAD (column three) as determined by SPAQ seasonality scale index scores. Table 7 displays the significant relationship between schizophrenia status and incidence of SAD, \( \chi^2 (2, 310) = 14.35, p<.001 \). Thirty-three percent of those with schizophrenia and only 9.2% of those without schizophrenia had SAD; significantly more people in the schizophrenia population experienced symptoms of SAD.

**Hypothesis (2):** Alaska residents diagnosed with schizophrenia suffer from higher intensity of depression than do Alaska residents without this diagnosis (archival data granted by Booker & Hellekson, 1992).

As can be seen in Figure 1, the scores on the CES-D of people with schizophrenia averaged 21.33 (SD = 12.29) which is over one standard deviation higher than the scores of those without schizophrenia, which averaged 7.84 (SD = 8.50). Because raw data from the Booker and Hellekson study is not currently available, inferential statistics could not be performed.

**Hypothesis (3):** There is a positive relationship between severity of negative schizophrenia symptoms and seasonal depression among Alaska residents diagnosed with schizophrenia.

To measure the relationship of negative symptoms in schizophrenia to general depression among Alaska residents diagnosed with schizophrenia, each participant’s NSRS summary score was correlated with her or his SPAQ seasonality scale index score. There was a weak, nonsignificant, positive relationship between seasonal depression and negative symptoms, \( r = .21, \text{ns} \).
Table 7

Chi Square Analysis of SAD and S-SAD in Alaskans with Schizophrenia and the General Fairbanks Population

<table>
<thead>
<tr>
<th></th>
<th>SAD</th>
<th>S-SAD</th>
<th>NoSAD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Schizophrenia</strong></td>
<td>9</td>
<td>3</td>
<td>15</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>33.3</td>
<td>11.1</td>
<td>55.6</td>
<td></td>
</tr>
<tr>
<td><strong>General Pop.</strong></td>
<td>26</td>
<td>44</td>
<td>213</td>
<td>283</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>9.2</td>
<td>15.5</td>
<td>75.3</td>
<td></td>
</tr>
</tbody>
</table>
Alaskans with schizophrenia Alaskans without schizophrenia

(Booker and Hellekson, 1992)

Figure 1

CES-D Depression Scale Scores of Far North Residents With and Without Schizophrenia
Hypothesis (4): There is a positive relationship between severity of negative symptoms and intensity of depression among Alaska residents diagnosed with schizophrenia.

To measure the relationship of negative symptoms in schizophrenia to general depression among Alaska residents diagnosed with schizophrenia, each participant's NSRS summary score was correlated with her or his CDSS global depression score. There was a weak, nonsignificant, positive relationship between general depression and negative symptoms, $r = .33$, ns.

Hypothesis (5): There is a positive relationship between alcohol abuse and seasonal depression among Alaska residents diagnosed with schizophrenia.

In order to measure how current (within the past year) alcohol use relates to seasonal depression among Alaska residents diagnosed with schizophrenia, each participant's Case Manager Rating Scale for Alcohol Use (CMRS) score was correlated with her or his SPAQ seasonality scale index score. No significant relationship was found, $r = .20$, ns.

The mean CMRS score ($1.7 \pm 0.67$) described a sample free of current alcohol problems (a score of 2 indicates nonproblematic drinking). The lack of relationship between the CMRS and SPAQ was due to an item floor effect on the CMRS, which possessed a restricted range with which the SPAQ could potentially covary.

Rates of current alcohol abuse were much lower than rates of lifetime problems in this sample. Only 11% ($n=3$) of the participants had current problems with alcohol use.
and these problems were rated as mild. Despite this low rate of current drinking problems, 33.3% (n = 9) of the participants had a DSM-IV diagnosis of Alcohol Abuse or Dependence, most of which were in remission. This disparity between current and lifetime alcohol problems is typical of the schizophrenia population (Bartels, Drake, & Wallach, 1995).

To investigate the relationship of lifetime alcohol problems to risk for seasonal depression in Alaskans with schizophrenia, the researcher conducted a post-hoc comparative analysis. A t-test was used to compare the mean SPAQ scale scores of those with a lifetime history of DSM-IV diagnosis of alcohol abuse or dependence (n = 9) to the mean SPAQ scores of those without lifetime alcohol problems (n = 18). The alcohol abuse/dependence group (M = 12.44, SD = 6.5) scored significantly higher than those without lifetime alcohol problems (M = 6.5, SD = 4.9) on SPAQ scores, t(25) = 2.68, p < .05.

Hypothesis (6): There is a positive relationship between alcohol abuse and intensity of depression among Alaska residents diagnosed with schizophrenia.

In order to measure how current alcohol use relates to seasonal depression among Alaska residents diagnosed with schizophrenia, each participant's Case Manager Rating Scale for Alcohol Use (CMRS) score was correlated with her or his CDSS global depression score. No significant relationship was found, r = .20, ns.

To investigate the relationship of lifetime alcohol abuse or dependence to risk for general depression among people with schizophrenia, the researcher compared CDSS
scores for those participants with a history of DSM-IV diagnosis of alcohol abuse or
dependence with those without alcoholism diagnosis. No difference emerged between
the groups $t(25) = -.08$, ns.

**Hypothesis (7):** Among Alaska residents diagnosed with schizophrenia, there is a
positive correlation between intensity of depression measured by a general depression
instrument and intensity of depression measured by a depression instrument designed for
people with schizophrenia.

A Pearson product moment correlation was used to evaluate the relationship
between CES-D (general depression) Scale scores and CDSS (schizophrenia depression)
global depression scores of Alaska residents diagnosed with schizophrenia. There was a
strong, positive, and statistically significant relationship between these two measures, $r = .92$, $p<.0005$.

**Hypothesis (8):** Among Alaska residents diagnosed with schizophrenia, there is a
positive relationship between seasonal depression and the severity of four dimensions of
negative symptoms: "thought processes," "cognition," "volition/motivation," and
"affect/relatedness."

A stepwise multiple regression was conducted to assess the magnitude of the
relationship between seasonal depression and negative symptoms. The dependent
variable (SPAQ seasonality scale index score) was regressed on 4 independent variables
(NSRS subscale scores: "thought processes," "cognition," "volition/motivation," and
"affect/relatedness"). Because the researcher is interested in exploratory work on
whether any particular dimension of negative symptoms is related to seasonal depression over other dimensions, the independent variables were entered in stepwise fashion. Table 8 provides the results of this analysis. The results of this regression were nonsignificant, and indicate that scores on all the subscales can only predict approximately 2% of the variance in SPAQ seasonality scale scores ($R^2 = .15$, ns).
Table 8

Stepwise Regression of SPAQ Scores NSRS Subscale Scores

<table>
<thead>
<tr>
<th>SPAQ</th>
<th>(DV)</th>
<th>Cog*</th>
<th>Affect</th>
<th>Thgt**</th>
<th>Volition</th>
<th>B</th>
<th>β</th>
<th>T</th>
<th>Sig T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cog*</td>
<td>.35</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.96</td>
<td>.45</td>
<td>1.7</td>
<td>NS</td>
</tr>
<tr>
<td>Affect</td>
<td>.18</td>
<td>.53</td>
<td></td>
<td></td>
<td></td>
<td>.15</td>
<td>.07</td>
<td>.28</td>
<td>NS</td>
</tr>
<tr>
<td>Thgt**</td>
<td>.13</td>
<td>.65</td>
<td>.65</td>
<td></td>
<td></td>
<td>-.36</td>
<td>-.14</td>
<td>-.41</td>
<td>NS</td>
</tr>
<tr>
<td>Volition</td>
<td>.05</td>
<td>.48</td>
<td>.47</td>
<td>.67</td>
<td></td>
<td>-.19</td>
<td>-.11</td>
<td>-.39</td>
<td>NS</td>
</tr>
<tr>
<td>Mean</td>
<td>8.48</td>
<td>3.63</td>
<td>2.48</td>
<td>2.74</td>
<td>4.67</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S.D.</td>
<td>6.04</td>
<td>2.87</td>
<td>2.94</td>
<td>2.28</td>
<td>3.89</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

R² = .15
Adjusted R² = -.009
R = .38

*Cog = Cognition

**Thgt = Thought
Hypothesis (9): Among Alaska residents diagnosed with schizophrenia, there is a positive relationship between intensity of depression and the severity of four dimensions of negative symptoms: "thought processes," "cognition," volition/motivation," and "affect/relatedness."

Stepwise multiple regression was also used to assess the relationship between intensity of depression and negative symptoms. The dependent variable (CDSS global depression score) was regressed on the 4 independent variables (NSRS subscale scores: "thought processes," "cognition," "volition/motivation," and "affect/relatedness"). Stepwise entry of independent variables was also used in order to assess their predictive value. Table 9 provides the results of this analysis. NSRS cognition subscale scores functional as a predictor of CDSS scores (t=2.3, p <.05). However, despite a statistically significant relationship. $R^2$ for the entire regression equation was only .17, ns.
Table 9

Stepwise Regression of CDSS Scores on NSRS Subscale Scores

<table>
<thead>
<tr>
<th>CDSS (DV)</th>
<th>Cog*</th>
<th>Affect</th>
<th>Thgt**</th>
<th>Volition</th>
<th>B</th>
<th>β</th>
<th>T</th>
<th>Sig T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cog*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.73</td>
<td>.42</td>
<td>2.30</td>
<td>.05</td>
</tr>
<tr>
<td>Affect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.05</td>
<td>.72</td>
<td>-.24</td>
<td>NS</td>
</tr>
<tr>
<td>Thgt**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.22</td>
<td>.58</td>
<td>.90</td>
<td>NS</td>
</tr>
<tr>
<td>Volition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.09</td>
<td>.77</td>
<td>-.44</td>
<td>NS</td>
</tr>
</tbody>
</table>

Mean | 21.33 | 3.63 | 2.48 | 2.74 | 4.67 |
S.D. | 12.29 | 2.87 | 2.94 | 2.28 | 3.89 |

R² = .17
Adjusted R² = .14
R = .42

*Cog = Cognition

**Thgt = Thought
Discussion

The main finding of this study was a high prevalence of SAD among people with schizophrenia living in the extreme North. The prevalence rate was three times higher than that found in the general population in Alaska, which itself is higher than found in the lower 48 States (Booker & Hellekson, 1992). Another important discovery is that Alaskans with schizophrenia have a high rate of unipolar depression. An additional important finding is that severity of SAD symptoms was higher among participants with a lifetime history of DSM-IV diagnosis of alcohol abuse or dependence. Noteworthy too is the fact that negative symptoms did not correlate significantly with either seasonal depression or general depression as measured by the instruments used in this study.

Schizophrenia vs. non-schizophrenia seasonal depression

The hypothesis that the prevalence of SAD among Alaska residents diagnosed with schizophrenia is similar to that of Fairbanks residents without this diagnosis was not supported. SAD is far more common among high latitude residents with schizophrenia than it is among those without. Because of this difference in prevalence between groups, these rates cannot be explained by latitude alone. Schizophrenia and SAD may be comorbid processes; alternatively, they may share certain biologic underpinnings. For example, disorder in serotonin transmission is implicated in both SAD (Rosenthal, Sack, et al., 1984; Rosenthal & Wehr, 1992) and schizophrenia (Ames et al., 1996).

The proportion of participants in the current sample who meet diagnostic criteria for SAD was much larger than found by Booker and Hellekson (1992) and others (Kasper...
et al., 1989b; Rosenthal, 1993) in studies in the general population. Previous research indicates that rates of SAD increase as degrees of latitude increase (Rosen et al., 1990; Potkin et al., 1986), so the present results are consistent with this trend. The high proportion of this sample with SAD, however, exceeded previous high latitude nonclinical samples. This indicates that people with schizophrenia who live in high latitudes are at high risk for developing SAD, and at higher risk than the general population living at high latitudes.

**Schizophrenia vs. non-schizophrenia depression**

Because raw data from the Booker & Hellekson (1992) study was not available, tests of this hypothesis using inferential statistics were not possible. The trend of the results appears to support the hypothesis that Alaska residents with schizophrenia suffer from higher intensity of depression than do those without schizophrenia. CES-D scores were substantially higher for the schizophrenia group. Two explanations for this trend are possible. First, previous research indicates that depression is more prevalent in people with schizophrenia than in the general population (Siris, 1991; Harrow et al., 1994; Bernard et al., 1998; Tollefson et al., 1998). The current data support these previous findings. Alternatively, it is possible that the present sample scored higher than the nonclinical sample in the Booker and Hellekson study because the CES-D was designed for use in the general population and does not take into account the overlap of schizophrenia symptoms. For example, a participant might have rated themselves as
strongly bothered by the CES-D items "I felt fearful" and "I felt that people disliked me" due to persecutory delusions rather than due to symptoms of depression.

Mindful of the limitations of the CES-D, the current data nonetheless suggests participants are experiencing a significant number of symptoms of depression. At some point in their illness, most people with schizophrenia will suffer from serious depression. Thirty-seven percent will attempt suicide (Palmer, Henter, & Wyatt, 1999) and 9-13% will succeed (Meltzer, 1999). The depression may be due either to the disease process of schizophrenia itself, the person’s reaction to the severity of the illness, or to medication side effects (Torrey, 1995). Anti-depressant, psychosocial, and cognitive-behavioral therapies have been successfully used to treat depression in people with schizophrenia (Torrey, 1995). Alaskans with schizophrenia need close monitoring and aggressive treatment in order to decrease their risk of depression and suicide.

Several psychosocial factors such as the loss, stigma, isolation and poverty endemic to this group of people also contribute to comorbid depression. One type of loss occurs from the time of initial psychotic break. A person with schizophrenia learns they may have these symptoms and need to take medication for the rest of her or his life. The person learns that the hopes and ambitions she or he had in college of a marriage, family and career will probably not be realized. Another type of loss for people with schizophrenia is that many close friends who share these unrelenting symptoms will commit suicide. The stigma of the schizophrenia diagnosis adds to depression as well. Many are denied basic employment privileges due to their diagnosis. The fact that many
of these individuals are cast into the margins of society leads to severe feelings of isolation. Most of the people I interviewed have very few friends and little family support. Indeed most of the participants in this study were quite poor. The outpatient volunteers generally shared their streets with gangs, drugs, and violence. These factors, associated with poverty, also function as risk factors contributing to the great amount of depressive symptoms found in this sample. These psychosocial elements need to be considered when interpreting these results and designing treatment plans to address depression.

**Relationship between seasonal depression and negative symptoms**

The hypothesis that there is a positive relationship between severity of negative schizophrenia symptoms and seasonal depression among Alaskans with schizophrenia was not supported. The correlation, though positive, was weak and nonsignificant. Symptoms of seasonal depression did not overlap with negative symptoms of schizophrenia as measured by the NSRS. Because SAD symptoms such as fluctuations in energy level, mood, and social activity resemble negative symptoms, a stronger relationship was anticipated.

These data suggest that negative schizophrenia symptoms and seasonal depression are two separate diagnostic entities. These results refute the hypothesis that negative symptoms such as withdrawal and anhedonia would keep people indoors, depriving them of sunlight, leading to SAD.
Relationship between general depression and negative symptoms

The hypothesis that there is a positive relationship between severity of negative symptoms and intensity of depression among Alaska residents with schizophrenia was not supported. The relationship, though positive, was weak and nonsignificant. This analysis indicates that negative symptoms and depression are distinct constructs in this sample, as evidenced by the low level of shared variance. Thus, negative symptoms experienced by participants in this sample are not secondary to depression. These findings lend support to the literature that distinguishes depressive from negative symptoms (Andreasen, 1997b; Carpenter, Buchanan, & Kirkpatrick, 1991; Moller, 1995; Kay & Sevy, 1990). These results must be interpreted with caution due to the small sample size and to the fact that the NSRS does not assess two important additional dimensions of negative symptoms: anhedonia and withdrawal.

Relationship between seasonal depression and alcohol use

The hypothesis that there is a positive relationship between alcohol abuse and seasonal depression among Alaskans with schizophrenia was not supported. However, only three participants were reported by their case managers to have had alcohol-related problems during the past year. Interviews were conducted during the winter months on the assumption that alcohol abuse would increase during this period; however, such a pattern did not emerge in this group of patients.

This is a very unusual sample in this regard. Estimates of comorbid alcohol abuse are quite high in most community samples of people with schizophrenia, ranging from
31% to 47% (Regier et al., 1990; Selzer & Lieberman, 1993; Mueser et al. 1990; Drake et al., 1990; Ziedonis & Fisher, 1994). The lack of a relationship between SAD and current alcohol use in this data is an artifact of restricted range in the CMRS. Only 3 of 27 members of this sample currently abuse alcohol. Hence, no relationship between CMRS scores and SPAQ scores could be found, as there was minimal variance in the CMRS scores. Such a relationship may exist in samples with drinking problems.

Current alcohol use was not characteristic of this sample. Thus, a strong correlation between current alcohol problems and seasonal depression was not found.

One of the most important findings of this study is that seasonal depression was far more common among those with DSM-IV diagnosis of alcohol abuse or dependence (either in remission or current). This indicates that patients with schizophrenia and a history of alcohol problems may be particularly vulnerable to SAD despite a lack of current alcohol problems.

One explanation for the comorbidity of lifetime alcohol problems and SAD is that they may share a biologic or genetic mechanism. That mechanism may predispose a person with schizophrenia to both long-term alcohol-related problems and SAD. Another explanation might relate to the use of alcohol as an ineffective coping strategy for seasonal depression and/or alcohol abuse exacerbating and potentiating seasonal depression. Because the relationship of alcohol use and SAD among people with schizophrenia has heretofore not been investigated, definitive conclusions cannot be drawn.
Relationship between depression and alcohol use

The hypothesis that there is a positive relationship between current alcohol abuse and intensity of depression among Alaskans with schizophrenia was not supported. The correlation was not significant. These results indicate that current alcohol abuse is not a significant predictor of depression. Estimates of comorbid substance abuse in schizophrenia are high (Ziedonis & Fisher, 1994). Because members of the sample have uncharacteristically few current alcohol-related problems, restricted range in the CMRS afforded limited variance to covary with levels of depression on the CES-D. Also, no considerable difference in depression was found between those with and without a history of drinking problems. Thus, the current data suggest that depression and lifetime drinking problems are not related in Alaskans with schizophrenia, but further research with larger samples more representative of the population is needed.

Relationship between two depression measures

As hypothesized, there was a positive relationship between the two measures of intensity of depression. In fact, this relationship accounted for virtually all of the reliable variance in the measures. The first depression measure, the CES-D, was conceived to detect depression in the general population. The second measure, the CDSS, was designed to assess depression in people with schizophrenia. The high correlation between CES-D and CDSS scores supports the conclusion that the two scales measure the same construct.
The relationship strength exceeds prior reports of the equivalency between the CDSS and other depression measures (Addington et al., 1992). However, although scores were highly intercorrelated, the respective diagnostic thresholds of each instrument conveyed greatly different rates of depression.

Although the CES-D is a screening instrument, and is generally not used to make a diagnosis of depression, scores higher than 15 may imply clinical depression (Radloff, 1977). By this standard, 70.4% (n=19) of those interviewed may have clinical depression. Using the CDSS as a diagnostic tool, 33% (n=11; scores greater than 6 indicate depression) of the current sample suffer from depression. These divergent results suggest that the cut-offs on the CDSS are a more stringent measure of depression and may be more appropriate for the population of people with schizophrenia. Further research is needed comparing the diagnostic sensitivity and specificity of these instruments in people with schizophrenia, using structured diagnostic interviews. The highly related scores coupled with the divergent diagnosis tallies suggests that the two scales measure the same construct, namely depression, but the CDSS uses a more conservative diagnostic threshold.

**Relationship between seasonal depression and negative symptoms**

The hypothesis that there is a positive relationship between seasonal depression and selected dimensions of negative symptoms was not supported. The four negative symptom components were found to have very little relation to symptoms of seasonal depression. The negative symptoms analyzed were alogia (poverty of thought and
speech), narrowing of ideation (loss of the ability to concentrate or focus attention), avolition (inability to formulate and execute plans), and flat or blunted affect (lack of emotional expression). None of these components were associated with seasonal depression. The weakness of the relationship reveals that negative schizophrenia and seasonal depression are two separate syndromes and even discrete dimensions of the negative symptom construct are unrelated to depression. However, further research on the relation of negative symptoms and depression is needed inferences are provisional.

**Relationship between depression and negative symptoms**

The hypothesis that there is a positive relationship between intensity of depression and the severity of negative symptoms was not supported. Neither alogia, narrowing of ideation, avolition, nor flat or blunted affect was related to depressive symptoms. Thus, these negative symptoms are distinct from depression. Previous research indicates that negative symptoms, though often confused with depressive symptoms, are nonetheless a separate psychological construct (Addington, Addington, & Maticka-Tyndale, 1994). The present results are consistent with these previous findings. These data lend further support to the ability of the CDSS to distinguish depressive from negative symptoms (Collins, Remington, Coulter, & Birkett, 1996). There was a modest relationship between depression and the negative symptom “cognition,” which only accounted for a small proportion of the variance.
Limitations

Several caveats need to be made regarding limitations of these results. First, the number of research participants was small, limiting generalizability of the results. Second, very few of the participants had current drinking problems. Thus the relationship of current alcohol abuse with depression and SAD could not be adequately tested. In addition, this suggests one important difference between this sample and the schizophrenia community at large, given the high rate of comorbid alcohol problems found in most epidemiological studies of schizophrenia in the community. Third, a one-frame analysis such as this cannot accurately detect the fluctuations of negative symptoms over time. Fourth, the negative symptom scale used, the NSRS, does not assess two important components of negative symptoms: anhedonia (loss of the ability to feel pleasure) and social withdrawal. Including the assessment of these two negative symptoms might have yielded different results.

Clinical Implications

These results call attention to a high level of SAD and problematic seasonal mood variations among patients with schizophrenia in the extreme North, and by extension, Northern latitudes. A mid-winter day in Fairbanks or Anchorage, including civil twilight, has a similar amount of daylight to that of many large Northern cities such as Vancouver, B.C., Seattle, and Tacoma, Washington, Minneapolis, Minnesota, and Boston, Massachusetts. Residents of these cities who have schizophrenia may be at high risk for developing SAD as well. One third of northerners with schizophrenia may benefit from
the addition of phototherapy, outdoor daytime exercise programs, season-targeted pharmacotherapy and/or counseling and education for SAD to their antipsychotic and behavioral treatments.

The current results suggest depression needs more clinical attention among northerners with schizophrenia. People with both schizophrenia and a DSM-IV diagnosis of alcohol abuse or dependence, in remission or not, are at particular risk for developing SAD. Therefore, dual diagnosis patients, their psychiatrists, case managers, and family members need to closely monitor seasonal variations in mood, appetite, energy, social activity, and sleep.

According to Palmer, Henter, & Wyatt, (1999), there is a pervasive notion in the psychiatric community that depression and schizophrenia are seldom associated, except in the case of depression as a manifestation of schizophrenia. They contend that the possibility that depression comprises a comorbid mood disorder is popularly rejected. A remarkably high percentage of the current participants suffer from depressed mood along with their schizophrenia symptoms. Mood symptoms are frequently dismissed as an inevitable, untreatable result of schizophrenia; thus, they do not receive the rehabilitative attention they warrant. These results indicate that northerners with schizophrenia have depression that must be treated too. Research shows that many people with schizophrenia patients benefit from adjunct anti-depressant therapy (Palmer, Henter, & Wyatt, 1999).
Directions for Future Research

Future research is needed to investigate the prevalence of SAD among lower latitude dwellers with schizophrenia as well; it is likely that those with schizophrenia who live in less extreme environments are also at risk for suffering symptoms of seasonal depression. A longitudinal study is needed to detect seasonal oscillations in negative symptoms, positive symptoms, and general depression in schizophrenia. Winter depression is characterized be hypersomnia, overeating, and carbohydrate craving (Rosen et al., 1990): these are three common side effects of antipsychotic medications. The participants in this study reported an increase in these symptoms during the winter months. Future research should evaluate these symptoms throughout the year in terms of their relation to the type of antipsychotic and dosage used. Investigations are also needed to determine whether people with schizophrenia have disorders in light sensitivity. Seasonal patterns of alcohol use among northern latitude residents, with and without schizophrenia, likewise need closer examination. The relationship between seasonal depression and alcohol use needs to be examined in samples with more typical, higher rates of current drinking problems. Also needed is a comparison of the diagnostic sensitivity and specificity of the CES-D and CDSS in people with schizophrenia, using structured diagnostic interviews. The current findings also suggest a closer look is warranted for the seasonal exhibition of features such as anhedonia, social withdrawal, depressed mood, and variations in sleep patterns and appetite among people with schizophrenia. Finally, further research comparing depression rates among people with
schizophrenia living in extreme northern regions and lower latitudes is required to ascertain the influence of latitude on risk of comorbid depression.

**Conclusion**

SAD is more common in northern latitudes. The current study suggests SAD is especially high among northern residents diagnosed with schizophrenia. In 80% of SAD patients, these depressive symptoms are at least temporarily relieved by phototherapy (Rosenthal, Genhart, Jacobsen, Skwerer, & Wehr, 1987). This is a viable treatment option for Alaska residents with schizophrenia. Northern latitude dwellers with schizophrenia need access to and instruction in the use of light boxes, the phototherapy device. Such boxes could be kept in halfway houses, day treatment facilities, or community mental health centers. Routine assessment and diagnosis for SAD and S-SAD would direct attention to these debilitating depressive features for which several empirically validated interventions exist. This has the potential to improve the quality of life of many people with schizophrenia who suffer from comorbid seasonal depression.
Appendix A

UAF Institutional Review Board consent form for the general study

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**UAF INSTITUTIONAL REVIEW BOARD CONSENT FORM**  
**IRB Log# ____________**

**Proposer:** Julie Doorack, B.A.  
**Signature:** _________________________  
**Date:** __________

**Proposal Title:** Seasonal Affective Disorder Comorbidity with Schizophrenia in the Extreme North

---

I am being invited to participate in a research study. This form is designed to provide me with information about this study. The principal investigator (researcher) will describe this study to me and answer any of my questions. If I have any questions or complaints about the informed consent process or the research study, I should contact the Institutional Review Board (IRB), the committee that protects human subjects, at (907) 474-7314 or Dr. Charles Geist, IRB Chair, at (907) 474-7792.

1. **Name of Subject**

2. **Title of Research Study**

   Seasonal Affective Disorder Comorbidity with Schizophrenia in the Extreme North

3. **Principal Investigator and Telephone Number**

   Julie Doorack, B.A., graduate student; (907) 474-7007  
   James Allen, Ph.D., advisor; (907) 474-6132

4. **The Purpose of the Research**

   The principal investigator (researcher) is conducting this research in order to fulfill a thesis requirement for the Master of Arts in Community Psychology program. The overall goal of this study is to discover if people diagnosed with schizophrenia who live in the Extreme North (Fairbanks, or Anchorage, Alaska) suffer from seasonal affective
disorder (the “Winter Blues”). Another purpose of this study is to look at the relationship between depression, alcohol use, and certain symptoms of schizophrenia.

5. Procedures for This Research

First, the study will be clearly explained to me and I will decide if I want to participate. If so, I will sign this “informed consent” form and the principal investigator (PI) will interview me once or twice. These interviews will take place in a private office located on the second floor of the Northern Door Clubhouse at Fairbanks Community Mental Health Center (FCMHC). During these meetings, the PI and I will fill out five (5) questionnaires. The questions are personal ones about my moods, thoughts, feelings, and alcohol use. The meetings will not take longer than fifty (50) minutes each. I will be interviewed on one or two afternoons between November 15, 1998 and February 15, 1999.

I am one of thirty participants in this study.

My questionnaires will be kept in a locked filing cabinet inside of a secure, locked office in the Psychology department at the University of Alaska Fairbanks. My name will not appear on them. Instead, they will be coded with a 3-digit number. This signed consent form will kept with other confidential materials at the FCMHC main center. If I desire, I may keep a copy of this form.

6. Potential Health Risks or Discomforts

Because the principal investigator (PI) will ask me personal questions about moods, thoughts, and feelings, there is a slight possibility that I may become upset during the interview session. In such a case, the PI will immediately contact FCMHC staff. Interview sessions will be scheduled during the working day while at least three Clubhouse staff (mental health professionals) are on the premises. In the unlikely event that FCMHC staff cannot provide immediate assistance, the PI (a community psychology graduate student with 20 hours of Ph.D.-supervised counseling training) will provide temporary, immediate counseling until FCMHC staff arrive.

7. Potential Health Benefits to You or to Others

No foreseeable health benefits are associated with participation in this study.
8. Potential Financial Risks

No foreseeable financial risks are associated with participation in this study.

9. Potential Financial Benefits to You or to Others

Because I will spend one or two afternoons participating in this study, I will be compensated for lunch and transportation. Instead of cash, I will receive a gift certificate or voucher not to exceed $15.00 in value.

10. Conflict of Interest

I understand that the principal investigator of this study is also a relief staff worker at Fairbanks Community Mental Health Center (FCMHC). I understand that if I don’t participate in this study, I will still get all FCMHC services to which I am entitled. I understand that if I decide to withdraw from the study at any time, I will still receive services from FCMHC as usual. I also understand that anything I tell the principal investigator is private, strictly confidential, and will not be repeated to other staff or clients of FCMHC.

11. Alternatives to Participating in this Research Study

I am free not to participate in this study. If I choose to participate, I am free to withdraw my consent and discontinue participation in this research study at any time without this decision affecting my medical care. If I have any questions regarding my rights as a subject, I may phone the Institutional Review Board (IRB) office at (907) 474-7314 or Dr. Charles Geist, IRB Chair, at (907) 474-7792.

12. Withdrawal From this Research Study

If I wish to stop my participation in this research study for any reason, I should contact Julie Doorack at (907) 474-7007. I may also contact the Institutional Review Board (IRB) Office at (907) 474-7314 or Dr. Charles Geist, IRB Chair, at (907) 474-7792.
13. Confidentiality

The University of Alaska Fairbanks will protect the confidentiality of my records to the extent provided by Law. I understand that the Institutional Review Board has the legal right to review my records.

The principal investigator (PI) and the Community Psychology graduate school will make special efforts to keep everything I say confidential. The PI will not repeat what I say to other people. My questionnaires will not have my name on them. They will be coded with a 3-digit number and kept in a locked cabinet within a secure, locked office in the Psychology department at the University of Alaska Fairbanks. This signed consent form will kept with other confidential materials at the FCMHC main center.
15. Signatures

Subject’s Name

The Principal or Co-Principal Investigator or representative has explained the nature and purpose of the above-described procedure and the benefits and risks that are involved in this research protocol.

Signature of Principal or Co-Principal Investigator or representative obtaining consent

Date

I have been informed of the above-described procedure with its possible benefits and risks and I have received a copy of this description. I have given permission for my participation in this study.

Signature of Subject or Representative

Date

If you are not the subject, please print your name and indicate one of the following:

- The subject’s parent
- The subject’s guardian
- A surrogate
- A durable power of attorney
- A proxy
- Other, please explain:

Signature of Witness

Date

If a representative signs and if appropriate, the subject of this research should indicate assent by signing below.

Subject’s signature

Date
Appendix B

UAF Institutional Review Board consent form for the pilot study

**UAF INSTITUTIONAL REVIEW BOARD CONSENT FORM**  
**IRB Log# __________________**

**Proposer:** Julie Doorack, B.A.  
**Signature:** __________  
**Date:** _____

**Proposal Title:** Interrater Reliability on Calgary Depression Scale

---

I am being invited to participate in a videotaped, structured interview with Julie Doorack, a graduate student in Community Psychology at UAF. This form is designed to provide me with information about this interview. A Community Psychology graduate student will describe this study to me and answer any of my questions. If I have any questions or complaints about the informed consent process or the interview, I should contact Dr. Charles Geist, Institutional Review Board Chair, at (907) 474-7792.

---

1. **Name of Participant**

2. **Title of Research Study:** Interrater Reliability on Calgary Depression Scale (A scale proposed for use in the thesis proposal entitled “Seasonal Affective Disorder Comorbidity with Schizophrenia in the Extreme North.”)

3. **Principal Investigator and Telephone Number:** Julie Doorack, B.A., graduate student; (907) 474-7007  
James Allen, Ph.D., advisor; (907) 474-6132

4. **Purpose:** The purpose of this interview is to enable three Community Psychology graduate students to establish interrater reliability on a nine-item assessment for depression.

5. **Procedures:** The interviewer will ask me questions about my mood in a structured interview format. The interview will last approximately 30 minutes. The interview will be videotaped and viewed separately by two other graduate students. I am one of 15 participants in this procedure.

6. **Risks:** No foreseeable health risks are associated with participation in this interview.

7. **Benefits:** A potential benefit of participating in this interview is gaining a better understanding of the research and structured interview processes.
8. **Withdrawal:** I understand that my participation in this interview is voluntary and I may discontinue at any time.

9. **Confidentiality:** The confidentiality of my responses will be protected to the extent provided by Law. My name will not appear on any rating forms. Instead, the forms will be coded with a 2-digit number. The videotape and the rating forms will be destroyed after interrater reliability is established.

10. **Signatures:**
The graduate student has explained the nature and purpose of the above-described procedure and the benefits and risks that are involved in this procedure.

<table>
<thead>
<tr>
<th>Signature of Graduate Student obtaining consent</th>
<th>Date</th>
</tr>
</thead>
</table>

I have been informed of the above-described procedure with its possible benefits and risks. I have given permission for my participation in this study.

<table>
<thead>
<tr>
<th>Signature of Participant</th>
<th>Date</th>
</tr>
</thead>
</table>
Appendix C

Seasonality Scale Index (Modified from the Seasonal Pattern Assessment Questionnaire—SPAQ, of Rosenthal, Brandt, and Wehr, 1984a)

To what degree do the following change with the seasons?

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no change</td>
<td>slight change</td>
<td>moderate change</td>
<td>marked change</td>
<td>extremely marked change</td>
<td></td>
</tr>
<tr>
<td>Sleep length</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social activity</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
</tr>
<tr>
<td>Mood (overall feeling of well being)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
</tr>
<tr>
<td>Appetite</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
</tr>
<tr>
<td>Energy level</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
</tr>
</tbody>
</table>

*With the seasons*
Appendix D

Center for Epidemiologic Studies Depression Scale (Radloff, 1977)

BELOW IS A LIST OF STATEMENTS THAT DESCRIBE THE WAYS YOU MAY HAVE FELT. Please indicate how often you have felt this way during the last week: (0) rarely or none of the time; (1) some or a little of the time; (2) occasionally or a moderate amount of time; or (3) most or all of the time.

1. I was bothered by things that usually don’t bother me.
2. I did not like eating; my appetite was poor.
3. I felt that I could not shake off the blues even with help from my family and friends.
4. I felt that I was just as good as other people.
5. I had trouble keeping my mind on what I was doing.
6. I felt depressed.
7. I felt that everything I did was an effort.
8. I felt hopeful about the future.
9. I thought my life had been a failure.
10. I felt fearful.
11. My sleep was restless.
12. I was happy.
13. I talked less than usual.
15. People were unfriendly.
16. I enjoyed life.
17. I had crying spells.
18. I felt sad.
19. I felt that people disliked me.
20. I could not get “going.”
Appendix E

Calgary Depression Scale for Schizophrenia (Addington, Addington, & Schissel, 1990)

Interviewer: Ask the first question as written. Use follow up probes or qualifiers at your discretion. Time frame refers to last 2 weeks unless stipulated. N.B. The last item, #9, is based on observations of the entire interview.

(1) Depression: How would you describe your mood over the last 2 weeks: Do you keep reasonably cheerful or have you been very depressed or low spirited recently?
   In the last 2 weeks about how often have you (own words) every day? All day?
   0. Absent
   1. Mild - Expresses some sadness or discouragement on questioning.
   2. Moderate - Distinct depressed mood persisting up to half the time over the last 2 weeks; present daily.
   3. Severe - Markedly depresses mood persisting daily over half the time interfering with normal motor and social functioning.

(2) Hopelessness: How do you see the future for yourself?
   Can you see any future-or has life seemed quite hopeless?
   Have you given up or does there still seem some reason for trying?
   0. Absent
   1. Mild - Has at times felt hopeless over the last week but still has some degree of hope for the future.
   2. Moderate - Persistent, moderate sense of hopelessness over last week. Can be persuaded to acknowledge possibility of things getting better.
   3. Severe - Persisting and distressing sense of hopelessness.

(3) Self deprecation: What is your opinion of yourself compared to other people?
   Do you feel better or not as good or about the same as most?
   Do you feel inferior or even worthless?
   A. Absent
   B. Mild – Some inferiority; not amounting to feeling of worthlessness.
   C. Moderate – Subject feels worthless, but less than 50% of the time.
   D. Severe – Subject feels worthless more than 50% of the time. May be challenged to acknowledge otherwise.

(4) Guilty ideas of reference: Do you have the feeling that you are being blamed for something or even wrongly accused? What about? (Do not include justifiable blame or accusation. Exclude delusions of guilt.)
   0. Absent
   1. Mild – Subject feels blamed but not accused less than 50% of the time.
   2. Moderate – Persisting sense of being blamed, and/or occasional sense of being accused.
   3. Severe – Persistent sense of being accused. When challenged acknowledges that it is not so.
(5) **Pathological guilt:** Do you tend to blame yourself for little things you may have done in the past?

Do you think you deserve to be so concerned about this?

0. Absent
1. Mild – Subject sometimes feels over guilty about some minor peccadillo, but less than 50% of the time.
2. Moderate – Subject usually (over 50% of the time) feels guilty about past actions the significance of which he exaggerates.
3. Severe – Subject usually feels s/he is to blame for everything that has gone wrong, even when not his/her fault.

(6) **Morning Depression:** When you have felt depressed over the last two weeks have you noticed the depression being worse at any particular time of the day?

0. Absent – No depression.
1. Mild – Depression present but no diurnal variation.
2. Moderate – Depression spontaneously mentioned to be worse in the a.m.
3. Severe – Depression markedly worse in the a.m., with impaired functioning which improves in the p.m.

(7) **Early wakening:** Do you wake earlier in the morning than is normal for you?

How many times a week does this happen?

0. Absent – No early wakening.
1. Mild – Occasionally wakes early (up to twice weekly) 1 h or more before normal time to wake or alarm time.
2. Moderate – Often wakes up early (up to five times weekly) 1 h or more before normal time to wake or alarm.
3. Severe – Daily wakes 1 h or more before normal time.

(8) **Suicide:** Have you felt that life wasn’t worth living?

Did you ever feel like ending it all?

What did you think you might do?

Did you actually try?

0. Absent
1. Mild – Frequent thoughts of being better off dead, or occasional thoughts of suicide.
2. Moderate – Deliberately considered suicide with a plan, but made no attempt.
3. Severe – Suicidal attempt apparently designed to end in death (i.e., accidental discovery or inefficient means).

(9) **Observed depression**

*Based on interviewer’s observations during the entire interview.*

The question ‘Do you feel like crying?’ used at appropriate points in the interview may elicit information useful to this observation.

0. Absent
1. Mild – Subject appears sad and mournful even during parts of the interview involving affectively neutral discussion.
2. Moderate – Subject appears sad and mournful throughout the interview, with gloomy monotonous voice and is tearful or close to tears at times.
3. Severe – Subject chokes on distressing topics, frequently sighs deeply or cries openly, or is in a persistent state of frozen misery.
**Appendix F**

**Negative Symptom Rating Scale**

<table>
<thead>
<tr>
<th>Not Rated (NR)</th>
<th>Normal (0)</th>
<th>Mildly Impaired (-1-2)</th>
<th>Moderately Impaired (-3-4)</th>
<th>Severely Impaired (-5-6)</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Speech content</td>
<td>More than 5 ideas, elaborated</td>
<td>4-5 ideas</td>
<td>2-3 statements</td>
<td>1 statement or mute or nonsense</td>
<td></td>
</tr>
<tr>
<td>II. Judgement/decision&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Good grasp of reality, independent decisions</td>
<td>Slow, incomplete judgement</td>
<td>Vague judgement</td>
<td>Uncertainty, ambivalence</td>
<td></td>
</tr>
<tr>
<td>III. Memory—Correct recall after 10 minutes&lt;sup&gt;2&lt;/sup&gt;</td>
<td>5 words</td>
<td>4 words, or five with prompting</td>
<td>2-3 words</td>
<td>No words or 1 word with prompting</td>
<td></td>
</tr>
<tr>
<td>IV. Attention—Correct subtraction on 30 seconds serial 3's&lt;sup&gt;3&lt;/sup&gt;</td>
<td>&gt; 10</td>
<td>5-10</td>
<td>2-4</td>
<td>0-1</td>
<td></td>
</tr>
<tr>
<td>V. Orientation</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>VI. Grooming—Clothing, face, hands</td>
<td>Clean, appropriate</td>
<td>Clean, untidy</td>
<td>Stained, disheveled</td>
<td>Soiled, uncared for</td>
<td></td>
</tr>
<tr>
<td>VII. Motivation—Plans, constructive activity</td>
<td>Without needs for supervision</td>
<td>Needs reminders</td>
<td>Needs ongoing supervision</td>
<td>Needs active assistance</td>
<td></td>
</tr>
<tr>
<td>VIII. Motion—Amount, speed (task, make 5 steps back and forth&lt;sup&gt;4&lt;/sup&gt;)</td>
<td>Complete task without hesitation</td>
<td>Lag/mild slowing</td>
<td>Sluggish/needs encouragement</td>
<td>May only stand up or remains motionless</td>
<td></td>
</tr>
<tr>
<td>IX. Emotional response</td>
<td>Full affect range</td>
<td>Mildly restricted range of affect</td>
<td>Blunted affect</td>
<td>Flat affect</td>
<td></td>
</tr>
<tr>
<td>X. Expressive relatedness</td>
<td>Freely initiates</td>
<td>Does not initiate but readily engages</td>
<td>Avoids interaction, no warmth</td>
<td>Withdrawn, seclusive, mechanical questions</td>
<td></td>
</tr>
</tbody>
</table>

**TOTAL**

---

<sup>1</sup> Should you receive cash as a gift, what would you do with the money? What are your current needs?

<sup>2</sup> Explain procedure. Present, only once, 5 words 1-second intervals; immediately ask the patient to repeat them. Ask the patient to remember those words for 10 minutes, at which time the patient is asked to recall the words: ball, orange, cigarette, snow machine, sky, piano, television, key, clock, apple.

<sup>3</sup> “Subtract “3” out loud serially, starting from 100 for 30 seconds.” Instruct patient that only the number of correct subtractions counts and there will be no penalty for wrong answers. Tell the patient that the procedure is to be timed and the rater will notify the patient when the timing starts.

<sup>4</sup> “Please stand up, make 5 steps, turn around, and return to the chair.”
The scoring principles are as follows: (1) the range of the scale lies between “0” and “-6” for each item. A score of “0” represents the average individual’s healthy, adaptive exercise of occupational functioning, social relations, self-care, constructive use of time, and efficient mental performance. The normal behavior range declines to a score of “-2” and may overlap with soft signs of pathology, which means that even normal controls may score as mildly impaired on negative symptoms. A score of “-6” in most cases represents the complete lack of the assessed function and is a strong indication of severe and pervasive pathology. A rating of NR (not ratable) is used when the rater cannot get enough information to rate that item confidently. This is different from a rating of “0,” which means that the rater possesses enough information to describe the behavior defined under that point on the scale. (2) Although there might not be a perfectly linear gradient in the way the points of the scale are being defined from “0” to “-6,” should the rater hesitate between two neighboring scores, the one that stands closer to “0” is to be marked. (3) As a rule, the ratings represent the best of the patient’s functioning during a rating period. (4) A rating period is the time span within which the raters collect the data necessary for completing one assessment. (5) Most of the data will be derived directly, during the rater’s interview with the patient. On those items for which the patient does not provide sufficient information, the staff will be asked to provide a patient’s cross-sectional behavioral profile at the time the assessment is being completed. Each rating should not be influenced by rater’s knowledge of the patient’s performance during the previous ratings.

(A) Thought Processes:

I. **Speech content**: Evaluates the amount of coherent, verbally expressed thoughts or personal ideas, regardless of their basis in fact. An absence of expressed thoughts may call for prompting on the rater’s part.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient’s speech contains many ideas (e.g., &gt;5) which sound clear, have meaning, and are readily volunteered.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient can coherently voice several personal, distinct ideas (4-5) but keeps quiet most of the time and seldom initiates discussion of them.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient expresses only 2-3 personal statements and is usually speechless, even when urged to talk.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient is either mute or the speech is so disorganized that one cannot clearly recognize a single thought.</td>
</tr>
</tbody>
</table>

II. **Judgment/decision**: Assesses the patient’s capacity to use personal judgment and to make decisions within the ordinary requirements of daily living. It assumed that the rater engages in a conversation with the patient, although when that is not possible the patient’s attitudes/actions are to be rated. For expedited rating, judgement questions may be offered to the patient (i.e., should you receive cash as a gift, what would you do with the money? What are your current needs?)

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient’s judgment sounds logical, clear, and sensible. Decisions are made independently, efficiently, and in keeping with the patient’s needs.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient’s judgment is marginal, leaves a lot of loose ends, and may require mild supervision with more complex issues. Decision-making may stall to a halt in difficult circumstances.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient needs a lot of assistance in making any reasonable decision for himself. Presents all issues in a vague manner.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient is incapable of judging or making own decisions, even with help. Constant uncertainty and ambivalence may be primary symptoms.</td>
</tr>
</tbody>
</table>
B) Cognition:

III. Memory: Evaluates the patient's ability to register information and retrieve it after 10 minutes. After explaining the procedure to the patient, the rater presents to the patient, only once, 5 words representing objects, at 1-second intervals, and immediately asks the patient to repeat them. The rater then asks the patient to remember those words for 10 minutes, at which time the patient is asked to recall the words. Only if the patient fails to recall should the rater use some clue and/or encouragement to elicit more. During the 10-minute interval, the patient is assessed on the following 7 items of the scale.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient recalls all 5 words without difficulty.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient recalls 4 words, or may recall the 5th word after prompting.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient recalls 2-3 words, in spite of prompting.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient does not recall any of the words, or may recall 1 after prompting.</td>
</tr>
</tbody>
</table>

IV. Attention: Measures the patient's ability to focus his concentration on simple mental tasks such as serial "3s." The rater asks the patient to subtract "3" aloud serially, starting from 100 for 30 seconds. In order to exclude practice effect, the rater may change the starting point to another 2- or 3-digit number. The task is administered by the rater and the patient is instructed that only the number of correct subtractions counts and there will be no penalty for wrong answers. The rater will tell the patient that the procedure is to be timed and will notify the patient when the timing starts.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient correctly does over 10 serial subtractions.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient correctly does only 5-10 serial subtractions with some hesitation.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient stalls, performs in a halting manner, and can do only 2-4 serial subtractions correctly.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient is unable to subtract more than once.</td>
</tr>
</tbody>
</table>

V. Orientation: Measures the patient's capacity to register occurrences within the surrounding environment, to react to them, and to know of the effect the events may have on him. It evaluates the patient's orientation, reality testing, and adaptive skills in a social environment.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient is oriented to time, place, and person (x3). Appears to follow conversation or action, participates actively and appropriately in activities, and can easily elaborate on what is going on in the community in which the patient lives.</td>
</tr>
<tr>
<td>(+) 1-2</td>
<td>The patient is oriented (x2). May have the day of the week or month wrong, but is oriented to the month of the year and the year, person, and place. Has trouble following conversation and may fall behind others when participating in activities. May seem mildly confused.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient is oriented to person, but is confused about time and place. May lose track of scheduled activities including personal needs and requires reminding. May have difficulty recognizing some people, but responds somewhat to reality testing and orientation by another.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient is totally disoriented and confused. May recognize only a few key people.</td>
</tr>
</tbody>
</table>
(C) Volition/Motivation:

VI. Grooming: Evaluates the care taken by the patient to maintain personal appearance, including grooming, clothing, hair, face, hands, and fingernails.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient is appropriately dressed with <em>clean</em> clothing, combed hair, a clean face, and hands, and clipped fingernails.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient’s clothing is clean, but <em>untidy</em>, and some neglect of shaving, make-up or nail care may be visible.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>Clothing may be stained and <em>disheveled</em>, hair is poorly combed, and facial and hand cleanliness have been neglected.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient and the patient’s clothing are grossly <em>soiled</em>, with <em>no attempt</em> at self-care.</td>
</tr>
</tbody>
</table>

VII. Motivation: Evaluates the patient’s ability to plan and carry out tasks of daily living and other constructive activities.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient’s living space, possessions, and clothing are clean and well organized. Plans and carries out work or school tasks <em>without supervision</em>.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>Encouragement and <em>reminders</em> are needed for the patient to maintain living space and clothing. Without supervision, the patient becomes unfocused and inefficient at work, school, or other activities.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td><em>Ongoing supervision</em> is required even for the performance of tasks of daily living. The patient is unable to make plans or carry out work or school assignments, even with supervision.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td><em>Active physical assistance</em> is required for simple tasks such as bathing, dressing, and eating. The patient is apathetic.</td>
</tr>
</tbody>
</table>

VIII. Motion: Evaluates the amount and speed of large, voluntary body movement. The patient is asked to *stand up and make 5 steps, turn around, and return to the chair.*

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient <em>completes</em> the entire task briskly with <em>no hesitation</em>.</td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient shows a <em>lag</em> in starting the task and/or <em>mild slowing</em> of gait.</td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient is <em>very sluggish</em>, but able to complete the task. The patient may require encouragement.</td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient may slowly stand up, but cannot complete the task or remains immobile.</td>
</tr>
</tbody>
</table>
(D) Affect/Relatedness:

IX. Emotional response: Evaluates the range of emotional expression and the promptness with which the patient is able to react to emotional stimuli such as humor (e.g., jokes), frustration (e.g., limit-setting), aggravation, danger, etc.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient reacts swiftly to emotional stimuli. Can be easily seen readily laughing, crying, expressing anger or fear, with voluntary movements and gestures, showing a full emotional range in both intensity and quality.</td>
<td></td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient is seen reacting slowly to emotions. Can often be seen smiling, weeping, looking upset or anxious, but there is a distinct gap between stimulus and reaction. Little movement or gesture accompanies the emotional reaction.</td>
<td></td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient rarely appears moved by the emotion (e.g., eyes at times may look teary without weeping; voice may be raised for an instant or a smile may last for a second) and then one would wonder what led to the change in affect. No other manifestations of emotion are present. The affect is blunted.</td>
<td></td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient’s affect is flat regardless of circumstances. Cannot be seen expressing emotion at all. Speech sounds monotonous.</td>
<td></td>
</tr>
</tbody>
</table>

X. Expressive relatedness: Evaluates the spontaneity, amount, and sincerity of interactions between the patient and others in the environment. Both verbal and nonverbal communication (including eye contact, facial expression, and gestures) are to be assessed.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>The patient freely initiates contacts and relates in a genuine and animated fashion.</td>
<td></td>
</tr>
<tr>
<td>(-) 1-2</td>
<td>The patient does not initiate conversation, but when engaged relates in an appropriate manner. He interacts with more than one “friend.”</td>
<td></td>
</tr>
<tr>
<td>(-) 3-4</td>
<td>The patient may actively avoid or cut short interactions. He can be engaged in a conversation but evokes no clear feeling of warmth.</td>
<td></td>
</tr>
<tr>
<td>(-) 5-6</td>
<td>The patient shows no meaningful interactions verbally, with no expression of thoughts or feelings and no eye contact. At best, his interactions consist of simple, mechanical requests for material items. He is withdrawn and seclusive.</td>
<td></td>
</tr>
</tbody>
</table>
Appendix G

Case Manager Rating Scale for Alcohol Use Disorder (Drake et al., 1990)

Please rate your client's use of alcohol over the past year according to the following scale. You should weigh evidence from self-report, interviews, behavioral observations, and collateral reports (family, group home, day center, community, etc.) in making this rating.

☐ 1 = none. Client has not used alcohol during this time interval.

☐ 2 = mild. Client has used alcohol during this time interval, but there is no evidence of persistent or recurring social, occupational, psychological, or physical problems related to use and no evidence of recurrent dangerous use.

☐ 3 = moderate. Client has used alcohol during this time interval and there is evidence of persistent or recurrent social, occupational, psychological, or physical problems related to use or evidence of recurrent dangerous use. Problems have persisted for at least 1 month. For example, recurrent alcohol use leads to disruptive behavior and housing problems.

☐ 4 = severe. Meets criteria for moderate plus at least three of the following: greater amounts or intervals of use than intended, much of time spent obtaining or using alcohol, frequent intoxication or withdrawal interferes with other activities, important activities given up because of alcohol use, continued use despite knowledge of alcohol-related problems, marked tolerance, characteristic withdrawal symptoms, and alcohol used to relieve or avoid withdrawal symptoms. For example, drinking binges and preoccupation with drinking have caused client to drop out of job training and non-drinking social activities.

☐ 5 = extremely severe. Meets criteria for severe—plus related problems are so severe that they make noninstitutional 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.
Appendix H

Demographic Data

1. Age: _____________________
2. Sex: _____________________
3. Ethnicity: __________________
4. Education Level: __________________
5. Employment Status: __________________
6. Marital Status: __________________
7. Housing Status: __________________________________________
8. Social Supports: _______________________________________________
9. Age At Onset: __________________
10. Medication __________________ mg/d
     __________________ mg/d
     __________________ mg/d
11. DSM-IV diagnoses __________________
12. Number of Consecutive Years in Alaska: ____________
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