Neurocognitive and Social Cognitive Predictors of Change in Objective versus Subjective Quality of Life Measures in Response to Rehabilitation in Schizophrenia

by

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For Mitchell
Abstract

Introduction: Quality of life (QOL) has been recognized as an important domain of outcome in patients with schizophrenia but the determinants of objective and subjective dimensions of this construct differ. Very few studies have examined the role of cognitive skills in predicting changes in QOL to integrated programs of rehabilitation in schizophrenia. Researchers have found that cognitive skills predict a patient’s ability to benefit from cognitive remediation (Kurtz et al., 2009). However, no studies have directly compared the roles and interactions between neurocognition, social cognition, and other disease factors in predicting improvements in the two domains of objective quality of life (QOL) and subjective QOL or satisfaction with life (SWL) in the same sample of patients in response to rehabilitation in schizophrenia. Methods: Forty-four clinically stable outpatients with schizophrenia were administered measures of neurocognition, social cognition, and symptoms at entry to a psychosocial and cognitive rehabilitation program. Objective QOL and subjective SWL scales were given before and after a year of treatment as a measure of outcome. Models of mediation and moderation were applied when intercorrelations of neurocognitive and social cognitive measures and change in QOL were evident. Results: A neurocognitive measure of verbal memory and a social cognitive measure of facial affect recognition were linked to improvement in objective QOL. Only verbal memory was linked to improvements in subjective SWL. Facial affect recognition partially mediated the relationship between verbal memory and improvement in objective QOL. Discussion: Our findings suggest that social cognition may be as essential to enhanced functional outcome as neurocognition after rehabilitation interventions in schizophrenia. These findings provide evidence for the types of patients most likely to benefit from current rehabilitation interventions and may help researchers develop novel interventions in the future.
1. Introduction

Schizophrenia is a devastating mental disorder characterized by cognitive, emotional, social, and motor disturbances. It is a chronic disorder that has high relapse rates and affects about 1% of the population (Frangou & Murray, 2000; Nolen-Hoeksema, 2001). Impaired neurocognitive functioning, including attention, memory, language, and problem solving, has been linked to a decline in functional outcome in schizophrenia (Kurtz, Wexler, Fujimoto, Shagan, & Seltzer, 2008). Along with impaired functioning, people with schizophrenia suffer from a variety of symptoms ranging from hallucinations and delusions to blunted affect, social withdrawal and depression (Frangou & Murray, 2000). Schizophrenia manifests itself in different ways in each person. Patients exhibit a variety of symptoms, neurocognitive abilities and functional outcomes (Grube, Bilder & Goldman, 1998).

Epidemiology

Many people are surprised to find out that over 2 million people in the United States are diagnosed with schizophrenia. The lifetime prevalence for schizophrenia is about 1%, similar to that of epilepsy (Butcher, Mineka, & Hooley, 2010; Frangou & Murray, 2000). The World Health Organization conducted an international pilot study of schizophrenia, which surveyed the incidence of schizophrenia around the world. They discovered that schizophrenia is present in all countries and that the annual incidence per 1000 people ranged from .15 in Denmark to .42 in India (WHO, 1973). Although the
peak onset for schizophrenia is between 15 to 25 years old for men and 25 to 35 years old for women, late onset is still possible (Hafner et al., 1994). Of the population of people with schizophrenia, about 28% were diagnosed after the age of 44 and about 12% after 63 (Castle & Murray, 1993). Schizophrenia affects both men and women in similar numbers, but the course of the illness varies greatly between genders. In general, men tend to have a worse social course, which may be a consequence of their earlier onset (Hafner, 2003). In addition to poorer social functioning, men also exhibit more severe symptoms (Leung & Chue, 2000). Researchers, in an attempt to understand the better clinical outcome of women, have found that estrogen may have a protective effect against schizophrenia. They found that high levels of estrogen may ward off early onset in females and that the decrease in estrogen later in life may explain why late onset is more common in women (Lindamer, Lohr, Harris, & Jeste, 1997). In addition to a later onset, women often present fewer negative symptoms, which are harder to treat and positive symptoms that are more responsive to pharmacotherapy (Kraly, 2006).

**Etiology**

The causes of schizophrenia are complex and likely include psychological, neurological and genetic factors. The stress-diathesis model is one etiological theory present in the current literature. This model proposes that one set of factors, often environmental or genetic, causes a predisposition or vulnerability. These factors then interact with stress, which triggers the actual onset of the
disorder. There are many factors that can cause a predisposition including, perinatal brain damage, prenatal virus exposure, birth complications, maternal stress, psychosocial events, and drug use (Jablensky, 2000; Nolen-Hoeksema, 2001). The HPA, hypothalamic-pituitary-adrenal, axis mediates the response to stressors. Stress activates the HPA axis, which in turn releases the glucocorticoid cortisol. Chronic stress leads to neural changes, sensitization and neuron death in hippocampus, impairing memory (Corcoran et al., 2003). Chronicly high levels of cortisol in the body can also affect neurotransmitters. The augmentation of dopamine in the mesolimbic pathway due to chronic stress can be partially responsible for the emergence of symptoms in schizophrenia (Frangou & Murray, 2000).

In line with the stress-diathesis model, it is likely that genetics play a large role in the etiology of schizophrenia. Family studies have shown that the amount of genetic material you share with a relative with schizophrenia has a direct effect on your risk of developing the disease as well. Twin studies have shown that monozygotic twins, sharing 100% of their DNA, have about a 50% risk of developing schizophrenia if their twin is diagnosed, whereas dizygotic twins only have about a 15-20% risk (Butcher et al., 2010; Gottesman, 1991).

While research has shed light on the familial nature of schizophrenia, the exact genes that are implicated in the inheritance are still unclear. Linkage analysis is beginning to reveal some of the genes that are associated with schizophrenia (Butcher et al., 2010). Researchers have identified two main genes that they believe may be linked to schizophrenia, COMT (catechol-O-
methyltransferase) and BDNF (brain derived neurotropic factor). COMT is found on chromosome 22q11 and is involved with dopamine catabolism (Craddock, O’Donovan, & Owen, 2006). Since increased function in the dopaminergic pathways is characteristic in people with schizophrenia, it is likely that COMT plays some role in its etiology. A polymorphism of the COMT gene that results in a valine to methionine mutation has been associated with the emergence of schizophrenia (Braff, Freedman, Schork, & Gottesman, 2007). BDNF is another gene that has been linked to the cause of schizophrenia. It is located on chromosome 11p13 and encodes for a brain-derived neurotropic factor that modifies the growth, development, plasticity, and survival of neurons (Craddock et al., 2006). More research needs to be done to further implicate COMT’s and BDNF’s causal relationship with schizophrenia, but it does seem likely that an inherited variant of these genes can predispose people to developing the disorder.

Technological advances have allowed researchers to more extensively examine the anatomical differences in the brains of people with mental illness. Magnetic resonance imaging (MRI) of the brains of humans very early in development have shown lesions that likely cause a vulnerability to schizophrenia (Butcher et al., 2010). These lesions may be the cause of the motor abnormalities and lack of facial emotion that can be seen in children even before they are diagnosed with schizophrenia (Walker, Grimes, Davis, & Smith, 1993). One of the most well researched brain abnormalities deals with enlargement of the ventricles in the brain. MRIs of people with schizophrenia have shown that
the lateral and third ventricles are about 20-30% larger than those in controls (Lawrie & Abukmeil, 1998; Wright et al., 2000). Enlarged ventricles indicates diminished brain tissue, which is present in recent onset patients suggesting that this may be a predisposing factor for the development of schizophrenia (Matsumoto et al., 2001).

Along with enlarged ventricles, people with schizophrenia often have irregularities in their frontal lobe, temporal lobe, hippocampus and amygdala (Butcher et al., 2010; First & Tasman, 2004). The frontal lobe plays a role in executive functions such as, working memory, attention, suppression of interference, and ability to recognize consequences. Volumetric analysis of structural MRIs have shown a 2-8% reduction in frontal lobe volume, which may be responsible for the decreased activation during cognitive tasks (Kindermann, Karimi, Symonds, Brown, &Jeste, 1997; Wright et al., 2000). Researchers have also found a 2-4% reduction in temporal lobe volume (Wright et al., 2000). The temporal lobe, which is associated with auditory processing, contains Wernicke's area, which is responsible for the conception and organization of speech (First & Tasman, 2004). Deficits and disruptions in the temporal and frontal lobes are likely related to auditory hallucinations and cognitive difficulties that are key characteristics of schizophrenia. The hippocampus and amygdala, which have important functions for memory, emotional expression, and social affiliation, also exhibit a bilateral volumetric reduction of 6-10% (Nelson, Saykin, Flashman, & Riordan, 1998; Wright et al., 2000). These
reductions may be attributed to some of the negative symptoms expressed in those with schizophrenia, such as blunted affect.

**Diagnosis**

The Diagnostic and Statistical Manual of Mental Disorders (DSM) provides the standard criteria for classifying various mental disorders. It is broken down in a five-axis system, with schizophrenia falling into axis 1. The DSM is currently in its fourth edition- text revision (IV-TR). The diagnostic characteristics for schizophrenia are broken down into six criteria, labeled A-F, and are as follows (APA, 1994; DSM IV-TR):

*Criterion A: Characteristic symptoms.* Two or more of the following must be present for the majority of a one month period; (a) delusions, (b) hallucinations, (c) disorganized speech, frequent derailment or incoherence, (d) grossly disorganized or catatonic behavior, (e) negative symptoms (flattened affect, alogia, avolition). Only one symptom is required for diagnosis if there is evidence of bizarre delusions or hallucinations of a voice that keeps a running commentary on behavior and thoughts, or hallucinations of two or more voices conversing with each other.

*Criterion B: Social/occupational dysfunction.* One or more areas of functioning such as work, interpersonal relations, or self-care are significantly below the level achieved prior to the onset.

*Criterion C: Duration.* Signs of disturbance must last for at least 6 months, including one month of symptoms that meet criterion A. This may include
prodromal or residual periods where the disturbances manifest themselves as negative symptoms or less severe symptoms than those in criterion A.

Criterion D: Schizoaffective and mood disorder exclusion. This excludes individuals who have more mood aspects of their illness because their symptoms may be related to schizoaffective or a mood disorder, not schizophrenia.

Criterion E: Substance/ general medical condition exclusion. This excludes individuals whose disturbances may be to due to the physiological effects of a substance, such as drugs, or a general medical condition.

Criterion F: Relationship to a pervasive developmental disorder. Schizophrenia can only be diagnosed in individuals with prior diagnoses of autism or a developmental disorder if they exhibit prominent delusions or hallucinations lasting at least one month.

The six criteria of diagnosis are an attempt to narrow the scope of schizophrenia and more accurately diagnose people with the correct mental illness. Although diagnosis is difficult, the DSM is always making revisions to their criteria and creating new guidelines to follow. In addition to the main diagnosis of schizophrenia, there are five subtypes including: paranoid, disorganized, catatonic, undifferentiated and residual (First & Tasman, 2004).

Symptomatology

Although individuals must exhibit a range of symptoms in order to be diagnosed with schizophrenia, these symptoms often vary by person and over
the course of the illness (Hughes et al., 2003). Symptoms are often divided into three categories, positive, negative and general, based on the Positive and Negative Symptom Scale (PANSS) (Kay, Fiszbein, & Opler, 1987). This differentiation began in the 19th century with Sir John Russell Reynolds, a British neurologist who proposed that physical signs of an illness could manifest themselves in positive and negative forms (Berrios, 1985.)

Positive symptoms are defined as an excess or distortion of normal behaviors and experiences (Butcher et al., 2010). The most common positive symptoms are delusions and hallucinations. Delusions are unfounded or unrealistic false beliefs that are not responsive to reasoning. They are expressed as persecutory, paranoid or grandiose. Hallucinations are sensory experiences that happen without the presence of any external stimuli and can manifest themselves as auditory, visual, olfactory or tactile (Butcher et al., 2010; Frangou & Murray, 2000; Kay et al., 1987). In a study of over 6,000 individuals with schizophrenia, researchers found that over 52% of patients exhibit positive symptoms. More specifically, 73-90% report delusions and 59% report hallucinations (Lecrubier, Perry, Milligan, Leeuwenkamp, & Morlock, 2007; Cutting, 1995).

Although positive symptoms are quite prevalent, they may not be as disabling as once thought. Studies have found that positive symptoms are not highly correlated to adaptive functioning, cognitive deficits or the general course of illness (Velligan et al., 1997). Positive symptoms are related to prognosis, and can often be controlled by medication. Since an excess of dopamine in the body
may be one of the causes of positive symptoms, neuroleptics and other antipsychotics are an effective treatment method (Frangou & Murray, 2000; Lecrubier et al., 2007).

Negative symptoms are defined by the deficit of normally present behaviors (Butcher et al., 2001). These symptoms are often difficult to diagnose because they are based on an absence rather than a presence of behavior and they lie on a continuum of normal to abnormal rather than being clearly unusual (Nolen-Hoeksema, 2001). Blunted affect is a negative symptom characterized by diminished emotional responsiveness. This manifests itself in a reduction of facial expression, regulation of feelings, and communicative gestures, often leading to monotone voice or complete lack of facial expression (Kay et al., 1987; Nolen-Hoeksema, 2001). Emotional and social withdrawal can be described as a lack of interest in and involvement with life’s events often resulting in diminished social interactions (Kay et al., 1987). Other negative symptoms include apathy, avolition, anhedonia, and passivity. In a study of symptomatology in a sample of 6,000 individuals with schizophrenia, 38% exhibited blunted affect and 54% exhibited social withdrawal (Lecrubier et al., 2007).

Even though negative symptoms seem to be slightly less prominent than positive symptoms, they are more correlated to poor prognosis. Negative symptoms are persistent over time and are only minimally responsive to medication. Therefore their relation to poor outcome is more troubling (Blanchard, Kring, Horan, & Gur, 2011). Other studies have found that negative symptoms may impede on long-term improvements of quality of life (Lecrubier
Negative symptoms are extremely hard to treat due to the fact that their cause is unknown. These symptoms may be caused by comorbid disorders such as depression, social isolation, and stigma, or side effects of medications used to treat positive symptoms (Nolen-Hoeksema, 2001).

Not all symptoms can fit easily into the categories of positive and negative, therefore the PANSS uses a category of general symptoms. These symptoms include anxiety, depression and lack of insight (Kay et al., 1987). Depression and anxiety are often comorbid in schizophrenia and cannot be treated as simply symptoms of schizophrenia (Butcher et al., 2010). Another newly described facet of symptomatology is disorganization, which is often listed with positive symptoms when it is not given its own category (Shean, 2004). Bizarre behavior is one symptom classified by disorganization that is defined as a disruption of goal-directed activity, often related to work, social relations, and self-care. These disruptions of behavior may be due to impaired functioning in the prefrontal cortex (Butcher et al., 2010). Disorganized speech is another symptom that is described as an external manifestation of a disorder in thought form. Individuals who present disorganized speech often use sentences that do not make syntactical sense or use made up words called neologisms, but these errors cannot be attributed to poor intelligence or cultural knowledge (Butcher et al., 2010). Although these symptoms reflect a type of cognitive slippage, they cannot be attributed to the various other cognitive deficits present in schizophrenia.
Neurocognitive Deficits

There has been increasing interest in the field of neurocognition in schizophrenia due to its link to functional outcome, but it is by no means a new topic of interest. Emil Kraeplin, a German psychiatrist who is known for his description of schizophrenia in the 19th century, may have been the first to mention the presence of neurocognitive deficits. In his description of dementia praecox, later known as schizophrenia, he noted that mental deterioration beginning in early life was a central feature of the disorder (Butcher et al., 2010; Frith & Johnstone, 2003). Researchers have found that these deficits are present at disease onset (Saykin et al., 1994), generally stable over time (Kurtz, Seltzer, Ferrand, & Wexler, 2005), mostly unaffected by pharmacological intervention (Keefe et al., 2007), and are not the results of positive or negative symptoms (Harvey, Green, Keefe, & Velligan, 2004). While many individuals with schizophrenia exhibit a decrease in general IQ scores, most of the research focuses on more specific factors of cognition. The National Institute of Mental Health- Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) have identified a handful of factors including, memory, attention, and executive function (Green et al., 2004b).

Memory is a complex function that is often compromised in individuals with schizophrenia. Working memory is the ability to hold information for a short period of time before recall. Researchers often use the letter-number sequence test from the Wechsler Adult Intelligence scale (Wechsler, 1997, 2008) to measure working memory. Researchers have found that working memory
deficits are attributable to attention or executive function factors (Goldberg, Patterson, Taququ, & Wilder, 1998). Declarative memory refers to unconscious memories of skills, personal information and facts. The California verbal learning test (Delis, Kramer, Kaplan, & Opler, 2000) is used to measure verbal memory. Many studies have shown severe deficits in explicit or conscious recollection based on verbal memory tasks (Frangou & Murray, 2000; Heinrichs & Zakzanis, 1998). Memory deficits in schizophrenia have been linked to a failure to comply with medication intake, unusual interpretations of external events and ultimately functional outcome (Johnstone, Humphreys, Lang, Lawrie, & Sandler, 1999).

The deficits associated with attention and schizophrenia have been studied for many years dating back to Broadbent’s theory about improper filtering or inhibition of irrelevant information (Johnstone et al., 1999). Most studies on attention deal with vigilance or sustained attention, which is often measured by the Penn continuous performance test (Kurtz, Ragland, Bilker, Gur, & Gur, 2001). Studies have found that sustained attention is significantly impaired in both medicated and non-medicated patients (Pandurangi, Sax, Pelonera, & Goldberg, 1994). Selective attention is the ability to focus on pertinent stimuli and avoid distraction. Researchers, using the Stroop test, have found that individuals with schizophrenia display deficits in selective attention when compared with healthy controls and this may be correlated with their difficulty with reality monitoring (Brebion, Smith, Gorman, & Amador, 1996). Improved sustained and selective attention has consistently been related to
clinical improvement in schizophrenia (Nopoulos, Flashman, Flaum, Arndt, & Andreasen, 1994).

While attention deficits are clearly a feature of schizophrenia, the impairments that pertain to attending to several tasks at once may reflect the general category of executive functioning (Johnstone et al., 1999). Executive functioning often refers to volition, planning, self-monitoring behavior, cognitive flexibility, abstract thinking, and complex information processing (Frangou & Murray, 2000; Green, Kern, Braff, & Mintz, 2000). Many studies have shown deficits in executive functioning as tested by the Wisconsin card-sorting test. Executive functioning has been linked to the number of hospitalizations, community functioning and overall outcome (Heinrichs & Zakzanis, 1998).

The interest in researching neurocognitive deficits in schizophrenia has been on the rise since it was implicated with functional outcome. Impairments range from one to two standard deviations away from healthy controls on a variety of neurocognitive measures including, episodic/working memory, attention, language, and problem solving (Heinrichs & Zakzanis, 1998). A meta-analysis found that neurocognition may explain 20-60% of the variation in functional outcome and may be a better predictor of course than other symptoms (Green et al., 2000). Neurocognition has also been connected to social and work aspects of community functioning (Brekke, Hoe, Long, & Green, 2007; Green, Kern, & Heaton, 2004a).
Social Cognitive Deficits

Social cognition refers to the mental activities that underlie social interactions. They are mainly perceiving, interpreting, and producing responses to the intentions, dispositions, and behaviors of other individuals (Brothers, 1990; Fiske & Taylor, 1984). Social cognition has been most commonly divided into several component processes, including affect recognition, perception of social stimuli, self-perception, and theory of mind (Penn, Combs, & Mohamed, 2001). Individuals with schizophrenia often display deficits in all of these social cognitive factors. These deficits are stable over the course of the illness and are key in determining daily functioning such as, instrumental behavior, interpersonal functioning, and vocational achievement (Mueser et al., 1996; Penn, Corrigan, Bentall, Racenstein, & Newman, 1997). The inability to understand social situations often leads to misconceptions, which can result in inappropriate interpersonal relations or social withdrawal (Penn et al., 1997; Green & Nuechterlein, 1999). Recently, studies have found relationships between social cognitive factors, such as social perception, theory of mind (ToM) and emotional perception, and functional outcome (Couture, Penn, & Roberts, 2006).

Social perception is an individual’s ability to discern social cues from behavior provided in a social context and adhere to social rules and conventions (Couture et al., 2006; Green, Oliver, Crawley, Penn, & Silverstein, 2005). This aspect of social cognition has been related to outcomes in community functioning in individuals with schizophrenia (Green et al., 2008). Studies have
found that social perceptions mediate the relationship between neurocognition and functional outcome (Sergi, Rassovsky, Nuechterlein, & Green, 2006; Vauth, Rusch, Wirtz, & Corrigan, 2004).

Theory of mind refers to the ability to understand that others have mental states that differ from one’s own and the capability to make accurate inferences about the content of other’s mental states. ToM often deals with understanding false beliefs or verbal hints (Couture et al., 2006). Deficits in ToM have been related to premorbid social functioning in individuals with schizophrenia (Schenkel, Spaulding, & Silverstein, 2005). Researchers often use The Hinting Task to assess patient’s ToM. This task measures indirect speech, the ability to express content without quoting explicit statements, through a series of statements. These statements can be interpreted in several ways, but participants are required to understand the real intended meaning via cues in the social setting in which the statement is made (Corcoran, Mercer, & Frith, 1995). Diminished performance on the hinting task is associated with poor social skills in schizophrenia (Pinkham & Penn, 2006). While preliminary studies have shown some relation between ToM and social skill, community functioning, and social behavior, it is difficult to draw conclusions about the relationship between ToM and functional outcome based on the paucity of data collected to date (Couture et al., 2006).

Emotional perception or affect recognition is the ability to infer emotional information from facial expressions and voice inflections (Couture et al., 2006). The inability to recognize emotional states is associated with community
functioning, successful social behavior, and social skill in schizophrenia (Green et al., 2008; Mueser et al., 1996). Researchers have found that affect recognition may mediate the relationship between neurocognition and functional outcome (Addington, Saeedi, & Addington, 2006; Brekke, Kay, Kee, & Green, 2005). It is possible that deficits in emotional perception lead to less successful social encounters, which may augment stress, leading to a greater chance of relapse and possibly less desire to interact with others (Penn et al., 2001).

Many aspects of social cognitive deficits in schizophrenia reflect the nature of negative symptoms. Researchers have found that social cognition, specifically ToM, is directly associated with negative symptom severity. Social cognition explains more than 1/3 of the variance in negative symptoms (Lincoln, Mehl, Kesting, & Rief, 2011). While it seems as if these two aspects of schizophrenia are related to each other, it is hard to decipher their relatedness due to their tautological relationship. It is difficult to measure negative symptoms without assessing social functioning (Corrigan & Penn, 2001).

**Objective and Subjective Measures of Quality of Life**

It is very difficult to define endpoints in studies of functional outcome in schizophrenia, but objective measures of community functioning and quality-of-life are often used (Green et al., 2000, 2004a). The quality of life (QOL) measure is a multi-dimensional construct that includes objective components of community function including occupational role, active acquaintances and social initiatives (Test et al., 2005). The domain of social initiatives may be measured
objectively by asking the question, e.g., “How often have you asked people to do something with you?” (Heinrichs et al., 1984).

Any complete assessment of outcome must also include patient’s self-reported quality of life. The Satisfaction with Life Scale (SWL) measures patient’s subjective satisfaction in various aspects of life including work, self-life, social relationships, and living situation, e.g., “How satisfied are you with your current social life?” (Stein & Test, 1980; Test et al., 2005). Generally, poor QOL and SWL are related to unemployment, lack of friends, and few leisure activities (Thornicroft et al., 2004).

There is much controversy over the reliability of SWL measures. Some researchers state that self-reports of functional ability may be problematic (Harvey, Velligan, & Bellack, 2007), while others state that self-reports are more valid than clinical reports due to patient’s accuracy and consistency (Becchi, Rucci, Placentino, Neri, & deGirolamo, 2004; Voruganti, Heslegrave, Awad, & Seeman, 1998). Despite the fact that researchers are stressing its importance, SWL as an outcome domain is a neglected area of research in studies of quality of life in schizophrenia. Since neurocognitive and social cognitive factors are related to functional outcome in schizophrenia, looking at their relationships to SWL and QOL may shed some light on new targets for treatment.

Researchers have found mixed findings about the relationship between subjective SWL and neurocognitive functions in schizophrenia, ranging from no relationship (Brissos, Dias, Lemos, Carita, & Martinez-Aran, 2008; Chino, Mercier, Diaz, & Martin, 2009), to negative relationships (Corrigan & Buican,
1995; Narvaez, Twamley, McKibbin, Heaton, & Patterson, 2008), to positive relationships (Alpetkin et al., 2005). Researchers found a moderately negative relationship between cognitive ability, social cognition, and SWL (Tolman & Kurtz, In press). Other researchers have displayed similar findings, relating an increase in global cognition, measured by a composite z-score in processing speed, working memory, learning and executive function, with a decrease in SWL (Narvaez et al., 2008).

Studies on the relationship between objective QOL and neurocognition have also led to mixed findings, with some researchers finding no relationship (Heslegrave, Awad, & Voruganti, 1997; Hofer et al., 2005), others a negative relationship (Corrigan & Buican, 1995), and still others a positive relationship (Addington & Addington, 2008; Lysaker & Davis, 2004). Researchers have found that QOL is positively related to functional capacity, memory, and number of hours worked, and inversely related to positive, negative, and depressive symptoms (Corrigan & Buican, 1995; Fiszdon, Choi, Goulet, & Bell, 2008; Palmer et al., 2002). Moderator studies have shown that facial affect moderates the relationship between neurocognition and QOL (Addington et al., 2006).

Even though recent research indicates that SWL is related to measures of QOL, different features of the illness may influence it. A meta-analysis of 20 cross-sectional studies revealed moderate relationships between crystallized verbal intelligence, working memory, verbal list learning, processing speed, executive function, and QOL, but non-significant or inverse relationships between processing speed, crystallized verbal ability, and SWL (Tolman & Kurtz,
In press). While neurocognition is generally not affected by symptomatology, studies have found that the severity of depression, not positive or negative symptoms, is related to SWL, while QOL is not greatly affected by symptoms (Kurtz & Tolman, 2011; Narvaez et al., 2008; Tolman & Kurtz, In press).

Although QOL and SWL are often used to measure outcome in schizophrenia, there are relatively few studies that compare clinical, functional, and cognitive predictors of change in subjective SWL and objective QOL. A cross-sectional study of 88 outpatients showed that more severe depressive symptoms and increased neurocognition predicted poorer SWL, whereas decreased neurocognition predicted poorer QOL (Narvaez et al., 2008). A longitudinal study of 55 participants during rehabilitation showed that better baseline visual memory predicted better objective community functioning, while poorer baseline attention predicted better SWL (Prouteau et al., 2005).

One possible explanation for inverse relationship between cognitive ability and subjective SWL might be related to a patient’s increased insight into their illness and disabilities as a result of higher cognitive functioning. This insight may enable negative social comparison and allow them to understand the stigma associated with schizophrenia and therefore decreases their SWL (Brekke, Kohrt, & Green, 2001; Karow & Pajonk, 2006; Narvaez et al., 2008). In contrast, poor cognition may directly interfere with the negotiation of successful relationships with others, the ability to carry-through on required tasks in competitive employment settings, and the ability to engage in community
activities. All of these aspects of outcome are measures by objective, but not subjective measures of outcome.

**Cognitive Remediation**

Many patients with schizophrenia take a variety of different antipsychotic medication. Studies have found that functional outcome, neurocognition, and negative symptoms are largely unaffected by these treatments, which led to the development of cognitive treatment for schizophrenia (Beck, Rector, Stolar, & Grant, 2009). Cognitive remediation is a course of cognitive exercises designed to improve attention, verbal and non-verbal memory, and language processing through repeated drills (Bracy, 1995; Kurtz, Seltzer, Shagan, Thime, & Wexler, 2007; Seltzer, Cassens, Ciocca, & O'Sullivan, 1997). It has been shown to enhance sustained attention, language processing (Wexler et al., 1997), executive functioning (Bell, Bryson, Greig, Corcoran, & Wexler, 2001; Wykes, Reeder, Corner, Williams, & Everitt, 1999), affect recognition (Bell et al., 2001), verbal memory (Hogarty et al., 2004; McGurk & Mueser, 2004), working memory (Bell et al., 2001; Kurtz et al., 2007), processing speed (Hogarty et al., 2004), and social problem solving (Kern et al., 2005).

Recent research has shed some light on the effect that cognitive remediation has on various aspects of neurocognitive performance and psychosocial functioning. A two-year study on the effect of cognitive enhancement therapy on cognition and behavior found improvements in neurocognition and processing speed after 12 months. During a follow-up after
24 months of cognitive enhancement therapy, researchers found improvements in neurocognition, processing speed, cognitive style, social cognition, and social adjustment (Hogarty et al., 2004). In a randomized control trial on the effects of computer assisted cognitive remediation therapy, researchers found improvements in working memory, reasoning, executive functioning, verbal and spatial episodic memory, and processing speed after 12 months of treatment relative to baseline measures (Kurtz et al., 2007).

A meta-analysis of 2,104 patients revealed that cognitive remediation produced durable effects on global cognition (effect size = .428) and functioning (effect size = .372) and had a moderate effect on cognitive outcome measures (effect size range = .25 - .65) (Wykes, Huddy, Cellard, McGurk, & Czobor, 2011). Researchers have also found that in patients receiving cognitive remediation therapy, sustained attention and working memory are linked to ability to benefit from cognitive remediation (Kurtz, Seltzer, Fujimoto, Shagan, & Wexler, 2009). This research suggests that cognitive skills predict a patient’s ability to capitalize on cognitive remediation programs. While research has proven that cognitive remediation does have a significant effect on outcome and neurocognition, the benefits of this treatment can be further enhanced when it is combined with other psychosocial rehabilitation programs (McGurk et al., 2007; Wykes et al., 2011).

Even though there is extensive research in the field of cognitive remediation, there are still many unanswered questions. It is very difficult to create a new cognitive remediation intervention because researchers and
clinicians are not positive about what cognitive functions should be targeted in order to enhance outcome. Also, while studies have shown a link between cognitive remediation and functional outcome, it is still unclear exactly how strong that link is (Reeder, Newton, Frangou, & Wykes, 2004). Many studies have investigated the effects that cognitive remediation has on functioning, but there has been little research on how these changes in functioning ultimately enhances different domains of patients’ lives.

**Present Study**

The present study explores the degree to which neurocognitive and social cognitive skills, as well as symptoms, measured at entry to a neurocognitive and psychosocial rehabilitation program predicted improvements in objective quality-of-life (QOL) scale (Heinrichs et al., 1984) and change in subjective satisfaction with life scale (SWL; Test et al., 2005) in a substantial sample of people with schizophrenia. We hypothesized first, that there would be an improvement in QOL and SWL scores after rehabilitation. We predicted that, (1) improvements in objective QOL would be associated with improvements in subjective SWL, (2) neurocognitive measures and social cognitive skills but not symptoms, measured at baseline would be linked to improvements in objective QOL but unrelated to measures of subjective SWL, and (3) the social cognitive skill of facial affect recognition would mediate the relationship between baseline neurocognition and change in QOL.
2. Methods

2.1. Design

All procedures met with institutional review board approval. Patients who participated in the study signed written, informed consent forms prior to beginning testing. Cognitive remediation was provided to participants coupled with other rehabilitation programs. All participants were randomly assigned to one of two groups: cognitive remediation or computer-skills training. The cognitive remediation procedures consisted of an extensive (100 hour target) program of computer-assisted exercises, which were ordered hierarchically based on task difficulty. Sessions were held three times per week, for 30-45 minutes each and focused on sustained visual and auditory attention, response inhibition, shifting attention, working memory, semantic processing, verbal recall, and speed of language processing (Kurtz et al., 2009). For the purpose of the current study the data was collapsed across the two computer training groups, but their potential effects were controlled for in the data analysis. In addition to cognitive remediation, participants attended bi-weekly social skills training groups. These groups targeted the improvement of personal interactions with others by using role-play scenarios to act out real life situations. Patients also typically participated in other rehabilitative interventions including vocational counseling, psychoeducation, groups focused on goal setting and goal attainment and exercise.
2.2. Participants

Forty-four outpatients diagnosed with schizophrenia or schizoaffective disorder based on the DSM-IV (APA, 1994) criteria as determined by the Structured Clinical Interview for DSM-IV (SCID, First et al., 1995) participated. Exclusion criteria included evidence of mental retardation as indicated by a history of services, lack of fluency in English, presence or history of neurological illness, traumatic brain injury with sustained loss of consciousness, and/or current substance abuse or dependence (Kurtz et al., 2009). Researchers administered a battery of neurocognitive and psychosocial functioning tests at study entry. The patients in the study were recruited from two mental health clinics in the greater Hartford area. The majority of the patients (n=42) were enrolled in an intensive outpatient program at The Institute of Living, Hartford Hospital’s Mental Health Network (IOL) in Hartford, CT, while two patients attended community health centers in East Hartford, CT. Participants were assessed 6-12 months after program entry at the termination of their computer training. Demographic and clinical characteristics of the sample are presented in Table 1.

2.3. Neurocognitive measures

Since the current study focused on predictors of objective and subjective measures of outcome, performance on neurocognitive measures at baseline were selected for analysis. Measures of neurocognitive function were chosen based on their validity, reliability, and relationship to social cognitive outcome measures as reported in the literature (Nuechterlein et al., 2008).
2.3.1. Penn Conditional Exclusion Test (PCET; Kurtz et al., 2004a,b)

The PCET is a computerized measure of executive functioning, mainly targeting problem solving (Fig.1). Evidence from several other studies shows construct validity in samples of both healthy controls (Kurtz et al., 2004a) and patients with schizophrenia (Kurtz et al., 2004b). The task requires participants to choose one of four items that is different from the others based on one of three sorting categories, including line thickness, shape, size, location, etc. (Kurtz et al., 2004b). Participants are given immediate feedback via a screen reading “correct” or “incorrect” for 500ms and then must infer the sorting rule (Kurtz et al., 2004a). After 10 consecutive “corrects” the sorting category shifts.

![PCET Forms 1 and 2](image)

Fig. 1. Illustration of forms 1 and 2 from the PCET. The highlighted items are the objects that do not belong with the other three.

2.3.2. Penn Continuous Performance Test (PCPT; Kurtz et al., 2001)

The PCPT is a computerized test that measures sustained visual attention. Evidence from Kurtz et al. (2001) has shown high levels of internal stability and reliability for the PCPT for healthy controls and patients with schizophrenia. In this task, participants must respond to vertical and horizontal lines arranged to form a digit. Patients are shown the digits 2, 4 and 5 on target trials and nonsense configurations on distracter trials (Fig.2). The stimuli are presented at a rate of one per second and are revealed for 300 ms each.
2.3.3. California Verbal Learning Test-II (CVLT-II; Delis et al., 2000)

The CVLT is a list-learning task that measures verbal learning and memory. The test consists of a list of 16 words from 4 semantic categories presented 5 times. After each trial, participants are asked to recall as many words from the list as they can remember. This examines trial 1 and 5 because they reflect the baseline and final number of words each participant can remember after all 5 trials.

2.3.4. Digit Symbol Coding (DSS; Wechsler, 1997, 2008)

The Digit Symbol subtest from the Wechsler Adult Intelligence scale (WAIS-III & IV; Wechsler, 1997, 2008) measures participants processing speed. Patients must copy symbols that are paired with numbers into a blank box that is shown below the corresponding number (Fig. 3).

Fig. 3. Illustration of a line of digit-symbol coding from the WAIS-IV.
2.3.5. Letter Number Sequencing (LNSS; Wechsler, 1997, 2008)

The Letter Number Sequencing subtest from the Wechsler Adult Intelligence scale measures working memory. Administrators present a sequence of letters and numbers at a rate of one per second. Participants must repeat the sequence with the numbers in numerical order, followed by the letters in alphabetical order (Fig. 4).

<table>
<thead>
<tr>
<th>Item</th>
<th>Correct response</th>
</tr>
</thead>
</table>

Fig. 4. Illustration of Letter Number Sequencing from the WAIS-IV. The item is what is presented to the participant and the correct response is the sequence after it has been put in numeric and alphabetical order.

2.4. Social cognitive measure

2.4.1. Penn Emotional Acuity Test (PEAT; Gur et al., 2006)

The PEAT is a computerized test that measures social recognition based on participant’s ability to link emotions to various faces. The test consists of 10 happy, 10 sad, and 20 neutral expressions of Caucasian faces that are rated on a 7-point Likert scale, from very sad to very happy.

2.5. Satisfaction with Life Scale (SWL; Stein & Test, 1980; Test et al., 2005)

The SWL scale provides a measure of subjective quality of life. It is a 21-item test answered on a scale of 0 to 4 ranging from not at all to a great deal. Participants rate each questions based on how they feel about their lives at that moment in terms
of their living situation, work, social relationships and psychological state. Scores range from 0 to 72 with higher scores indicating a greater satisfaction with self.

2.6. Quality of Life Scale (QOL; Heinrichs et al., 1984)

The QOL scale is a clinician-rated test that measures objective psychosocial functioning. It is a semi-structured interview containing 21 items with four domains including intrapsychic foundations, interpersonal relations, instrumental role function and common objects and activities (Fizdon et al., 2008). The rating is based on a 6-point Likert scale and scores range from 0 to 126 with a higher rating meaning a better quality of life. This study used the abbreviated seven-item version of the test (Bilker et al., 2003), which has been correlated with scores from the complete version with coefficients from .96 to .98 in patients with schizophrenia. Interrater reliability was maintained throughout the study for all raters by achieving a minimum intraclass correlation coefficient (ICC) of .70 based on four consecutive, independently rated QOL interviews.

2.7. Symptom Assessment

The Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987) was used to assess symptoms. This is an interview-based assessment that measures symptoms on 30 items based on a 7 point Likert scale. The items were then grouped into categories based on the Bell et al. (1993) five-factor model (Table A). The grouping categories include, positive, negative, hostility, cognitive, and emotional discomfort factors. In order to limit method variance in this study the cognitive
subscales was dropped from the analysis. Bell et al. (1993) created the five factors in order to offer more realistic symptom data that relates better to genetic, premorbid, functional and outcome variables. Symptom raters for the study maintained inter-rater reliability through periodic rater training session, and all raters were trained to a reliability of .75 ICC.

**Table A**

The five-component model of schizophrenia symptoms as stated in Bell et al. (1993)

<table>
<thead>
<tr>
<th>Bell et al. factor</th>
<th>PANSS item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive symptoms</td>
<td>Unusual thoughts (G9), Delusions (P1), Suspiciousness (P6), Grandiosity (P5), Hallucinations (P3), Lack of Insight (G12)</td>
</tr>
<tr>
<td>Negative Symptoms</td>
<td>Passive withdrawal (N4), Emotional withdrawal (N2), Blunted affect (N1), Lack of spontaneity (N6), Poor rapport (N3), Motor retardation (G7), Active social avoidance (G16)</td>
</tr>
<tr>
<td>Hostility</td>
<td>Tension (G4), Stereotyped thinking (N7), Hostility (P7), Poor impulse control (G14), Uncooperativeness (G8), Excitement (P4)</td>
</tr>
<tr>
<td>Cognitive</td>
<td>Disturbance of volition (G13), Conceptual disorganization (P2), Poor attention (G11), Difficulty in abstraction (N5), Mannerisms and posturing (G5), Disorientation (G10)</td>
</tr>
<tr>
<td>Emotional discomfort</td>
<td>Preoccupation (G15), Somatic concern (G1), Anxiety (G2), Depression (G6), Guilt (G3)</td>
</tr>
</tbody>
</table>

2.8. **Data Analysis**

The Statistical Package for the Social Sciences (SPSS 19.0) was used to compute statistical analyses. All test measures were centered around their mean and graphed to check for normality prior to analysis. Raw scores for each of the measures were converted into age-corrected z-scores based on published or local normative
data. Paired-sample t-tests were performed for the QOL scale, and the SWL scale to assess improvement over time.

In stage 1, Pearson’s correlations were calculated between the change scores in objective (QOL) and subjective (SWL) quality of life. This assessed whether improvements in QOL were associated with improvements in SWL during rehabilitation treatment. Pearson’s correlations were also calculated for each of the neurocognitive and social cognitive variables at baseline and measures of change in objective and subjective psychosocial functioning (QOL, SWL).

In stage 2, two regressions models were tested using the baseline neurocognitive, social cognitive and symptom measures that were linked to change in objective QOL or SWL from stage 1. In step 1 of this model, baseline QOL or SWL measures were entered, along with group membership, so that the regression results would take into account any potential differential effect of the two cognitive rehabilitation programs. In step 2, measures of neurocognitive function linked to QOL or SWL were entered. In step 3, a measure of social cognition, the PEAT, was entered to the QOL regression (PEAT was omitted from SWL due to lack of significance). This step illuminates the unique contribution of social cognition to QOL beyond that of neurocognitive function. It also determines whether measures of social cognition mediate the relationship between cognitive function and change in objective QOL separate from neurocognitive function. If social cognition partially mediates the relationship between the neurocognitive variables and QOL: (a) measures of neurocognition would be related to QOL, (b) social cognitive function would be related to QOL, (c) social cognition would be related to neurocognitive
functioning, and (d) when social cognition is controlled for, the relationship between neurocognition and QOL would be statistically reduced or fully non significant (Barron and Kenney, 1986). In the final step of each regression, the interaction term for the main effects of the neurocognitive variables and social cognitive variables was entered to investigate whether social cognition moderated the relationship between cognitive function and QOL or SWL. All analyses were computed as two-tailed and set to an alpha level of .05.
3. Results

3.1. Effects of rehabilitation on objective QOL and subjective SWL

Participants improved on objective and subjective measures of quality of life at the end of the rehabilitation intervention (t(36) = 2.226, p < .05, and t(41) = 2.658, p < .05, respectively). There was a moderate relationship between improvement in QOL and SWL (r = .533, p < .01).

3.2. Relationship among neurocognition, social cognition, QOL, SWL, and symptoms

Correlations between standardized neurocognitive and social cognitive measures, and symptom ratings at study entry and change in objective QOL and subjective SWL after the rehabilitation intervention are presented in Table 2. The neurocognitive measure, CVLT trial 1z was positively related to QOL (r = .457, p < .01), and the social cognitive measure, PEATz was also positively related to QOL (r = .360, p < .05). While only CVLT trial 1z was positively related to SWL (r = .367, p < .05).

3.3. Associations between cognitive function and objective QOL and subjective SWL

The regression models for QOL and SWL are shown in table 3 and 4 respectively. When the neurocognitive measure correlated with change in QOL, CVLT trial 1z, was entered into a regression model, with group membership and baseline QOL scores controlled for in the first step, CVLT T1z produced an almost significant change in R² (.076, F = 8.964, p = .067). This means that CVLT T1z explained an additional 6.7% of the variance in QOL at the end of the trial beyond that associated with baseline QOL scores and group membership.
When the neurocognitive measure linked to change in SWL, CVLT T1z, was entered into a regression model, with group membership and baseline SWL scores controlled for in the first step, CVLT T1z was again produced an almost significant change in $R^2 (\cdot 053, F= 11.299, p=.068)$.

3.4. Social cognition as a mediator of the relationship between neurocognition and objective QOL

The correlation analyses revealed support for the prerequisite relationships between CVLT T1z, PEATz, and objective QOL for potential mediation. Mediation was tested with a block regression model (see Table 3, step 3). The regression shows that the relationship of CVLT T1z and QOL after rehabilitation becomes no longer significant when PEATz was added to the regression model. Since PEATz reduced the relationship between CVLT T1z and QOL, we can conclude that PEAT partially mediates the predictive relationship between CVLT T1z and change in QOL. A prerequisite relationship was not evident for the social cognitive measure and subjective SWL; therefore no tests for mediation were conducted.

3.5. Social cognition as a moderator of the relationship between neurocognition and objective QOL

Results from the regression analyses testing the moderating effects of social cognition on the relationship between neurocognition and QOL found no significant interaction ($p>.05$, see Table 3, step 4).
4. Discussion

The results of this study revealed five main findings: (1) consistent with hypotheses, improvements in objective quality of life across the treatment trial were associated with improvements in subjective life satisfaction, (2) consistent with hypotheses, a baseline measure of cognition, verbal memory, and a baseline measure of social cognition, facial affect recognition, were associated with improvement on measures of QOL related to intensive outpatient rehabilitation, (3) inconsistent with hypotheses, a baseline measure of cognition, verbal memory, was associated with improvement on measures of SWL after rehabilitation, (4) consistent with hypotheses, symptom assessment was not associated with improvement in QOL or SWL after rehabilitation, and (5) consistent with hypotheses, a baseline measure of social cognition, facial affect recognition, partially mediated the predictive relationship between verbal memory and improvements in QOL.

This is one of the first studies, to my knowledge, to evaluate the relationship of neurocognitive and social cognitive variables for predicting longitudinal improvement in objective QOL and subjective SWL in response to psychosocial and cognitive rehabilitation in schizophrenia. In this study, the predictive relationship between neurocognition measures and change in QOL and SWL is different from other findings comparing cross-sectional (Brekke et al., 2001; Narvaez et al., 2008; Tolman & Kurtz, In press), and longitudinal (Prouteau et al., 2005) relationships between neurocognition and objective and subjective QOL measures. Differences in the findings of the present study
compared to the only other longitudinal study comparing objective and
subjective quality of life may reflect the variation in life satisfaction measures or
be due to sample size differences. The study by Prouteau et al. (2005), used the
subjective measure Client’s Assessment of Strengths, Interests, and Goals
(CASIG) subscale for quality of life, which assesses items such as: finances, fun,
health, personal safety, housing, family, friends, and life in general. These items
may differ from those assessed in the Heinrich's QOL scale (Heinrichs et al.,
1984) and the Satisfaction with Life Scale (Test et al., 2005).

The results of the current study suggest that cross-sectional predictors of
subjective quality of life may differ from longitudinal predictors of the
effectiveness of rehabilitation interventions. The findings suggest that
neurocognition may influence the ability of patients to benefit from psychosocial
and cognitive remediation interventions in schizophrenia. Successful functional
outcome was measured by objective outcomes of psychosocial success and
subjective experiences of these successes, such as employment and number and
quality of social interactions, in this study.

The link between neurocognition, social cognition, and objective
measures of functional outcome in this study are consistent with emerging
literature in schizophrenia. Many studies have found evidence supporting the
rate-limiting role of baseline neurocognitive and social cognitive function in the
ability to predict benefits from behavioral rehabilitation programs in
schizophrenia (Brekke et al., 2007; Kurtz et al., 2008; Prouteau et al., 2005). A
small but growing body of literature suggests that social cognitive skills mediate
the relationship between elementary aspects of neurocognition and objective community function (Sergi et al., 2006; Vauth et al., 2004). The finding from this study that facial affect recognition partially mediates the predictive relationship between verbal memory and change in objective QOL adds to this list. This finding also extends the work by using a longitudinal design and studying these relationships in the context of an intervention. It suggests that the mediation reflects on the likelihood of patients to attain benefits from rehabilitation programs in schizophrenia.

The present study sheds light on which patient factors influence response to rehabilitation interventions, as well as how these factors influence this response. Our findings suggest that social cognition may be as essential to enhanced functional outcome as neurocognition after rehabilitation interventions in schizophrenia. These findings provide evidence for the types of patients most likely to benefit from current rehabilitation interventions. Novel interventions aimed to enhance neurocognitive and social cognitive variables prior to entry to the types of rehabilitation interventions studied in this report, may lead to heightened functional outcome measurements at the termination of these rehabilitation interventions.

Several caveats of the present study should be considered when interpreting the results. First, this study measured neurocognitive, social cognitive, and symptom variables at baseline and related them to change in objective and subjective quality of life after the treatment trial. Therefore, the mechanisms by which change in cognitive variables influences change in
objective and subjective quality of life remains to be explicated. Recent research suggests that these longitudinal relationships may be complex and very different from cross-sectional relationships (Fiszdon et al., 2008). Second, given the investigative nature of this study, coupled with the moderate sample size, multiple statistical comparisons were not corrected for and therefore the risk for Type I error increased. Third, in order to minimize the amount of statistical comparisons, demographic factors were not evaluated. This may be a limitation because previous studies have linked some demographic factors to different aspects of QOL (Caron, Mercier, Diaz, & Martin, 2005). Fourth, there are concerns pertaining to the specificity of cognitive deficits in schizophrenia and whether impairments on cognitive tests reflect global cognitive impairment (Dickinson, Ragland, Gold, & Gur, 2008). However, in the current study, links between verbal memory at baseline and change in QOL and SWL were somewhat specific as evidenced by their correlative relationships relative to other measured cognitive functions. Lastly, there was no control condition that did not receive psychosocial and cognitive remediation intervention in this study. Thus, it remains unclear to what degree the predictor variables are specific to the behavioral intervention studied in this report.
**Table 1**

Mean demographic and clinical characteristics of schizophrenia patients (n=44)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>30.75 (11.01)</td>
<td>19-56</td>
</tr>
<tr>
<td>Percent Male</td>
<td>72.7</td>
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</tr>
<tr>
<td>Education</td>
<td>12.23 (2.091)</td>
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<tr>
<td>Duration of Illness (years)</td>
<td>9.1 (9.443)</td>
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<tr>
<td>Number of hospitalizations</td>
<td>4.51 (4.728)</td>
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<td>Vocabulary Scaled Score (WAIS-III, IV)</td>
<td>8.37 (3.871)</td>
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<tr>
<td>Percent treated with atypical antipsychotic medication</td>
<td>97.7</td>
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</table>

WAIS= Wechsler Adult Intelligence Scale.
<table>
<thead>
<tr>
<th>Variable</th>
<th>DSSz</th>
<th>LNSSz</th>
<th>PCPTpz</th>
<th>CVLT1z</th>
<th>CVLT5z</th>
<th>PCETerrz</th>
<th>PEATz</th>
<th>QOL</th>
<th>SWL</th>
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<th>Host.</th>
<th>Pos.</th>
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<td>.058</td>
<td>.110</td>
<td>.108</td>
<td>.266</td>
<td>.175</td>
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</tbody>
</table>
Table 3

Multiple regressions for the prediction of objective QOL at the termination of the rehabilitation intervention (controlling for baseline QOL and group membership [cognitive remediation or computer skills]) by baseline measures of word learning and memory (CVLT trial 1z), facial affect recognition (PEATz), and the interaction of word recall and facial affect recognition.

<table>
<thead>
<tr>
<th>Step</th>
<th>Group Variables</th>
<th>Beta</th>
<th>Significance</th>
<th>F-value</th>
<th>R²</th>
<th>Change in R²</th>
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<td>QOL 1 Group ID</td>
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<td>8.964</td>
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Table 4

Multiple regressions for the prediction of subjective SWL at the termination of the rehabilitation intervention (controlling for baseline SWL and group membership [cognitive remediation or computer skills]) by baseline measures of word learning and memory (CVLT trial 1z).

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References


