SOCIAL COGNITION AND SOCIAL DISABILITY IN SCHIZOPHRENIA: THE ROLE OF EMOTIONAL INTELLIGENCE

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Abstract

Schizophrenia is a severe and persistent mental illness that results in substantial burden and disability for the individuals who suffer from it, their families, and society. Social disability, in particular, is one of the most crippling aspects of the disorder that dramatically limits functioning and quality of life. Recently, social-cognitive impairments in emotional intelligence have shown to be promising potential contributors to social disability in schizophrenia, and consequently might serve as effective targets for treatment. However, measurement in this area has been limited, and no study has examined the longitudinal relationship between emotional intelligence and social disability in schizophrenia within an experimental context. This study makes use of baseline and 1 year follow-up data from an outpatient sample of individuals in the early course of schizophrenia (n = 57 at baseline, n = 47 at year 1) participating in a randomized-controlled trial of Cognitive Enhancement Therapy to investigate the psychometric properties of a promising new measure of emotional intelligence, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT), and elucidate its longitudinal relations with social disability in this population. A comprehensive psychometric analysis was used to examine the reliability, discriminant validity, and factor structure of the instrument with individuals with schizophrenia; and general linear modeling, including hierarchical linear regression, was used to examine the

cross-sectional and longitudinal relations between MSCEIT performance and social disability after accounting for demographic, clinical, and cognitive confounds. Psychometric results generally supported the reliability and discriminant validity of the MSCEIT when applied to individuals with schizophrenia, but also revealed a potential shift in the latent factor structure of the instrument in this population. Analyses of relations with social disability indicated little to no cross-sectional associations between MSCEIT performance and social disability, and modest longitudinal associations between changes in these domains. In particular, significant relationships were observed between longitudinal improvements in emotion regulation and reductions in overall social disability (r = -.31) and household/family relationship problems (r = -.34). These relationships persisted after adjusting for demographic characteristics, neurocognitive function, and psychopathology. Tentative evidence from mediator analyses pointed to the possibility of longitudinal improvements in emotion regulation to serve as a mechanism by which Cognitive Enhancement Therapy achieves its beneficial effects on social disability, although reverse mediation could not be ruled out. Together these findings suggest that changes in emotion regulation may be uniquely associated with changes in social disability in schizophrenia. Future research will need to replicate these findings with larger and more heterogeneous samples, and focus on the development of additional measures to study broader domains of social cognition, beyond emotional intelligence, that may also bear relevance to social disability.

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PREFACE

Acknowledgments

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I. INTRODUCTION

A. OVERVIEW

Schizophrenia is a severe and persistent mental disorder that places significant burden on the individuals who suffer from it, as well as their families and society. The illness is defined by the presence of positive (i.e., hallucinations, delusions, disorganized thinking) and negative (i.e., poverty of speech, lack of motivation, flat affect) symptoms, affects approximately 1% of the population, and is considered to be one of the top 10 leading causes of disability in developed countries (Murray & Lopez, 1996; see Chapter 2 for a detailed description of schizophrenia). Many people with schizophrenia experience significant social disability, such that few individuals are able to work, interact effectively with others, sustain significant friendships and intimate relations, live independently, and ultimately achieve a level of quality of life that most would consider even minimally adequate (Thaker & Carpenter, 2001). To date, the factors that contribute to this social disability have remained largely elusive, as many individuals continue to experience poor social relationships even after the remission of the overt signs of psychosis. Consequently, little progress has been made in treating what people with the disorder experience as one of the most disabling aspects of schizophrenia.

Recent evidence indicates that one of the most likely contributors to social disability in schizophrenia is a range of deficits in emotion processing, including the ability to accurately perceive emotions, to use emotions to facilitate thinking and decision making, and to understand and manage emotions in oneself and others (Couture, Penn, & Roberts, 2006). Together, these emotion processing constructs are commonly referred to as emotional intelligence (Mayer & Salovey, 1997). Recently, exciting early research among persons with schizophrenia has begun to point to the importance of deficits in emotional intelligence to social disability in the disorder (e.g., Brekke, Kay, Lee, & Green, 2005; Mueser et al., 1996), however these investigations have

been remarkably limited to cross-sectional studies of individuals living on inpatient units that focus exclusively on only one aspect of emotional intelligence (the ability to accurately perceive emotion in others). Consequently, there is an important need for longitudinal studies that assess emotional intelligence more thoroughly and test its connection to the different domains of social disability among the more general population of persons with schizophrenia living in the community.

Emotional intelligence has been theorized to be a cognitive skill that is malleable over time. That is, with repeated practice people may be able to enhance their emotional intelligence (Mayer & Salovey, 1997). In fact, recent experimental evidence from Eack and colleagues (2007) has shown when given relevant cognitive treatment, persons with schizophrenia can learn to become more emotionally intelligent. The contribution of this dissertation is to extend our previous work showing that emotional intelligence can be improved in schizophrenia, by examining whether such individuals become less socially disabled as they make gains in their emotional intelligence. This research is conducted within the context of a randomized-controlled trial, but is not focused primarily on the effect of treatment on emotional intelligence or social disability, both of which have previously been documented in other studies (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007; Hogarty et al., 2004; Hogarty, Greenwald, & Eack, 2006). Rather, this research uses the experimental context to conduct a robust examination of the link between changes in emotional intelligence and changes in social disability. Such an investigation is particularly important because it begins to test a likely underlying mechanism (emotional intelligence) by which social disability can be reduced in schizophrenia, and therefore may serve to focus treatment development efforts aimed at reducing social disability among this population.

Using data from a 1 year randomized-controlled trial of Cognitive Enhancement Therapy

(CET; Hogarty & Greenwald, 2006) for persons with early course schizophrenia (n = 57 at baseline, n = 47 at 1 year follow-up) living in the community, this study investigates the relationship between emotional intelligence and social disability. This randomized-controlled trial is the first and only experimental treatment study to have documented treatment-related improvements in emotional intelligence among persons with schizophrenia. Specifically, the experimental treatment employed in this trial (CET) has been shown to produce reliable and dramatic improvements in emotional intelligence among persons with schizophrenia using a comprehensive, performance-based measure of emotional intelligence (the Mayer-Salovey-Caruso Emotional Intelligence Test [MSCEIT; Mayer, Salovey, Caruso, & Sitarenios, 2003]). Conversely, the active control condition of this randomized-controlled trial has been shown to produce no appreciable improvement in emotional intelligence among this population (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007). Consequently, this trial provides a unique opportunity to examine the longitudinal relationship between emotional intelligence (measured by the MSCEIT) and social disability in schizophrenia, due to the systematic variability introduced into emotional intelligence by the experimental treatment. This makes possible a particularly unique investigation of whether the changes in the emotional intelligence that occur during CET are related to systematic changes in social disability. The demonstration of such a relationship within this experimental context, after adjusting for potential confounders, would provide convincing evidence for a link between emotional intelligence and social disability, as well as point to a promising avenue for improving social disability among this population, through addressing deficits in emotional intelligence.

What follows is a brief introduction to the significance of social disability in schizophrenia, as well as an overview of the status of current research with regard to understanding the factors that contribute to social disability among this population that illustrates

the need for further research on the relationship between emotional intelligence and social disability in schizophrenia. The following section is only intended to serve as a broad introduction to the topic, as a greatly expanded review of the concepts and research discussed herein are provided in Chapter 2.

B. THE PROBLEM OF SOCIAL DISABILITY IN SCHIZOPHRENIA

Severe and persistent social disability is a key characteristic of schizophrenia that often plagues the lives of many individuals with the disorder. Psychiatrists as early as Emile Kraepelin (1919) described schizophrenia as being characterized by social withdrawal and a progressive deterioration in social functioning. Today's nosology of mental disorders considers the presence of significant social dysfunction to be a key and defining characteristic of all forms of schizophrenia (American Psychiatric Association, 2000). Such dysfunction is defined as the disruption or cessation of interpersonal relations and meaningful social activities that were present before the onset of the illness or are developmentally and culturally appropriate for a person's age (American Psychiatric Association, 2000). In fact, because individuals with schizophrenia tend to experience such severe social disability, early taxonomists have classified it with the autistic spectrum of illnesses (Bleuler, 1950), although recent taxonomies of mental disorders have separated autism and schizophrenia-spectrum illnesses (American Psychiatric Association, 2000).

Research over the past century has supported the conceptualization of schizophrenia not only as a disorder of psychosis, but also a disorder of social functioning. Early longitudinal studies of schizophrenia have consistently shown that such individuals often have prolonged periods of social dysfunction that are characterized by social withdrawal, a lack of close social relationships, and poor social skill, at both onset and years after the development of the illness

(Mueser & Tarrier, 1998). Such studies have indicated that during the course of schizophrenia, at least 70% of individuals will experience these symptoms of social deterioration (Harding, Brooks, Ashikaga, Strauss, 1987a; Harrow, Sands, Silverstein, & Goldberg, 1997; Strauss & Carpenter, 1974a), although some improvement does accrue over time (Harding, Brooks, Ashikaga, Strauss, 1987b; Harrow, Grossman, Jobe, & Herbener, 2005). For example, in a 25-year follow-up study of individuals with schizophrenia released from a long-term stay at a state hospital, Harding and colleagues found that as many as 75% of persons with schizophrenia showed signs of recovery (e.g., lack of positive symptoms, more frequent social interactions) from the disorder at 25-year follow-up (Harding et al., 1987b). Although recovery does not mean that individuals have been cured from schizophrenia, the documentation of a substantial proportion of persons who experience some level of recovery after experiencing decades of illness has also been bolstered by many personal accounts of living with schizophrenia, and ultimately focused research on facilitating and hastening improvements in functioning and quality of life among persons with the disorder. As such, while seemingly contradictory, evidence that people with schizophrenia do show some level of recovery has allowed treatment foci to move beyond symptom reduction and address the aspects of the illness that people with schizophrenia experience as most disabling, such as social disability.

Recent research following individuals in their first episode of schizophrenia throughout the initial stages of the illness has documented just how problematic social disability can be in the disorder. Robinson, Woerner, Mcmeniman, Mendelowitz, and Bilder (2004) followed a cohort of individuals with schizophrenia or schizoaffective disorder for five years after their first psychotic episode and found that fewer than 25% of persons experienced sustained periods of close interpersonal relations and frequent social contacts. Further, the remaining 75% of individuals had little to no contact with peers outside of family members, and were not able to

work or live independently. Another study by Ho, Nopoulos, Flaum, Arndt, and Andreasen (1998) found that when following a cohort of 50 individuals for two years after their first episode, 58% characterized their relations with friends as poor, and nearly half of the participants indicated that they had little to no enjoyment or participation in social leisure activities. Social network analyses of individuals with schizophrenia also show that such individuals tend to have fewer and more distant social relations than other psychiatric populations (Cohen & Sokolovsky, 1978; Hammer, Makiesky-barrow, & Gutwirth, 1978), and that their social networks tend to remain small and consist of few non-familial members over the course of the illness (Horan, Subotnik, Snyder, & Nuechterlein, 2006). Most importantly, a growing body of research has indicated that these areas of social disability are significant contributors to poor quality of life among persons with schizophrenia (Eack, Newhill, Anderson, & Rotondi, 2007; Ritsner, 2003; Ritsner et al., 2000). Consequently, extant research supports early observations that schizophrenia is frequently, though not always, characterized by severe social disability; and that when present, such disability significantly limits the recovery of persons with the illness.

Unfortunately, while the evidence is clear that social dysfunction is an important and common symptom of schizophrenia, the factors that contribute to social disability in schizophrenia have remained largely elusive. As noted above, emotional intelligence is a likely contributor to social disability in the illness, however a number of other factors have also been found to contribute to social disability. Gender has been one of the most notable predictors of social functioning among this population, as females tend to experience less social disability from the disorder, perhaps due to better premorbid adjustment, a later age of onset, or differences between males and females in the socialization process (Leung & Chue, 2000; Salokangas & Stengard, 1990). The highly noticeable and disturbing symptoms of psychosis would also seem to be a likely contributor to poor social functioning, however studies examining the contributions

of psychopathology to social dysfunction among this population have found only modest relations with the cardinal symptoms of the illness (e.g., hallucinations, delusions, lack of motivation, social anhedonia). Studies examining the impact of positive symptoms (i.e., hallucinations, delusions, thought disorder) on social functioning in schizophrenia have yielded mixed results, with some investigators finding small to moderate relationships between hallucinations, delusions, and social disability (e.g., Strauss & Carpenter, 1974a), and others reporting no significant relationships (e.g., Dickerson, Boronow, Ringel, & Parente, 1999). Investigations of the impact of negative symptoms (i.e., lack of motivation, poverty of speech, affective flattening, social anhedonia) on social disability have been more consistent, with several studies showing moderate and significant associations between deficit schizophrenia (defined by prominent negative symptoms) and poor social functioning (Addington & Addington, 2000; Dickerson et al., 1999). Nonetheless, gender and negative symptoms are far from accounting for all the variance in social functioning in schizophrenia, suggesting that there are other important factors conspiring against social recovery from the disorder. One particularly promising contributor to social disability in schizophrenia is the presence of social-cognitive deficits in emotional intelligence, or the ability to accurately perceive emotions, to use emotions to facilitate thinking and decision making, and to understand and manage emotions in oneself and others.

Over the past several decades, research has increasingly suggested that certain cognitive deficits may conspire against social recovery among individuals with schizophrenia (e.g., Hogarty & Flesher, 1999a). Much of the research investigating this issue has examined the correlations between neurocognitive deficits (e.g., problems in sustaining attention, making decisions, using/manipulating working memory) and behavior, and has largely focused on the inpatient behavior of more severely ill, male individuals (Green, 1996; Green, Kern, Braff, &

Mintz, 2000). Such research has indicated that many individuals with schizophrenia have profound deficits in neurocognition, particularly in working memory, (Barch, Csernansky, Conturo, & Snyder, 2002; Burglen et al., 2004; Silver, Feldman, Bilker, & Gur, 2003), and that these deficits are correlated with functional outcomes such as psychosocial skill acquisition (e.g., learning conversation skills), social problem-solving (e.g., resolving a conflict with a friend or family member), and social adjustment (e.g., adequacy of peer relations, engagement in social leisure activities) (Green, Kern, Braff, & Mintz, 2000). However, evidence concerning how deficits in neurocognition are "linked" to social functioning in schizophrenia (beyond simple correlations) has been limited (Green & Nuechterlein, 1999), and the relations between neurocognitive dysfunction and social functioning have not been particularly strong (Green, Kern, Braff, & Mintz, 2000). Furthermore, neurocognitive rehabilitation approaches that influence broad areas of social functioning have also been sparse, indicating that other higher-order cognitive factors, such as social-cognitive deficits in emotional intelligence, may be important contributors to social disability in schizophrenia (Addington & Addington, 1999, 2000; Milev, Ho, Arndt, & Andreasen, 2005; Reeder, Newton, Frangou, Wykes, & Reeder, 2004; Twamley, Jeste, & Bellack, 2003).

Recent research has suggested that deficits in *social cognition (*of which emotional intelligence is one component), or the ability to process and interpret socio-emotional information in oneself and others (e.g., taking the perspective of others, recognizing social cues, managing one's own emotions) (Newman, 2001), may indeed be an important cognitive factor limiting the recovery of individuals with schizophrenia (Pinkham, Penn, Perkins, & Lieberman, 2003). Such research has indicated that individuals with the illness have difficulty discerning emotions in faces (e.g., Mueser et al., 1996), recognizing and interpreting social cues (e.g., Corrigan & Green, 1993; Corrigan, Green, & Toomey, 1994), and predicting and inferring the

mental states of others (Brune, 2005b). The most consistent finding concerning the presence of any social-cognitive deficit in schizophrenia, is that such individuals have marked deficits in the processing of emotion, primarily within the domains of what has come to be known as emotional Intelligence (Mayer & Salovey, 1997). The domain of emotional intelligence is theorized to include such emotion processing abilities as emotion recognition (e.g., accurately recognizing emotions in faces) and management (e.g., inhibiting behavioral manifestations of negative emotions), as well as knowledge of the causes and meanings of different emotions (Mayer & Salovey, 1997; Salovey & Mayer, 1990). Unlike other intelligences, emotional intelligence is thought to be malleable and to develop throughout the lifespan (Mayer & Salovey, 1997). Individuals with schizophrenia have shown some level of impairment within all of these domains of emotional intelligence (Bechdolf, Schultze-Lutter, & Klosterkoetter, 2002; Mandal, Pandey, & Prasad, 1998), and recent neurobiological evidence suggests that selected cerebral pathways associated with emotional intelligence, such as the amygdala, are compromised among some individuals with schizophrenia (Pinkham et al., 2003). Unfortunately, while social-cognitive deficits in emotional intelligence appear to be present in schizophrenia, their functional significance has been less clear.

Relatively little research has examined the contributions of any deficit in social cognition to social disability and functional outcome in schizophrenia. Nonetheless, among the studies that have been conducted, the most promising social-cognitive contributor to social dysfunction in schizophrenia is deficits in emotional intelligence. A recent review of the relations between social-cognitive deficits and social disability in schizophrenia found that among the four most investigated social-cognitive constructs in schizophrenia research (i.e., emotional intelligence, social perception, theory of mind, and attributional style), only deficits in emotional intelligence showed consistent and moderate relations with nearly every examined indicator of social

functioning (i.e., in vivo social behavior, social skill, and community functioning or activities of daily living) (Couture, Penn, & Roberts, 2006). Conversely, theory of mind and attributional style were only related to community functioning and in vivo social behavior on the inpatient unit, respectively, although social perception was more broadly associated with in vivo social behavior, community functioning, and social problem-solving.

Work in this area has certainly highlighted the importance of emotional intelligence deficits to social functioning in schizophrenia. A compelling investigation by Penn, Spaulding, Reede, and Sullivan (1996) examined the relationships between emotion perception and the behavior of 27 persistently ill individuals with schizophrenia living on an inpatient ward. The investigators found that the ability to accurately perceive emotions in faces (i.e., emotion perception) was a significant predictor of more adaptive behaviors (e.g., neatness, interest in social interactions) on the inpatient unit. Further, they observed that measures of emotion perception and basic neurocognition shared little common variance, and that deficits in emotion perception made a unique contribution toward inhibiting socially appropriate behavior on the ward beyond deficits in neurocognition. Subsequent research on emotion perception among other individuals living on inpatient units has largely replicated and extended these results (see Couture, Penn, & Roberts, 2006 for review). For example, Mueser and colleagues (1996) examined 28 inpatients with schizophrenia and found that not only were deficits in emotion perception significantly related to poor social adjustment in ways similar to Penn et al.'s findings, but that such deficits were also moderately related to individual's level of social skill, suggesting that the acquisition of appropriate social skills may depend on intact emotional intelligence. Taken together, these findings highlight the significance of deficits in emotional intelligence to social disability in schizophrenia.

C. LIMITATIONS OF PREVIOUS RESEARCH

1. Few longitudinal investigations

While there has been research investigating the relationship between deficits in emotional intelligence and social dysfunction in individuals schizophrenia, such research continues suffer from several substantial limitations. First, to date examinations of emotional intelligence in schizophrenia have employed almost exclusively cross-sectional designs. In a recent review of studies examining the relationship between social-cognitive deficits and functional outcome in schizophrenia, only one study was found that examined the longitudinal contribution of emotional intelligence deficits to functional outcome (Couture, Penn, & Roberts, 2006). Brekke, Kay, Lee, and Green (2005) found that among 100 outpatients with schizophrenia, those who experienced more deficits in emotion perception at baseline were more likely to have impaired social functioning and less likely to be working and living independently at 1-year follow-up. Further, these relationships continued to ensue after accounting for shared variance with neurocognitive dysfunction. In a subsequent similar study, Brekke and colleagues (2007) followed 102 outpatients with schizophrenia for 1 year, and examined the relative contribution of initial neurocognitive and social-cognitive function to changes in functional outcome over the course of the study. Here too, they found that emotion perception was a significant predictor of longitudinal change in social and major role functioning.

Unfortunately, these investigations only assessed emotional intelligence deficits in emotion perception, leaving questions concerning the functional significance of broader deficits in emotional intelligence (e.g., understanding and managing emotions); and did not account for the likely contribution of symptomatology to social dysfunction. In addition, since both were naturalistic follow-up studies, no information can be gleaned with regard to the effect of treating emotional intelligence on social disability, which could provide a particularly strong test of the

relationship between emotional intelligence and social disability. Longitudinal, randomized-controlled treatment studies that can systematically improve emotional intelligence over time, and thus introduce systematic variability in the construct, are needed for such examinations. Such studies could provide a very powerful examination of the link between emotional intelligence and social disability, by allowing for the examination of the association between systematic *changes* in emotional intelligence and *changes* in social disability over time. To date, only one longitudinal treatment study exists that has known effects on the broad domains of emotional intelligence, and this research will be the first make use of a unique dataset from this treatment study to provide a rigorous test of the relationship between emotional intelligence and social disability among this population.

2. Little attention to related/confounding constructs

Second, most studies have failed to examine the unique contribution of emotional intelligence deficits to social disability in schizophrenia, while simultaneously accounting for the likely overlap between emotional intelligence, neurocognition, and psychopathology, as well as the known contribution of neurocognition and psychopathology to social functioning. Several investigations have suggested some overlap between social-cognitive and neurocognitive constructs (Sergi, Rassovsky, Nuechterlein, & Green, 2006; Vauth, Rusch, Wirtz, & Corrigan, 2004), as well as an interaction between social-cognitive deficits and some forms of psychopathology (e.g., paranoia; Blackwood, Howard, Bentall, & Murray, 2001). Additionally, a large body of evidence has accumulated pointing to the predictive utility of both neurocognitive deficits and some forms of psychopathology to social disability in schizophrenia (Addington & Addington, 1999, 2000; Green, 1996). To date, no longitudinal study has accounted for the shared variance among emotional intelligence, neurocognition, and psychopathology, when examining the contribution of deficits in emotional intelligence to social

disability among this population. Consequently, it continues to remain unclear whether such deficits have a unique and independent longitudinal relationship with social disability in schizophrenia.

3. Narrow focus on inpatient samples

Third, the majority of investigations examining the relationship between emotional intelligence and functional outcome in schizophrenia have overwhelmingly focused on inpatient samples (Couture, Penn, & Roberts, 2006). While such examinations provide important evidence concerning the psychopathological correlates of emotional intelligence deficits, they are less optimal for examining the functional correlates of emotional intelligence. Individuals living on inpatient units are by definition not working and unable to live independently, which removes a substantial amount of variance from a large component of measures of functional outcome. Such individuals are also frequently severely socially disabled, which restricts the range of social functioning among inpatients in a manner than could obscure correlational results (Pearson, 1903). Furthermore, as some studies have suggested (Eack & Newhill, 2007; van der Does, Dingemans, Linszen, Nugter, 1996), variability in such severe levels of social dysfunction may be largely attributable to exacerbations in symptomatology, whereas the social relations and functioning of individuals living in the community may have markedly different sources of influence. Additionally, with the introduction of atypical antipsychotic medications (which have yet to show effects on social cognition [Sergi et al., 2007a]), those residing on inpatient units represent a dwindling number of persons with schizophrenia, as most are now expected to achieve some level of subsistence in the community. Such a limitation questions the generalizability of emotional intelligence studies examining the behavior of inpatients to those living in the community, and highlights a need for future social-cognitive research among individuals with schizophrenia living in the community.

4. Limited examination of emotional intelligence constructs

Finally, studies of emotional intelligence in schizophrenia have focused primarily on emotion perception, and largely neglected the other relevant aspects of emotional intelligence (e.g., emotion management). This limitation appears to be attributable primarily to a lack of comprehensive measurement strategies in schizophrenia research to assess the domains of emotional intelligence (Green, Olivier, Crawley, Penn, & Silverstein, 2005), as few tests assessing aspects of emotional intelligence beyond emotion perception are routinely used in social-cognitive research among persons with schizophrenia, despite the relevance of these constructs to social and functional disabilities among this population. Such a measurement bottleneck has severely limited the field's understanding of deficits in the broader domains of emotional intelligence among those with schizophrenia, and the contribution of such deficits to social disability.

Recently, a promising performance-based measure of emotional intelligence, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; Mayer, Salovey, Caruso, & Sitarenios, 2003), has been developed, extensively validated among healthy individuals, and recommended as a key measure of social cognition in schizophrenia research by the National Institute of Mental Health Measurement and Treatment Research to Improve Cognition in Schizophrenia committee (Green, Olivier, Crawley, Penn, & Silverstein, 2005). The MSCEIT is an 8-task (with multiple items per task) instrument that assesses all hypothesized domains of emotional intelligence (Mayer & Salovey, 1997), which include the perception of emotion, understanding of emotion, use of emotion to facilitate cognition, and management of emotion. This instrument has been shown to successfully yield comprehensive assessments of emotional intelligence that are independent of both general intellectual ability and personality traits among healthy individuals, as well as among individuals with low emotional intelligence (Mayer, Salovey, &

Caruso, 2004). Unfortunately, the psychometric properties of this promising instrument have yet to be confirmed among those with schizophrenia. Based on the limitations of previous research and the considerable promise of this measure, this study sought to both confirm the well-established psychometric properties of the MSCEIT among persons with schizophrenia and examine the contribution of its assessment of emotional intelligence to social disability, in the context of a longitudinal clinical trial of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) for individuals in the early course of schizophrenia living in the community.

D. STUDY AIMS

This study aimed to conduct a longitudinal investigation of the relationship between deficits in emotional intelligence, as measured by the MSCEIT, and social disability in schizophrenia, using secondary data collected from an ongoing randomized clinical trial of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) for individuals living in the community with early course of schizophrenia (n = 57 at baseline, n = 47 followed-up at 1 year). This research was not focused on elucidating the treatment effects of this trial, but rather made use of its experimental context to conduct a robust examination of the link between emotional intelligence and social disability. The experimental cognitive rehabilitation treatment provided in this trial (CET) has already been shown to be highly effective at improving both emotional intelligence and social disability among persons with schizophrenia, and thus provides a unique dataset for examining whether changes in emotional intelligence are related to changes in social disability, by introducing systematic variability in these constructs over time. Using data from this trial, this research specifically aimed to:

Aim #1: Confirm the psychometric properties of the MSCEIT when applied to persons with

schizophrenia. Baseline data (n = 57) were used to confirm the internal consistency and discriminant validity (from neurocognitive function and psychopathology) of the MSCEIT. Additionally, given the modest sample size, an exploratory investigation of the factor structure of the instrument's 8 tasks was also conducted.

Aim #2: Examine the unique cross-sectional relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology. Baseline data (n = 57) were used to compute correlation matrices and hierarchical linear regression analyses to examine the zero-order and unique associations (beyond neurocognitive function and psychopathology) between emotional intelligence and social disability.

Aim #3: Examine the unique longitudinal association between changes in emotional intelligence and changes in social disability, above and beyond changes in neurocognitive function and psychopathology. A mediator-analytic framework for clinical trials was used to capitalize on the experimental design employed in this dataset, and complete follow-up data (n = 47) were used to examine the unique relationship between experimentally manipulated (i.e., treatment-induced) changes in MSCEIT scores and changes in social disability through a series of multiple regression analyses.

Taken together, the results of these aims are used to derive implications for future treatment development efforts for persons with schizophrenia. The analytic aims examined herein take an important step in identifying the significance of emotional intelligence deficits as a treatment target for schizophrenia. In the presence of significant relationships between emotional intelligence deficits and social disability, findings from this research can be directed toward novel and existing treatment efforts to enhance their effects on emotional intelligence deficits in an effort to reduce social disability among this population.

II. LITERATURE REVIEW

A study proposing to examine the contributions of deficits in emotional intelligence to social disability among persons with schizophrenia brings together a diverse body of multidisciplinary research from social work, psychology, and psychiatry. This chapter provides a review of the literature within and across these disciplines that highlight the significance of social disability in schizophrenia to persons with the disorder and broader society, as well as evidence pointing to the promise of the construct of emotional intelligence for understanding and remediating social disability in this population. This review begins by providing foundational information regarding the nature, social, and political significance of schizophrenia, and then examines the literature surrounding the presence of, and known contributors to, social disability among this population. The review then proceeds with a detailed analysis of the construct of emotional intelligence that highlights its promise for understanding social disability among persons with schizophrenia, by first providing an overview of the construct and evidence surrounding its significance to adaptive social functioning, and then moving into a critical examination of the evidence regarding the presence of deficits in emotional intelligence among persons with schizophrenia and the relationship between these deficits and social disability among this population. Finally, this review concludes with a brief discussion of the current issues regarding the measurement of emotional intelligence, which highlights the importance of confirming the psychometric properties of a promising measure of the construct among persons with schizophrenia.

A. OVERVIEW OF SCHIZOPHRENIA

Schizophrenia is a complex and disabling mental disorder that poses significant challenges to society, family members, and mental health professionals. The disorder has

progressed through various conceptualizations throughout history, and provoked both horrific and moralistic responses from society. Today schizophrenia is conceptualized largely as a biologically-based disorder of the brain that is often, but not always severe and persistent in nature, and currently has no known cure. The disorder is now widely recognized as a major public health concern due to its disabling effects and considerable cost to society and families. This section will provide a brief introduction to schizophrenia and its significance as a public mental health issue in the United States, as well as an overview of the evolution of social policies for persons with schizophrenia that bear direct relevance to this investigation and exemplify its significance to both science and society.

1. Description and Social Significance

Schizophrenia is a severe and persistent mental disorder that is frequently thought of as being composed of two broad symptom clusters. The first major symptom cluster is perhaps the most commonly recognized psychopathology attributed to schizophrenia, largely due to its easy perceptibility, and consists of what is today known as positive symptoms. These symptoms include hallucinations (experiencing sensory stimulation in the absence of a clear stimulus), delusions (persistent erroneous beliefs based upon false perceptions that are sustained despite incontrovertible evidence to the contrary), and/or prominent thought disorder (disorganized thinking and speech). The second major symptom cluster present in schizophrenia consists of negative symptoms. These symptoms, while less perceptible than positive symptoms, are nonetheless very disabling in terms of psychosocial functioning and include alogia (i.e., poverty of speech, low verbal expressiveness), affective flattening (i.e., reduced range/intensity of emotional expression), and avolition (i.e., lack of motivation, difficulty in engaging in goal directed behavior). While positive and negative symptoms frequently co-occur, negative symptoms tend to persist even when positive symptoms are adequately controlled with medicine.

According to the Diagnostic and Statistical Manual of Mental Disorders, the continuous presence of any two signs of positive and/or negative symptoms for at least a month, in conjunction with significant functional impairment for the past six months, warrants the consideration of a diagnosis of schizophrenia (American Psychiatric Association, 2000).

The onset of schizophrenia usually occurs during late adolescence or early adulthood, and once present, the disorder frequently (though not always) takes on a persistent course of recurrent acute positive symptom exacerbation (usually resulting in short-term psychiatric hospitalization) and persistent functional and social disability, even in the presence of adequate pharmacological treatment (Hogarty et al., 1974b). There are notable exceptions to this pattern however, as evidence has indicated that females tend to have a later age of onset, and a somewhat better prognosis from the disorder (Leung & Chue, 2000). In addition, persons with schizophrenia in developing countries have been shown to have better long-term outcomes than those in developed countries, perhaps due to wider social acceptance of those with persistent disabilities or cultural differences in stressors and life demands in developing countries (Hopper & Wanderling, 2000). Further, as individuals age, the acute exacerbations and positive symptoms of the disorder tend to remit, such that as many as two-thirds of individuals can meet broad definitions of recovery (e.g., remission of positive symptoms, increased social interactions) after being ill for several decades (Harding, Brooks, Ashikaga, Strauss, 1987b). However, such individuals are usually not considered "cured" from schizophrenia, as many still frequently experience lasting negative symptoms and disability in a variety of domains. Finally, of those who develop psychosis, a significant proportion recover relatively quickly (Harrow, Grossman, Jobe, & Herbener, 2005). Such individuals are sometimes classified as having schizophreniform disorder (psychosis without persistent disability), and may never go on to develop schizophrenia. It is estimated that approximately 1% of the U.S. population (2.2 million

people) is affected by schizophrenia at any given time point (American Psychiatric Association, 2000), with relatively equal prevalences between genders. The disorder has been shown to have a strong genetic component (Kendler & Diehl, 1993) and to be associated with a litany of neurobiological abnormalities (Shenton, Dickey, Frumin, & McCarley, 2001), although to date, no genetic or neurobiological marker has been found that is specific to schizophrenia.

Despite its relatively low prevalence rate, schizophrenia presents a major public health issue to both the United States and other countries throughout the world. Within the U.S., it is estimated that approximately 2.5% of all health care expenditures and 15% of all public funds available for the treatment of mental disorders, or 33 billion dollars, are used to care for individuals with schizophrenia annually (Dixon et al., 2001; Rupp & Keith, 1993), making it one of the most costly mental disorders in the world. Unfortunately, despite the high price of caring for individuals with schizophrenia, many continue to be almost completely functionally disabled from the disorder. It is estimated that nearly 10% of all individuals diagnosed with schizophrenia commit suicide (Freedman, 2003), ranking suicide as one of the leading causes of death for individuals with the disorder (Allebeck, 1989). For those who survive, systematic investigations of homelessness suggest that as much as 45% of people who are homeless also have schizophrenia (Folsom & Jeste, 2002; Susser, Lin, & Conover, 1991). For example, a study of an emergency homeless shelter in Boston found that 36% of individuals met diagnostic criteria for schizophrenia (Bassuk, Rubin, & Lauriat, 1984). Additionally, it is estimated that nearly 80% of individuals with schizophrenia are unable to work (Marwaha & Johnson, 2004). For example, a recent follow-up study of 313 individuals with schizophrenia by Mueser, Salyers, and Mueser (2001) found that over the course of 2 years, the percentage of individuals who maintained some form of competitive employment never rose above 25%, despite more than half of the individuals indicating that were interested in working. Furthermore, evidence throughout

the past century suggests that many (between 62% and 84%), though not all individuals with schizophrenia are unable to survive in the community without some form of professional assistance (Robinson, Woerner, Mcmeniman, Mendelowitz, & Bilder, 2004). As many as 60% of individuals with this disorder also have comorbid substance use problems, which significantly adds to the disability persons with schizophrenia experience (Fowler, Carr, Carter, & Lewin, 1998). Indeed, the high costs of the disorder coupled with its frequently severe and persistent nature has placed schizophrenia at the top of public mental health concerns (Thaker & Carpenter, 2001), and led to its ranking as one of the top 10 leading causes of disability in developed countries on the Global Burden of Disease list (Murray & Lopez, 1996).

In summary, schizophrenia often presents with a number of persistent social and vocational disabilities that place the disorder among the leading causes of disability, and make it a significant public mental health concern throughout the world. Current evidence suggests that these disabilities (particularly in social functioning) are seen despite dramatic improvements in positive symptomatology that have come from the recent introduction of ayptical antipsychotic agents (Leucht et al., 2003). Consequently, the effective treatment of these disabilities is at the forefront of the international schizophrenia research agenda, and continues to be a major focus of mental health policy and services throughout the U.S. and abroad.

2. Socio-Political Trends in Schizophrenia

As can be seen above, schizophrenia presents with a number of social and functional disabilities that make the disorder a significant public mental health problem throughout the world. Consequently, any investigation attempting to address these disabilities must be grounded in not only the scientific evidence-base, but also in the socio-political context that drives society's response to schizophrenia and a good proportion of its treatment. Social and political responses to schizophrenia have historically been driven by prevailing

conceptualizations of mental illness and its treatment, and have evolved from warehousing and inhumane practices to targeted psychological approaches and social policies that attempt to foster community integration and functioning. Today's social policies focus primarily on ensuring that individuals with schizophrenia can survive in the community and recovery some level of functioning and quality of life, which highlights the relevance of a study examining the contributors to social disability in this population to current mental health policies. This section briefly reviews the evolution of socio-political trends concerning the welfare (psychological and otherwise) of individuals with schizophrenia. This review is not intended to be a detailed analysis of U.S. mental health policy and its limitations, but rather provides an overview of the nature and historical context of current social policies attempting to address the disabilities that characterize the disorder, in order to delineate the significance and relevance of an investigation of social disability in schizophrenia to current social policy. It should be noted that these social policies generally affect individuals with severe and persistent mental illness in similar ways as they have affected those with schizophrenia, even though the focus here is on persons with schizophrenia.

Over the centuries, social welfare responses to the problem of schizophrenia have been influenced by dramatically shifting conceptualizations of the nature and etiopathology of the illness. Early conceptualizations were surprisingly scientific, with Hippocrates and other sixth century B.C. medical scholars suggesting that the source of mental illnesses could be located in humoral imbalances (Porter, 2002). Consequently, early social responses to schizophrenia focused on medical treatment to bring the humors into balance through dietary restriction or blood letting. Following the great thinkers of the sixth century and culminating with the rise of Christianity in the fourth century A.D., schizophrenia was thought to be the result of religious influences that centered primarily around morality, and schizophrenia in particular, was

sometimes thought of as a mental exemplar of immoral behavior. Such a conceptualization led to rather ghastly social welfare response to the disorder, usually through exorcism or death (Porter, 2002). Finally, with the rise of the Enlightenment in the early 18th century and the introduction of psychiatry, society's response to schizophrenia again returned to the medical model (Palha & Esteves, 1997). However, despite the re-medicalization of schizophrenia, in the absence of a clear understanding of its pathophysiology, treatment remained primarily exploratory and rarely effective (Goffman, 1961). The lack of efficacy of current treatments left many individuals with schizophrenia with significant functional and social disabilities. As such, the problem of schizophrenia in the 19th century was primarily the problem of the poor, and the social welfare solution was institutionalization. During this time, sustaining the poor in their homes was widely unpopular due to thoughts about work disincentives and pauperism. Consequently, individuals who could not sustain themselves in the community were required to go to the poorhouse, where they would be "cared" for and required to work if so able (Trattner, 1999). At the same time, there was no government policy for treating the mentally ill, and since many persons with schizophrenia were poor, they were subjected to the treatment of the poorhouse; a treatment which was widely feared by the poor and commonly known to be inhumane for the mentally ill (Trattner, 1999).

Seeing the abhorrent conditions of the poorhouse, in the mid-1800s Dorthea Dix argued for separate mental asylums sponsored by the federal government, and although her proposal was vetoed, states began erecting massive asylums to care for individuals with schizophrenia (Trattner, 1999). This became part of the "moral treatment" approach to schizophrenia, where the absence of effective treatment led to a palliative social welfare response to the disorder - institutionalization in an asylum (Grob, 1983). The basic premise of moral treatment was that insanity could be curred by caring for those with mentally illness in a humane and hospitable

manner (Grob, 1983). Such a notion was in stark contrast to the therapeutic nihilism that spread rampantly throughout Europe and the United States upon the formation of psychiatry, and was as much a result of a slight, but important change in conceptualizations of the nature of mental illness, as it was of the documentation of the horrific conditions of those with mental illness living in the poorhouse. In particular, the moral treatment movement brought with it the Enlightenment notions of mental illness being biologically based, however Lockean philosophy and other environmental conceptualizations of the etiology and management of schizophrenia became the cornerstone of the moral treatment movement. Such conceptualizations optimistically suggested that the "insane" could be cured by environmental modifications (Ozarin, 1954). When blended with the horrific conditions that individuals with mental illness experienced in poorhouse, environmental perspectives on the etiopathology of mental disorders led directly to the philosophy of the moral treatment approach: If individuals with mental illness are taken out of horrendous conditions and given kind and caring treatment, they could be cured. Such a philosophy paved the way for the construction of the first psychiatric institutions in the United States.

Unfortunately, as in the poorhouse, conditions rapidly deteriorated in the state asylum. The battle was uphill from the start, beginning with a federal reaffirmation that the poor, and particularly those with severe mental illness, were under the exclusive purview of the state (Trattner, 1999). The consequence of this reaffirmation and the subsequent century of social policy that followed was that if and when state resources for the asylum dwindled, the environmental conditions moral treatment advocates held so dear could become expendable. Without any federal support for state institutions, states asylums began to buckle under the pressure of raises in persons with severe mental illness that stemmed from widespread immigration to the United States and World War I (Trattner, 1999). Soon the environmental

conditions needed for moral treatment became untenable and many state institutions began to mirror the conditions of the poorhouse.

This state of affairs spurred the mental hygiene movement, another national wave of reform that would set the stage for deinstitutionalization and community care for persons with schizophrenia and other severe and persistent mental illnesses. Perhaps the most infamous documentation of the conditions of the asylum came from the founder of this movement, Clifford Beers. Beers, a consumer who had recovered from schizophrenia, in 1908 wrote an extensive critique of his experiences in the state asylum, calling attention to the inadequate and quickly deteriorating conditions of these institutions (Beers, 1908). In 1909, Beers founded the National Association of Mental Hygiene, which became an influential political body advocating for improved treatment and care of individuals with severe mental illness. One of the primary recommendations of this group was that individuals should be provided with outpatient care, and when released from the institution, provided with coordinated aftercare (Archer & Gruenberg, 1982). This group also was largely concerned with the prevention of mental illness, which was where they had their largest impact. This response, stemming from the ineffective treatments for those who already were ill, resulted in the widespread introduction of mental asylums for children, hoping to curb later chronic disability through early intervention. As a consequence, the mental hygiene movement became a significant driving force for documenting the inadequate treatment of individuals with schizophrenia and other severe mental illnesses in the state asylums, and laid the groundwork for moving treatment to community-based care and prevention.

With the introduction of the mental hygiene movement and incisive effect of its findings, several federal studies of the conditions of the asylum were conducted that largely supported Beers' observations (Grob, 1994). These studies, coupled with two world wars, the great

depression, and the discovery of phenothiazines began to swing social policies concerning America's treatment of the indigent mentally ill against institutionalization and favored more community-based approaches by the mid-20th century (Archer & Gruenberg, 1982; Grob, 1994). Severe mental illnesses, such as schizophrenia, became conceptualized as the byproduct of institutionalization - a notion which had little empirical support, but was largely based on observations of the conditions of the asylums and the fact that few individuals with severe mental illness ever got a chance to leave such institutions. As a consequence, in 1963 congress passed the Community Mental Health Centers Act to allow states to deinstitutionalize individuals with schizophrenia by providing federal funding to build local community mental health treatment centers (Goldman & Morrissey, 1985).

Despite all the optimism of community mental health, by 1970 it was becoming increasingly apparent that schizophrenia was a severe and persistent disorder that was not going to go away, even with the introduction of community treatment (Grob, 1994). The deinstitutionalization movement, which was deeply rooted in civil libertarian ideologies, did much to help improve the freedoms of the many individuals living in state hospitals whose civil liberties were unjustly revoked. Unfortunately, while many individuals with schizophrenia and other severe mental illnesses saw significant benefits from leaving wretched state institutions and regaining their freedom, a significant number of individuals who were deinstitutionalized continued to be very disabled in community. For many individuals, the years of institutionalization they endured made it increasingly difficult for them to acclimate to community living (Porter, 2002). Further, unlike the asylum, the community offered little to no material supports. While community mental health centers were intended to be the "safety nets" and primary service centers for individuals leaving state hospitals after deinstitutionalization, they were poorly funded and tended to cater to individuals already living in the community with

less severe problems. As with previous mental health policy movements, the funding stream dedicated to the asylum also did not follow the individuals who needed it as they transitioned to the community, and as a consequence, the community service infrastructure was greatly lacking (Goldman & Morrissey, 1985). In the absence of adequate material, psychological and community supports, family members became some of the primary carers for persons with schizophrenia and severe mental illness. In 1979, the Alliance for the Mentally Ill (later the National Alliance for the Mentally Ill [NAMI]) was formally erected to bring together the many family members who were experiencing great distress and burden from attempting to provide compassionate care for their loved ones, with little to no information on effective treatments. The group became a powerful political body that was in some ways instrumental in calling national attention to the plight of individuals with severe mental illness struggling to make it in the community.

From the political efforts of family members and federal initiatives studying the success of community care, it quickly became recognized that the provision of community supports was key to basic social and community functioning among this population (Grob, 1994). This realization led to the community support movement, which spurred a number of social welfare policies for individuals with severe mental illness, starting in the late 1960s and early 1970s with Social Security amendments to Medicaid and Supplemental Security Income that provided cash and in-kind benefits to individuals with schizophrenia living in the community (Cutler, Bevilacqua, & Mcfarland, 2003), and finally culminating with the introduction of Community Support Programs in 1977 to coordinate social welfare services for these individuals (Turner & Tenhoor, 1978). This represented an important shift in the treatment of schizophrenia, where social policy now recognized the requisite *resource* determinants of community functioning in this population.

Unfortunately, funding for the Community Support Programs was revoked three years after their introduction, and in the 1980s funding for community mental health centers was substantially reduced with the Omnibus Budget Reconciliation Act and the repeal of the Mental Health Systems Act. Consequently, community treatment for schizophrenia was placed in jeopardy as states were no longer able to sustain their community programs. The result was that by 1990, although schizophrenia was costing the public more than 20 billion dollars a year, such individuals continued to make up nearly one-third of the homeless population (Folsom & Jeste, 2002; Susser, Lin, & Conover, 1991), more than 80% were not able to work (Mueser, Salyers, & Mueser, 2001), and most were not able to survive in the community without some form of professional assistance (Robinson, Woerner, Mcmeniman, Mendelowitz, & Bilder, 2004).

Such a state of affairs prompted the introduction of the Alcohol, Drug Abuse, and Mental Health Administration Reorganization Act (ADAMHA Reorganization Act; P.L. 102-321) in 1992, which sought to formally reintroduce and expand the provisions of the Community Support Programs and related mental health policies, in an effort to improve social, vocational, and independent living outcomes among individuals with schizophrenia living in the community. The Act called for the introduction of a block grant mechanism to fund the further development of *comprehensive care programs* within the United States, which in large part echoed the temporary service provisions called for by the Community Support Programs. These provisions broadly included the use of case management services to help coordinate material and social welfare resources for individuals with severe mental illness, in order to ensure their survival in the community, as well as a mandate that states receiving grants also provide outreach services to homeless individuals, provide services to rural areas, and *develop detailed plans for how they will ensure consumers are able to function in the community.* Although over a decade has passed since the introduction of this policy, it continues to remain the most current major federal social

policy that focuses explicitly on the treatment of individuals with schizophrenia and other severe mental illnesses, as such policy issues have recently taken a backseat to mental health parity and related policies in the United States.

What can be seen from this brief review of the socio-political trends concerning schizophrenia in U.S. mental health policy is that ever since deinstitutionalization, social policies have focused on methods to ensure the ability of individuals with schizophrenia to function within the community. Unfortunately, these policies have all focused exclusively on the provision and coordination of community resources for individuals with schizophrenia, and largely ignored the psychological determinants of community functioning. As such, although current social policies, such as ADAMHA Reorganization Act, do much to help ensure that individuals with schizophrenia have the resources and supports they need to "survive" in the community, they do less to ensure that these individuals are able to lead successful and fulfilling lives outside the institution.

Recently, grass-roots initiatives that emphasize the importance of recovery and not just survival with a mental illness have begun to gain the attention of policy makers. The most notable of these initiatives is the recovery movement, which proposes to move beyond helping individuals with schizophrenia and other severe mental illnesses merely survive in the community, and focuses on the need to help such individuals lead satisfying and full lives (Anthony, 1993). While definitions of recovery have varied considerably, most include some notion that persons need to feel empowered and hopeful for a future that contains friends, lovers, meaningful activities, and other factors that lead to a satisfying quality of life (Substance Abuse and Mental Health Services Administration, 2004). This movement is undoubtedly a response, in part, to limited mental health policies, such as the ADAMHA Reorganization Act, that overwhelmingly conceptualize mental illness as an impersonal phenomenon and ignore the need

for individuals suffering from mental disorders to enjoy a full quality of life beyond symptom reduction and stable housing. Unfortunately, while the recovery movement has gained national attention by both policy makers and scientists, the degree to which recovery from schizophrenia is possible is still widely debated, as is the exact definition of the term recovery itself. Although the movement has instilled hope in many individuals, it has also fostered much controversy, and current mental health policy concerning the treatment of persons with severe mental illness has not been significantly expanded since the ADAMHA Reorganization Act. Rather, despite repeated evidence that individuals with schizophrenia need not only social welfare services (e.g., case management) to ensure their survival (Mueser, Bond, Drake, & Resnick, 1998), but also targeted psychosocial approaches to facilitate recovery by improving their ability to function in complex social situations and become productive members of society (Hogarty et al., 2004; Rosenheck, 2000), no social policy currently mandates the provision of such services. As a consequence, current social policies have had a limited impact on the community functioning and recovery of persons with schizophrenia, because they only attend to the basic, material needs of these individuals.

The omission of requisite psychosocial approaches designed to maximize recovery and social adjustment from mental health policy undoubtedly stems from the fact that the psychological determinants of social disability and community functioning in schizophrenia have remained largely elusive (Green, 1996; Green, Kern, Braff, & Mintz, 2000), which has restricted effective treatment options to improve functional outcomes among this population (Hogarty & Flesher, 1999a). Consequently, this investigation has the potential to make important contributions to address these limitations in current mental health policies by building an evidence-base that points to a potentially important psychological determinant of social disability in schizophrenia (emotional intelligence), and deriving treatment implications for addressing this

determinant to facilitate the recovery of persons with schizophrenia by improving social and functional outcomes among this population.

B. SOCIAL DISABILITY IN SCHIZOPHRENIA

As can be seen by the brief overview of schizophrenia provided above, the disorder is frequently characterized by a number of severe and persistent disabilities in several social and vocational arenas. This section will provide an overview of the evidence regarding the presence and significance of social disability in schizophrenia, as well as a comprehensive and critical review of the key factors currently known to affect social functioning among this population, which highlights the need for broader investigations of the factors that contribute to social disability among those who experience this illness. This review is presented with a justification of the relevance of continued investigations of psychosocial outcome in schizophrenia that exemplifies the important role of social work in this area, and the need for further examinations regarding the contributors to social disability among this population.

1. Relevance of Psychosocial Investigations

Schizophrenia is a disorder with a considerable, although somewhat unclear, biological basis. As such, the investigation of psychosocial factors in the course of the disorder has been considerably less of a focus of research than neurobiological and molecular studies. Nonetheless, the investigation of psychosocial treatments and outcomes in schizophrenia has made significant progress over the past several decades, much of which has come from social work, despite its somewhat marginalized presence in the research literature. Such investigations have broadly encompassed examinations of (1) the environmental and social factors associated with the onset and severity of the disorder, (2) various psychosocial interventions to address these factors, and (3) the influences of key psychosocial outcomes associated with schizophrenia

(e.g., social disability). The large number of social work researchers that have contributed to these investigations highlight the unique relevance of this area of study to social work, particularly with regard to the development of psychosocial treatments and empirical models of psychosocial outcomes.

While schizophrenia is known to have a substantial neurobiological basis (Lewis & Lieberman, 2000), research on the psychosocial factors associated with the onset and exacerbation of the illness has also highlighted the important role of the environment in the pathogenesis of the disorder. Investigations of environmental risk factors for the development and exacerbation of schizophrenia have all highlighted the important role environmental (particularly interpersonal) stress plays in the hastening of the relapse of psychotic symptoms. Such basic factors as perinatal trauma and maternal influenza have been shown to be consistent risk factors for the development of schizophrenia (Adams, 1993; Brown, 2006; Clarke, Harley, & Cannon, 2006). Furthermore, interpersonal stressors in the social environment have also been shown to be a dominant risk factor in symptom exacerbation and psychotic relapse (Nuechterlein et al., 1992). Perhaps the most widely studied area with regard to the role of interpersonal environmental stressors in the course of schizophrenia has focused on the family environment and the construct of expressed emotion or high degrees of criticism, expressions of hostility, and emotional over-involvement. A recent meta-analysis of such studies has indeed shown that individuals with schizophrenia living in highly critical family environments are nearly twice as likely to have a psychotic relapse as those living in environments with low levels of criticism (Butzlaff & Hooley, 1998). Such findings have been extended beyond the family to professional caregivers as well (Barrowclough et al., 2001; Heresco-Levy, Ermilov, Giltsinsky, Lichtenstein, & Blander, 1999), and suggested that individuals with schizophrenia are particularly susceptible to stress as a result of criticism, and that such stress plays a strong component in shaping the

course of the disorder (Wearden, Tarrier, Barrowclough, Zastowny, & Rahill, 2000). These and other findings concerning the prominent role of environmental stressors in the exacerbation of psychotic illnesses have led to the development and wide acceptance of a *stress-vulnerability* model of schizophrenia, which posits that the disorder consists of a biological susceptibility to stress that interacts with environmental stressors to make individuals vulnerable to psychotic illness (Zubin & Spring, 1977). Consequently, consistent with the biopsychosocial models of health and mental health in social work (Williams, Karls, & Wandrei, 1989), investigations of the role of psychosocial factors in schizophrenia have made important and substantial contributions to working models of disorder that move beyond biological determinism to a more accurate presentation of the complex nature of the illness.

The investigation of the role of psychosocial factors in the course of schizophrenia has not only enhanced the field's understanding of the nature of the disorder, but also paved the way for a series of psychosocial treatment development efforts, spearheaded primarily by social workers Carol M. Anderson and Gerard E. Hogarty, to address the environmental stressors that contribute to the exacerbation of psychosis. Based on early work with families and expressed emotion, Anderson and Hogarty developed a dual approach of family psychoeducation and patient social skills training for addressing criticism and stress within the family. The investigators posited that teaching family members about the nature of schizophrenia and the important role of stress in the disorder would demystify the illness and reduce critical attributions toward the affected relative (e.g., mistaking negative symptoms for laziness). Additionally, seeing that the affected relative has his/her own responsibility for facilitating harmony within the family environment, Hogarty and his group also sought to help persons with schizophrenia learn the social skills (e.g., table manners) they need to promote a healthy interpersonal environment within the home. The result of this dual intervention was a dramatic success, with no individuals

relapsing within the first year (as opposed to the traditional 50% with adequate prophylactic medication [Hogarty et al., 1974a]) who received family psychoeducation and social skills training combined (Anderson, Reiss, & Hogarty, 1986). This landmark study illustrated not only the importance of environmental stress in the course of schizophrenia, but also the ability of psychosocial approaches to effectively address these stressors and produce favorable effects on the course of the disorder. Since its publication, countless other family approaches to the treatment of schizophrenia have been developed (e.g., Falloon et al., 1985; McFarlane et al., 1995), along with personal approaches to teach individuals with schizophrenia their own stress management techniques (e.g., Hogarty, 2002), all of which have been shown to be very effective when combined with appropriate pharmacological treatments (Dixon, Adams, & Lucksted, 2000; Hogarty et al., 1997a; Tarrier et al., 1993).

Investigations of psychosocial factors in the course of schizophrenia have focused not only on psychosocial predictors of illness exacerbation and their treatment, but have also increasingly attended to the importance of *psychosocial outcomes*, such as social and vocational functioning, in the course of the disorder. As efforts to develop both novel pharmacological strategies to reduce patient's biological susceptibility to stress and psychosocial approaches to reduce environmental stress have successfully ensued, many individuals with schizophrenia have gained great control over the positive symptoms of the illness (Leucht et al., 2003; Mueser et al., 2002). While some evidence has also indicated very modest improvements in social functioning following the introduction of atypical antipsychotic medications (Swartz et al., 2007), many individuals with schizophrenia continue to remain functionally and socially disabled, despite the remission of positive symptoms that can come from these pharmacological advances. Many (though not all) individuals with schizophrenia have been repeatedly noted to have limited vocational capacity (Marwaha & Johnson, 2004), few significant relationships outside the family

(Horan, Subotnik, Snyder, & Nuechterlein, 2006), small social networks (Cohen & Sokolovsky, 1978), and poor social skills (Liberman, 1982), all of which have led to a concentrated focus on the predictors and treatment of these psychosocial outcomes (Brekke, Kay, Lee, & Green, 2005; Carter, 2006). To date, treatment strategies have largely focused on behavioral approaches to improve social skill (Smith, Bellack, & Liberman, 1996), with limited effects on broader dimensions of psychosocial outcomes (e.g., vocational functioning, interpersonal relations) and the generalization of social skills to "real world" settings (Dilk & Bond, 1996; Hogarty et al., 1991). Investigations of predictors of psychosocial outcome in schizophrenia have largely highlighted the important contribution of negative symptoms (Addington & Addington, 1999), and more recently begun to suggest the importance of deficits in basic neurocognition (Green, 1996). However, the contributions of these domains have not been particularly strong, suggesting additional contributory factors to impaired psychosocial functioning in schizophrenia. Consequently, an important task of future psychosocial investigations in schizophrenia research is to further elucidate the contributors to poor psychosocial outcome and direct such findings to novel treatment development efforts. This is an area particularly well suited for social work research, as the profession has had a long history in leading both psychosocial treatment development efforts (e.g., Anderson, Reiss, & Hogarty, 1986; Hogarty, 2002; Stein & Test, 1980) and investigations of psychosocial outcome (e.g., Brekke, Kay, Lee, & Green, 2005) among this population. Further, the results of research in this area are also likely to be particularly important to practicing social workers, who provide the majority of direct mental health services to persons with schizophrenia (Substance Abuse and Mental Health Services Administration, 2001).

As can be seen by this brief overview of psychosocial investigations in the course of schizophrenia, such investigations have been an important part of social work research and added

substantially to the field's understanding of the pathogenesis of the disorder and to the development of effective psychosocial treatments. However, despite these advances, many individuals with schizophrenia continue to exhibit marked deficiencies in a number of psychosocial domains, particularly social functioning, which has pointed to the importance of future investigations that focus on the factors that contribute to poor psychosocial outcomes. What follows is a review of the literature concerning the presence and degree of social disability experienced by persons with schizophrenia. This review is then supplemented with a critical examination regarding the current state of knowledge surrounding the factors known to contribute to social disability in schizophrenia.

2. Presence of Social Disability

Defining social disability. Before embarking on a review of the presence and significance of social disability in schizophrenia, it is first important to define the construct that will be the focus of this review. Social disability has been defined in many different ways, with some definitions focusing on the quality of interpersonal relations and others focusing on quantity of interpersonal relations (Mueser & Tarrier, 1998). Within schizophrenia research, definitions frequently include statements about impairments in interpersonal functioning and social skill, the relative absence of friends and significant others, and the poor quality of existing social relationships (Birchwood, 1990; Liberman, 1982; Schooler, Weissman, & Hogarty, 1979; Wiersma et al., 2000). All of these aspects reflect either deficits in the quantity or quality of social relations and functioning, and address components of classical definitions of social disability (e.g., Nagi, 1976). This research will build from these broader definitions of social disability and define the construct as consisting of three interrelated domains: (1) impaired social skill, (2) diminished quantity of interpersonal relations/social network size, and (3) diminished quality of interpersonal relations. The presence of these different domains of social disability

among individuals with schizophrenia is reviewed separately to outline the range of social impairments experienced by some members of this population.

Social skill impairments. Perhaps the most commonly investigated area of social disability in schizophrenia concerns impairments in social skill. Research in this area has repeatedly highlighted the prevalence of substantial and enduring social skills deficits among many persons with schizophrenia compared to both psychiatric and healthy controls. Early work by Bellack, Liberman, and other skills training research groups had observed the prevalence of social skill deficits among persons with schizophrenia for several decades, and developed training programs to address these deficits (Smith, Bellack, & Liberman, 1996). Unfortunately, the observations of these investigators went undocumented for some time, as adequate measurement had been lacking for many years in the precise assessment of social skills (Bellack, 1983).

Within the past decade, some measurement limitations in social skill assessments have been overcome, and investigations of the prevalence and nature of social skill impairments among individuals with schizophrenia have ensued. In a 1-year follow-up study, Mueser, Bellack, Douglas, and Morrison (1991) examined the prevalence and stability of social skill deficits among of 36 individuals with schizophrenia admitted to an inpatient unit, using a series of role play tasks to test conversation and assertiveness skills. They found that 67% of individuals showed social skill deficits that were worse than the worst healthy control participant at baseline, and that 64% of individuals continued to exhibit such deficits at 1-year follow-up. In another follow-up study by Bellack, Morrison, Mueser, Wade, and Sayer (1990), 37 individuals with schizophrenia living on acute inpatient units were assessed on various social skill domains at study intake, and six months later. Similar to the findings of Mueser et al., the researchers again found that many individuals with schizophrenia showed significant impairments in social

skill, compared to healthy controls, and that no significant improvement in social skill occurred during the course of the six month study. Investigations of social skill deficits from other research groups have largely found similar results (Smith, Bellack, & Liberman, 1996). For example, using a comprehensive role-play assessment social and life skills, Patterson, Goldman, McKibbin, Hughs, and Jeste (2001) examined the communication skills of 50 individuals with schizophrenia and 20 healthy controls. Despite using a new measure of social skill not previously adopted by the predominant social skills investigators in schizophrenia research, and examining social skill among schizophrenia outpatients, Patterson et al. largely replicated the findings of previous research, by indicating significant impairments in communication skills among individuals with schizophrenia, compared to healthy controls. All of these findings seem to suggest that social skill deficits are both present and stable among a substantial proportion of persons with schizophrenia, compared to healthy controls.

Investigations of social skill deficits among individuals with schizophrenia have also indicated that not only are these deficits common in comparison to healthy individuals, but that they are also present when compared to individuals with other psychiatric disabilities. One study by Bellack (1990) examined social skill deficits among 58 individuals with schizophrenia, compared to 33 affective disordered patients, and 20 healthy controls. While the individuals with an affective disorder did display some deficits in social skill compared to healthy controls, those with schizophrenia exhibited significantly greater deficits in nearly every domain of social skill assessed than both affective disordered patients and healthy controls. Another study by Mueser, Bellack, Morrison, and Wixted (1990) examined social skill impairments using a role play test of conversational and assertiveness skills among 57 individuals with schizophrenia, 16 individuals with schizoaffective disorder, and 33 individuals with pure affective disorders. Similar to Bellack's findings, the researchers found that individuals with schizophrenia tended to

have the largest impairment in social skill, followed by those with schizoaffective and affective disorders, respectively. In conjunction with investigations comparing social skill deficits in schizophrenia to healthy controls, these findings suggest that not only are social skill impairments frequently present among persons with schizophrenia, but when present, they appear to be more severe than in other psychiatric conditions.

Small social networks. Another area that has received some attention from investigations of social disability in schizophrenia concerns the quantity of social relationships maintained by persons with schizophrenia. This research has taken on the sociological tradition of social network analysis (Wasserman & Faust, 1994), and has suggested limitations in the nature and size of the social networks of some persons with schizophrenia. In an early social network analysis of persons with schizophrenia, Cohen and Sokolovsky (1978) studied the social networks of recently discharged individuals living in a hotel in New York city. They observed that while individuals with schizophrenia rarely were completely isolated, such individuals frequently had significantly fewer social network linkages than non-psychotic persons. Further, the presence of smaller social networks was observed to be a significant predictor of psychiatric relapse among persons with schizophrenia, even when accounting for psychopathology. A more recent examination of social network size among 120 outpatients with schizophrenia by Bengtsson-Tops and Hansson (2001) found that not only did patients have significantly smaller social networks than controls, but that nearly half of all individuals with schizophrenia surveyed indicated a desire for more access to social contacts. Another study by Dozier, Harris, and Bergman (1987) examined both the size and density of the social networks of 30 individuals with schizophrenia or other severe mental illnesses living in Washington, D.C. They calculated network density as a function of the number linkages between network members and the total size of the social network. The investigators found that among their sample of individuals with

schizophrenia and severe mental illness, the average network size consisted of 16 members, only half of whom knew each other, suggesting that some persons with schizophrenia have modest and poorly connected social networks. Other studies of social network size and density among individuals with schizophrenia have largely reported similar findings, all of which point the presence of small and poorly connected social networks among some members of this population (Mueser & Tarrier, 1998).

While these early studies have tended to suggest that persons with schizophrenia have smaller and less connected social networks than healthy individuals and persons with other psychiatric disabilities, more recent investigations have elaborated on limitations in the nature of the social networks of some persons with schizophrenia. One such study by Hansson et al. (2002) examined the perceived availability of social network supports in a multi-site European investigation of social networks and support among 418 individuals with schizophrenia. The investigators found that regardless of living situation, individuals with schizophrenia on average rated the availability and adequacy of access to social support and integration through their network as poor to fair, lending support to earlier work documenting the lack of connectedness that characterizes the social networks of many individuals with schizophrenia. Another study by Semple et al. (1997) examined the social network structures of 66 persons with schizophrenia and compared them to healthy control participants. These investigators found that the social networks of persons with schizophrenia contained significantly fewer friends, offspring, and intimate relationships than those of healthy controls. Surprisingly, however, the familial networks of persons with schizophrenia did not differ significantly from healthy participants with regard to frequency of contact, support received, or geographic proximity. Consequently, this study seems to suggest that the social network deficit in schizophrenia may be specific to non-familial social relationships. A recent comprehensive analysis of the nature of social networks

among 89 persons with schizophrenia by Horan et al. (2006) largely echoed these results, where family members tended to account for 64% of members in persons' social network, again suggesting adequate (if not slightly excessive) connections with family members among individuals with the illness. In addition, Horan and colleagues found reciprocity within these social networks to be slightly skewed toward unidirectional support provided to the patient. Taken together, these findings suggest that not only do some persons with schizophrenia have social networks that are limited in size and connectedness, but these networks are frequently overly represented by family members, and absent of significant non-familial friendships and intimate partners.

Impoverished quality of relationships. A final area that has received considerable attention with regard to social disability in schizophrenia concerns the quality of the interpersonal relationships of individuals with the illness. While literature surrounding the social networks of this population has suggested diminished network sizes and poor interconnectedness, research on the quality of interpersonal relations among individuals with schizophrenia has suggested that the few non-familial relationships these individuals have at times characterized by infrequent and superficial contact. Research in this area of social disability among individuals with schizophrenia has had a long and illustrious history, much of which began with early longitudinal investigations of outcomes among this population. For example, Harding and colleagues (1987a) reported on a 32-year investigation of individuals with schizophrenia and other severe mental illnesses who had been discharged to the community from state hospitals in Vermont. This study provided an ideal opportunity to examine the prevalence and course of social disability among persons with schizophrenia living in community settings. The investigators found that for the first decade after hospital discharge, individuals often had infrequent social contact with friends outside of family members, and tended to limit their

"friendships" to other patients and staff at the community mental health center. At the end of their 10-year analysis of social disability among these persons, Harding and colleagues concluded that at least 70% of patients experienced significant social disability, as evidenced by an impoverished quality of interpersonal relations. In a subsequent 25-year follow-up of these individuals, Harding and colleagues (1987b) found continued, although somewhat diminished, evidence of social disability among persons diagnosed with schizophrenia, such that nearly 40% of individuals still reported the absence of any friendships that they considered close and that were characterized by at least weekly interactions.

These concerning findings regarding the quality of interpersonal relations among individuals with schizophrenia have not been unique the work of Harding and colleagues, but rather have been a consistent finding in both domestic and international studies of the long-term course and outcome of schizophrenia (e.g., Carpenter & Strauss, 1991; Harrow, Grossman, Jobe, & Herbener, 2005; Strauss & Carpenter, 1974a). For example, a 15-year domestic multi-year follow-up study by Harrow and colleagues found that individuals diagnosed with schizophrenia consistently exhibited the poorest level of social functioning compared to individuals with other psychotic and non-psychotic disorders. In fact even after over 15 years of having schizophrenia, few individuals ever reached the highest quartile of social functioning on the Strauss-Carpenter outcome scale, a well-established measure of the quality of interpersonal relations in schizophrenia (Strauss & Carpenter, 1972), and less than 20% could be considered as achieving a functional and social recovery from the disorder. These quantitative findings are buttressed by more recent qualitative examinations of the social lives of persons with schizophrenia. In a unique qualitative investigation of first-person accounts of social disability in schizophrenia, Davidson & Stayner (1997) found that such accounts were characterized by a sense of loneliness and loss, and a strong desire for companionship and love, pointing to a very real and compelling

sense of social disability as experienced first-hand by persons with schizophrenia. Another study by Angell (2003) examined the social relationships of 20 individuals with schizophrenia in an Assertive Community Treatment team through the use of structured interviews, and found that not only were the social networks of persons with schizophrenia heavily dominated by mental health staff and fellow consumers, but that patients clearly recognized and were dissatisfied with this characteristic of their social networks, exclaiming that they wanted more friendships with people who did not already have "their own problems." Such relationships were often perceived as being both superficial and characterized by cold and disabled social interactions. Taken together, both quantitative and qualitative evidence regarding social disability in schizophrenia suggests that in addition to the presence of social networks of limited size, the relationships within these networks tend to be of limited quality and frequently perceived as superficial by a number of persons with schizophrenia.

3. Known Contributors to Social Disability

Significant progress has been made over the past several decades not only with regard to understanding the nature and significance of social disability in schizophrenia, but also in elucidating the potential contributors to such disability. This research has primarily examined the contributions of gender, psychopathology and cognition to social dysfunction in schizophrenia. Unfortunately, while some of these demographic and clinical characteristics have shown consistent relations with social disability, a critical examination of this literature would suggest much is still unknown about the factors that contribute to social disability in schizophrenia.

Gender. An area of investigation that is not a common focus of recent research on psychosocial outcome in schizophrenia, but has very real relevance to such inquiries is the well-known relationship between gender and outcome among this population. A recent review of the

sex differences literature in schizophrenia research concluded that there are important and significant differences between males and females in the course and prognosis of the disorder (Leung & Chue, 2000). While differences have shown up in a wide array of outcomes, such as vocational functioning (Childers & Harding, 1990), independent living and community adjustment (Wattie & Kedward, 1985), frequency of rehospitalization (Goldstein, 1988), and psychopathology (Shtasel, Gur, Gallacher, Heimberg, & Gur, 1992), sex differences with regard to social disability and functioning have also been an important focus of this research. It has been hypothesized that since women often have a later onset of schizophrenia, they have been able to reach more social milestones and achieve better premorbid social functioning than those who develop schizophrenia earlier in life, which in turn leads to a better prognosis and limits social deterioration associated with the disorder (Leung & Chue, 2000). Also, some individuals have suggested that the socialization process for women, at least in Western cultures, focuses more on the early learning of important relationship and social skills, which can also limit the impact of schizophrenia development on social disability (Riecher-Rossler & Hafner, 2000).

Perhaps the most consistent findings regarding gender differences in social disability among persons with schizophrenia concern rates of heterosexual and interpersonal relations (Leung & Chue, 2000). Studies of heterosexual relations among persons schizophrenia have found substantial differences by gender in both cross-sectional and longitudinal examinations. One study of gender differences in schizophrenia by Salokangas (1983) found that while only small differences were noted in psychopathology and clinical symptoms, significant differences were found favoring women with regard to social adjustment among 175 individuals with schizophrenia followed-up over the course of 8 years. In particular, it was noted that women were significantly more likely to be married at follow-up than men. In a cross-sectional study of 85 individuals with schizophrenia living in the community, Andia and colleagues (1995) found

that while only 9% of men were married, 38% of women were married, and that these differences could not be accounted for by differences in symptom severity. Another long-term follow-up study by Wattie and Kedward (1985) found that when following 182 persons with schizophrenia over the course of 10 years, women were 2.5 times more likely to be married than men, suggesting that outcomes in heterosexual relations are significantly more favorable among women with schizophrenia.

Studies have also examined gender differences in general social relations among persons with schizophrenia, and some have found that here too women tend to be less socially disabled than men. For example, McGlashan and Bardenstein (1990) found that when following 163 individuals with schizophrenia over the course of 15 years, women tended to have more frequent social contacts than men, although who these contacts were with were not specified. However, not all studies have shown a positive effect for women in social relations. For example, Test, Burke, and Wallisch (1990) found that while females in their sample of persons with schizophrenia were significantly more likely to be married and engage in heterosexual relations over the course of a 2-year study period, no significant differences were found with regard to frequency of peer contact. Indeed, reviews of this literature show that findings regarding general social relations among persons with schizophrenia vary considerably, with about half of the studies favoring women, and half showing no differences between the sexes (Angermeyer, Kuhn, & Goldstein, 1990). Further, since most studies have commingled sexual and non-sexual social relations in their analysis, it is not clear whether the positive effects for women in these investigations merely represent the well-documented gender difference in heterosexual relations, as seen in Test et al.'s work. Consequently, evidence concerning gender differences in non-sexual relations among persons with schizophrenia is largely mixed.

Few studies have examined the gender differences and social disability in schizophrenia

with regard to social skill or social networks. The only known study to date that has examined gender differences in social skill was published in 1990 by Mueser and colleagues. They found that women with schizophrenia consistently displayed significantly better social skills than men during the course of a longitudinal 1 year investigation, but that this improved social skill was not associated with significant gender differences in general social adjustment over the course of study (Mueser, Bellack, Morrison, & Wade, 1990). Surprisingly, while these findings argue for gender differences in social skill, they do not support this difference as a mechanism of overall gender differences in social disability. Rather, gender differences within the domains of interpersonal relations and social skill appear to be surprisingly independent. Studies examining gender differences with regard social network size or characteristics among persons with schizophrenia have also been limited. Test et al.'s (1990) found that there were no significant gender differences in number of close friends within the social networks of 122 young adults with schizophrenia followed-up over the course of 2 years. Similarly, Bengtsson-Tops and Hansson (2001) found no appreciable differences between sexes in access to or satisfaction with social contacts in the social networks of 120 individuals with schizophrenia living in the community. Thus, while evidence regarding gender differences in social network characteristics among persons with schizophrenia is notably limited, prominent differential effects yet to be consistently reported.

In summary, the extant literature on gender differences with regard to social disability in schizophrenia suggests that women have more favorable sexual social adjustment than men. Further, some evidence suggests that women may also have better premorbid social adjustment with regard to general social relationships as well; however it remains unclear whether this is primarily due to improved sexual relations. Evidence regarding differences in actual social competence is lacking, with only one study to date suggesting that women with schizophrenia

may possess better social skills than men, and the few studies examining social networks among this population have not found appreciable differences. Thus, the gender effect with regard to social disability in schizophrenia appears to be largely circumscribed to heterosexual adjustment.

Psychopathology. Perhaps the largest body of clinical research examining the factors that are associated with social disability in schizophrenia has centered on investigations of the relations with prominent psychopathology that frequently characterizes the disorder (i.e., positive and negative symptoms). Investigations of the relationship between positive symptoms and social dysfunction in schizophrenia has generally yielded small to non-significant relationships. With regard to social skill impairments, Mueser, Bellack, Morrison, and Wixted (1990) examined the relationship between positive symptoms and social skill performance among 45 inpatients with schizophrenia. They found that positive symptoms were only modestly, and not significantly related to social skills among men (r = -.07) and women (r = -.26) with schizophrenia. Another study by Mueser et al. (1991) examined the relations between changes in social skill and changes in positive symptomatology among 36 persons with schizophrenia living on an inpatient unit. These investigators found that no significant relationships existed between changes in positive symptoms and changes in social skill across a 1-year follow-up. Finally, Macdonald, Jackson, Hayes, Baglioni, and Madden (1998) found small, non-significant relationships between social skills and positive symptoms (r = -.08) in a study of 45 outpatients with schizophrenia.

Studies examining broader areas of social functioning than mere social skill have largely yielded similar results. For example, in a cross-sectional examination of the symptom correlates of social disability in schizophrenia, Smith et al. (1999) examined the relationship between positive symptoms and social functioning among 46 outpatients with schizophrenia. The investigators found a non-significant, small relationship between positive symptoms and

interpersonal functioning (r = -.08). Another study by Norman et al. (1999) examined the symptom predictors of social functioning in a longitudinal study of 50 outpatients with schizophrenia. They also found modest, although statistically significant relations between positive symptoms and interpersonal functioning (r = -.35) and social contact (r = -.32). In addition, Macdonald et al. (1998) examined the relationship between social network size and positive symptoms among 45 outpatients with schizophrenia. Here too, they found a modest and non-significant relationship between positive symptoms and social network size (r = -.20). Other investigations of both social networks and broader social adjustment among persons with schizophrenia have yielded consistent results (Breier, Schreiber, Dyer, & Pickar, 1991; Hamilton, Ponzoha, Cutler, & Weigel, 1989; van der Does, Dingemans, Linszen, Nugter, 1996). Consequently, these findings seem to suggest that positive symptoms surprisingly bare little relation to social functioning among persons with schizophrenia. While these results may represent attenuated correlations due to restrictions in range of social functioning among more disabled individuals, they are consistent with evidence from experimental trials of antipsychotic medications showing significant reductions in positive symptomatology, but not social disability (Leucht et al., 2003; Swartz et al., 2007).

The same studies that have examined the relationship between positive symptomatology and social disability among persons with schizophrenia have also frequently assessed relations with negative symptoms (e.g., poverty of speech, affective flattening or blunting, amotivation, social withdrawal) and found more significant and robust relationships. For example, with regard to social skill, when Mueser et al. (1990) examined the relations between social skill performance and negative symptoms in their study of 45 inpatients with schizophrenia, they found sizable and significant relations between social skill and negative symptoms among both men (r = -.39) and women (r = -.61). Mcdonald et al. (1998) also found significant relationships

between negative symptoms and social skill (r = -.31) in a cross-sectional study of 45 individuals with schizophrenia receiving outpatient treatment. Investigations of social adjustment and social networks among persons with schizophrenia have also shown significant relations with negative symptomatology. Early work by Pogue-Geile and Harrow (1985) followed 39 individuals with schizophrenia over a 5-year period, and found that negative symptoms were a robust longitudinal predictor of social and role functioning outcomes. Subsequent research has continued to support these results. For example, Villalta-Gil et al. (2006) recently reported on the relations between interpersonal functioning and negative symptoms. Similar to Pogue-Geile and Harrow, they found a significant and sizable correlation between poor social functioning and negative symptoms (r = .56) in a cross-sectional study of 113 outpatients with schizophrenia. Hofer et al. (2006) also reported on the relationship between social disability and negative symptoms in schizophrenia. They found that among 60 outpatients with schizophrenia, both satisfaction with social relations (r = -.32) and the presence of a significant social partner (r = -.28) were modestly related to severity of negative symptoms.

Investigations of deficit schizophrenia, which is characterized by prominent negative symptoms (Carpenter, Heinrichs, & Wagman, 1988), have also helped to illuminate the symptomatic correlates of social disability in the disorder. For example, Mueser, Douglas, Bellack, and Morrison (1991) found that the 1-year prognosis of individuals with deficit schizophrenia was significantly worse with regard to social adjustment, than those without deficit schizophrenia. In a retrospective study of 46 individuals with deficit schizophrenia and 141 with non-deficit schizophrenia, Fenton and McGlashan (1994) found that over the course of 15 years, individuals with deficit schizophrenia were less likely to be married and had significantly less frequent social contact than those without deficit schizophrenia. Subsequent investigations have largely yielded consistent results (Galderisi et al., 2002; Kopelowicz,

Liberman, Mintz, & Zarate, 1997), bringing forth proposals for a re-categorizing of the nosology of psychotic illnesses to include one of the most socially disabling forms of the disorder: deficit versus non-deficit schizophrenia (e.g., Carpenter, Heinrichs, & Wagman, 1988). As such, these findings suggest that negative symptoms are a significant and modest correlate of social disability in schizophrenia.

Nonetheless, there have been some notable exceptions to these results, as Mueser et al. (1991) found no significant relationship between changes in negative symptoms and social skill over the course of a year among 36 inpatients with schizophrenia. Additionally, Smith et al. (1999) also found a small, non-significant relationship between negative symptoms and interpersonal functioning (r = -.21) among 46 outpatients with schizophrenia. Likewise, when examining the relationship between negative symptoms and social functioning in a sample of 50 outpatients with schizophrenia, Norman et al. (1999) found small and non-significant relations between negative symptoms, frequency of social contact (r = -.22), and social skill (r = -.19). While these exceptions give pause to firm conclusions regarding the link between negative symptoms and social functioning, the weight of the evidence would seem to suggest that negative symptoms bear a modest and significant association with social disability in schizophrenia.

In summary, evidence regarding the contributions of primary psychopathology indicated in schizophrenia have suggested small to modest relationships with social disability, with negative symptoms emerging as the most consistent symptomatic correlate of social disability, and positive symptoms often showing little to no relation with social functioning. Such findings have led to investigations of other correlates of social disability in schizophrenia, many of which center around the importance of deficits in basic cognitive functioning.

Neurocognition. As research on the most likely contributors to social disability in

schizophrenia (i.e., positive and negative symptoms) has accumulated, suggesting only modest relations between social disability and psychopathology, schizophrenia researchers have begun to turn their attention to the role of cognition in facilitating and maintaining social disability among this population (Green, 1993; Hogarty & Flesher, 1992). This area of investigation has largely focused on the presence of deficits in basic cognition or "neurocognition," which includes a broad array of cognitive constructs representing areas of attention, memory, and executive function (or problem-solving). Within the past decade, studies have increasingly noted the prominence of deficits within all of these areas of neurocognition among many individuals with schizophrenia (Heinrichs & Zakzanis, 1998), and such deficits have been hypothesized as playing a highly significant role in social disability among this population (Green, 1993).

Perhaps one of the most commonly studied areas of neurocognitive dysfunction in schizophrenia, as it relates to social disability is executive function. The ability to problem-solve and engage in social decision-making bears important conceptual relevance to adequate social functioning, and many studies have found significant relations between these constructs. In a cross-sectional investigation of 80 outpatients with schizophrenia, Addington and Addington (1999) found modest and significant correlations between measures of executive function and different domains of social skill, as assessed by a role-playing measure (range of r = -.35 to -.43). Of particular note, these investigators found that the relationship between executive functioning and social skill remained significant, even after removing shared variance with negative symptomatology. In a 2.5-year follow-up study of the same 80 patients (65 of whom were available for follow-up), Addington and Addington (2000) again found significant, although somewhat attenuated relationships between baseline measures of executive function and select follow-up measures of social skill (r = -.36). In addition, these relationships again continued to persist after removing shared variance with negative symptoms. Research by independent

investigators has largely yielded results consistent with the work of Addington and Addington, and shown significant cross-sectional and longitudinal relations between executive function and social disability (see Green, Kern, Braff, & Mintz, 2000 for review). For example, Velligan, Bow-Thomas, Mahurin, Miller, and Halgunseth (2000) followed a sample of 40 individuals hospitalized with schizophrenia for 3.5 years after discharge, and found that measures of executive functioning were a modest longitudinal predictor of overall interpersonal functioning (r = .39). Such findings point to the importance of deficits in executive functioning to social disability among persons with schizophrenia, which is not surprising given the obvious conceptual link between planning, decision-making, behavioral inhibition, and social functioning.

In addition to neuropsychological studies of executive functioning ability, many investigators have also examined the relations between deficits in verbal and working memory, and social and functional disability in schizophrenia. Such deficits have been widely documented among persons with schizophrenia (Barch, Csernansky, Conturo, & Snyder, 2002), and research has begun to suggest that these deficits are indeed associated with poorer social recovery from the disorder. For example, in the same series of studies by Addington and Addington (1999, 2000), the researchers found that not only were deficits in executive functioning related to social skill, but that verbal working memory ability (e.g., the ability to hold and manipulate verbally relayed information in memory) was also significantly related to cross-sectional (range of r = .38-.44) and longitudinal (range of r = .27-.30) measures of social skill, after removing shared variance with negative symptomatology. Work by Villalta-Gil and colleagues (2006) has also provided evidence for a link between neurocognitive dysfunction in working memory and social disability in schizophrenia. These investigators conducted a cross-sectional examination of 113 individuals with schizophrenia living in the community and found

small, but significant relationships between interpersonal functioning and both verbal (r = -.22) and operative working memory (e.g., the ability to hold logical rules and operations in working memory) (r = -.28). Another study by Kopelowicz, Liberman, Ventura, Zarate, and Mintz (2005) found that in a cross-sectional examination of 56 individuals with schizophrenia (28 of whom had been specifically selected due to their uncommon social recovery), those individuals who were considered as experiencing a significant social recovery from the disorder had significantly higher verbal working memory (and executive function) scores than those who remained socially disabled. Perhaps most intriguingly, the working memory scores of individuals who experienced social recovery from schizophrenia mirrored those of healthy controls, providing further evidence of a link between working memory deficits (e.g., difficulty holding and manipulating information in work memory) and social disability in schizophrenia.

A final area that has received considerable attention from investigations of the functional significance of neurocognitive deficits in schizophrenia concerns the vigilance and attentional capacity of persons with the disorder. Similar to studies examining executive function and working memory deficits, research on attention in schizophrenia has also pointed to the presence of marked deficits in attentional capacity and the relevance of such deficits to social functioning. In a recent 7-year follow-up study of 99 individuals with first-episode schizophrenia, Milev, Ho, Arndt, and Andreasen (2005) found that deficits in attentional capacity explained the most amount of variance (7%) in a broad measure of social disability, after measures of working memory impairment and negative symptomatology. In another long-term follow-up study of persons with schizophrenia, Kurtz, Moberg, Ragland, Gur, and Gur (2005) examined the predictive utility of neurocognitive dysfunction to social disability among 70 individuals with schizophrenia, over a 4-year follow-up period. At one year, baseline measures of sustained attention significantly predicted interpersonal functioning (r = .28). Among the 25 individuals

who completed 4-year assessments, attention also predicted interpersonal functioning (r = .36), although this effect size did not reach statistical significance due to the modest sample size. In addition, these investigators found that deficits in sustained attention continued to remain modest, but significant predictors of interpersonal functioning after removing shared variance with positive and negative symptomatology. Recently, Hogarty, Greenwald, and Eack (2006) provided an experimental investigation of the contributions of neurocognitive deficits to social disability in schizophrenia. They examined the neurocognitive mechanisms of action for the large effects of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) on social disability among 106 outpatients with schizophrenia randomly assigned to either CET or a supportive control condition, and followed-up 1 year after receiving 2 years of treatment. These investigators found that measures of processing speed (a proxy for attention) proved to be the strongest *partial* mediator of the effects of CET on long-term social adjustment, compared to a large number of other neurocognitive domains. When combined with previous evidence regarding the functional significance of attention deficits in schizophrenia, this study provided promising evidence of the effects of remediating attention deficits on social disability among this population.

In summary, three broad domains of neurocognitive ability (executive function, working memory, and attention) have been consistently shown to be negatively associated with social disability among persons with schizophrenia. However, what can be seen from this brief review of the literature regarding the functional significance of neurocognitive deficits in schizophrenia is that similar to relations between social disability and psychopathology, the overall contribution of neurocognitive dysfunction to social disability is not large. In fact, a recent meta-analysis of the relationship between neurocognitive deficits and functional disability in schizophrenia found that such deficits explained on average only 8% of the variance in functional and social

outcomes among this population (Green, Kern, Braff, & Mintz, 2000). When studies have incorporated the association between psychopathology and social functioning, the amount of variance explained increases, although together neurocognitive deficits and psychopathology still explain only a small proportion of the variance in social disability. For example, when examining the correlates between neurocognitive deficits in working memory and attention, negative symptomatology, and social disability, Milev et al. (2005) found that such factors together explained only 15% of the variance in interpersonal functioning among persons with schizophrenia. As such, while there appears to be a consistent link between psychopathology (particularly negative symptoms), neurocognitive dysfunction, and social disability in schizophrenia, this link is not particularly strong, suggesting that additional factors may play a prominent role in conspiring against social recovery from individuals with the disorder. Nowhere else has this been highlighted more effectively than in the schizophrenia treatment literature, where pharmacological and rehabilitation studies that have successfully targeted psychopathology and neurocognitive dysfunction have yet to show substantial improvements in social disability (Swartz et al., 2007; Twamley, Jeste, & Bellack, 2003). Clearly more work is needed to elucidate the correlates of social disability among this population. Recent evidence is emerging suggesting that investigations of emotional intelligence in schizophrenia may provide particularly significant insights into social disability among this population.

C. EMOTIONAL INTELLIGENCE AND SOCIAL DISABILITY IN SCHIZOPHRENIA

Emotional intelligence concerns the ability to accurately perceive, recruit, understand, and manage emotions; and is emerging as a promising construct for understanding social disability in schizophrenia. Research has consistently shown that individuals with schizophrenia possess deficits in emotional intelligence, and some evidence has emerged suggesting that these

deficits are uniquely associated with social disability among this population. Unfortunately, methodological limitations inherent in these studies beckon the need for further investigation of the relevance of emotional intelligence to social disability. This section provides a brief introduction to the construct of emotional intelligence and its relevance to adaptive social functioning. Further, an overview of the application of emotional intelligence to schizophrenia research is provided through a review of evidence regarding the presence of deficits in emotional intelligence among persons with schizophrenia and a critical examination of the emerging literature surrounding their relation to social disability among this population.

1. Overview and Relevance of Emotional Intelligence

The study of emotional intelligence has been a broad area of social-cognitive investigation for over a century. Beginning with Charles Darwin's (1872/1965) theory of the universality of facial expressions of affect, emotions have been posited as providing important information that allows human beings to successfully adapt to and navigate the social world. Over the years, the different ways of using emotion to understand and adapt to varying social contexts has been refined into a concrete set of abilities, each with their own distinct body of evidence, which is commonly referred to today as emotional intelligence. Such abilities include (1) the ability to perceive and appraise emotions in oneself and others, or *emotion perception;* (2) the ability to understand the meaning and causes of emotions, or *emotion understanding;* (3) the ability to use emotions to direct attention and facilitate problem-solving, or *emotion facilitation;* and (4) the ability to manage ones' own and others' emotions, or *emotion regulation* (Mayer & Salovey, 1997). While investigators have focused more heavily on some areas of emotional intelligence (e.g., emotion perception and regulation) than others (e.g., emotion facilitation), social psychological research on emotional intelligence has provided substantial evidence for the important role of the construct in adaptive social functioning.

Perhaps the largest area of work concerning the significance of emotional intelligence to social functioning has stemmed from investigations of emotion perception. Early work by Ekman and Izard on emotion perception documented the universality of facial expressions of emotion, and the cross-cultural cues such expressions provide concerning the affective state of others (see Ekman, 1993 for review). Such cues have been subsequently documented as providing key information regarding a person's intentions and the nature of interpersonal interactions (Keltner & Kring, 1998; Knutson, 1996). For example, a recent meta-analysis on the effect of different emotional facial expressions on inferences about interpersonal verticality found that facial expressions of happiness (i.e., smiling) led individuals to make social inferences about the dominance of their counterpart in interpersonal interactions (Hall, Coats, & Lebeau, 2005). Other studies have found that emotions also provide critical information about the social environment (Keltner & Kring, 1998). For example, landmark work by Sorce and Emde (1981) found that parental facial expressions of emotion affected infants' readiness to crawl across an illusory visual cliff, indicating that emotion perception can provide pertinent information about environmental safety. These findings highlight the importance emotion perception to social functioning by suggesting that when emotions are perceived in others, at least through facial expressions, they can provide important information about the feelings of others, the nature of interpersonal relationships, and the social environment.

Research on the social/informational aspects of emotion has also focused on how experiences of emotion by the self can provide salient information about the social environment, underlying the importance of *understanding* one's own emotions. For example, several research studies have found consistent correlations between feelings of embarrassment and inferences about one's social status, and other studies have found feelings of anger and guilt to cue perceptions about equality in interpersonal relations (Keltner & Kring, 1998). Perhaps the most

illustrative work concerning the social significance of understanding one's own emotions concerns the study of individuals with alexithymia. Such individuals have, by definition, difficulty in understanding and vocalizing their own emotional states (Taylor, 1984), and research has consistently linked the presence of alexithymia to significant social disability. For example, Vanheule, Desmet, and Meganck (2007) recently found that in both a clinical and healthy sample, the presence of alexithymia was significantly related to poor interpersonal functioning in the domains of assertiveness and closeness. Another study by Spitzer, Siebel-Jurges, Barnow, Grabe, and Freyberger (2005) found that in a clinical sample, those individuals who exhibited high levels of alexithymia also tended to experience significantly more interpersonal problems than those without alexithymia. Such findings suggest that not only do the emotions of others provide important social information, but that one's understanding and interpretation of his/her own emotional state can also guide inferences about social phenomena and promote adaptive social functioning.

In addition to a large body of research showing that the emotions of others and the self can convey important social information that allows people to adaptively navigate their social environment, a significant body of research has also indicated that emotions *facilitate* a large number of basic and social-cognitive processes (Clore, Schwarz, & Conway, 1984). For example, a long line of research has shown that emotions can facilitate both attention to relevant stimuli and recall of information (Dolan, 2002). One such study by Doerksen and Shimamura (2001) found that individuals were nearly twice as likely to recall emotionally valanced words, compared to neutral words, suggesting an emotional enhancement in attending to and encoding affective laden stimuli. Studies of interpersonal attributions and social perception among both healthy and clinical populations have also found that a person's affective state markedly influences how he/she perceives the disposition of others (e.g., Forgas & Bower, 1987; Moore et

al., 2006). A widely replicated example of this phenomenon is Forgas and Bower's social judgment experiments, where happy individuals have been shown to be more likely to make positive judgments and remember positive characteristics about others, than sad individuals, again pointing to facilitative effects of emotion on attention and memory. Other research has also found emotion to have an important influence social decision making tasks (Forgas, 1991; Mayer, Salovey, & Caruso, 2004). For example, Forgas found that when trying to choose a partner for a problem-solving task, participants who were happy tended to decide on a partner based on his/her perceived skill for solving the task, whereas those who were sad tended to choose a partner who had good social skills, but who was not necessarily competent for completing the task. Such findings have repeatedly suggested an important role for emotion in basic and social-cognitive processes, and indicated that in some (but not all) cases emotions can be recruited to facilitate information processing (Salovey & Mayer, 1990).

Just as emotions can facilitate information processing, they can also inhibit or bias cognitive efforts by producing excessive load on cognitive systems or negatively biasing information processing (Gross, 1998). For example, individuals with high trait anxiety have been reported to overly attend to threatening stimuli at the consequence of disrupting goal-oriented behavior, when compared to non-anxious persons (Williams, Mathews, & MacLeod, 1996). The inhibitory effects of unchecked emotion on cognitive processing and behavior has lent rise to a large body of research on *emotion regulation*. Several psychologists have theorized that if emotions can taint and impair cognitive processes, then their management is likely to be paramount to optimal cognitive function and adaptive social behavior (Gross, 1998). In fact, recent research has suggested an important link between the dysregulation of emotion and poor interpersonal functioning. For example, one study by Schwartz and Proctor (2000) found that emotion dysregulation was significantly associated with both social-cognitive biases and degree

of social adjustment among maladjusted youth. Another study of youth by Eisenberg et al. (2001) found that emotion dysregulation was predictive of behavioral problems among school-aged children, continuing to suggest a link between emotion regulation and social behavior. In addition, the American Psychiatric Association (2000) has implicated the role of emotion mis-management or dysregulation in the etiology in most Axis I, and all Axis II disorders, suggesting the relevance of the construct to mental illness and adaptive social behavior. As such, it would appear that emotions can selectively enhance or inhibit information processing, and that the appropriate regulation of emotion may be vital for successful interpersonal functioning.

In summary, emotional intelligence is a unique social psychological construct that holds particular relevance to adaptive social functioning. The components of the construct include the ability to perceive and appraise emotions in oneself and others (emotion perception), the ability to understand the meaning and causes of emotions (emotion understanding), the ability to use emotions to direct attention and facilitate problem-solving (emotion facilitation), and the ability to manage ones' own and others' emotions (emotion regulation) (Mayer & Salovey, 1997). Considerable research in both social and cognitive psychology has progressed over the past century pointing to the validity of the construct of emotional intelligence and its relevance as a key social-cognitive component in adaptive social functioning, highlighting the promise of the construct for understanding social disability in schizophrenia, if deficits in emotional intelligence are present. What follows is a review of research examining the presence of emotional intelligence deficits in schizophrenia.

2. Emotional Intelligence Deficits in Schizophrenia

Deficits in emotion perception. Perhaps the most widely studied area of emotional intelligence deficits in schizophrenia research concerns the perception of emotion. As early as the 1970s, studies began to document the deficits individuals with schizophrenia possess in

emotion perception (for an early review see Morrison, Bellack, & Mueser, 1988). Initial work in this area by Muzekari and Bates (1977) examined the ability of 32 individuals with schizophrenia to identify the emotions represented in various male and female pictures of facial expressions. Compared to healthy controls, individuals with schizophrenia identified fewer correct emotions in facial expressions, particularly in expressions of sadness, anger and fear. Subsequent research conducted in the 1990s has largely confirmed these results and provided general support for a valence-specific deficit in emotion perception (see Edwards, Jackson, & Pattison, 2002 for review). One study by Archer, Hay, and Young (1994) examined the perception of emotion in pictures of facial expressions among 10 persons with schizophrenia, compared to 10 individuals with depression, and 10 healthy controls. The investigators found that persons with schizophrenia were significantly less accurate at identifying emotions in faces than both healthy and depressed participants. Additionally, the most profound emotion perception deficits among individuals with schizophrenia were found in the identification of fear and sadness. A similar study by Bellack, Blanchard, and Mueser (1996) also found significant impairments in facial emotion perception among 38 inpatients with schizophrenia; however deficits were circumscribed to the identification of negative emotions (i.e., sadness). Such findings have generally suggested that persons with schizophrenia have difficulty identifying negative emotions in the facial expressions of others (Mandal, Pandey, & Prasad, 1998), although debate ensues about the relative contribution of valence to these results (Edwards, Jackson, & Pattison, 2002).

Studies of emotion perception in schizophrenia have not been limited to the visual modality, but also have been extended to the auditory perception of emotions in affective prosody. Although fewer and less comprehensive investigations have been conducted in this area of research, findings thus far seem to suggest that emotion perception deficits in

schizophrenia are not limited to the identification of emotions in faces. An early uncontrolled evaluation by Fricchione, Sedler, and Shukla (1986) found that 2 (25%) out of 8 individuals diagnosed with schizophrenia had significant difficulty in identifying the emotions portrayed in valance-laden sentences presented by the investigators. A subsequent controlled evaluation of the recognition of emotions in affective prosody by Murphy and Cutting (1990) found that among 15 inpatients with schizophrenia, patients were significantly less accurate at identifying emotions in valance-laden sentences read by the investigators than healthy controls. However, no significant differences were found in emotion perception among individuals with schizophrenia and those with other severe and persistent psychiatric disabilities (i.e., bipolar disorder and major depression). Finally, in an international investigation of affective prosody recognition deficits in schizophrenia, Motomura (1994) examined the ability of 19 individuals with schizophrenia to appropriately label the valence of five different sentences presented by the investigators. Here too, they found that individuals with schizophrenia were significantly less accurate at identifying the emotions presented in the sentences than healthy controls. Consequently, while the investigation of emotion perception in affective prosody among persons with schizophrenia is arguably in its infancy, these studies seem to suggest the presence of emotion perception deficits in speech, as well as facial expressions.

Deficits in emotion understanding. A second area of investigation regarding emotional intelligence deficits in schizophrenia concerns the ability of such individuals to understand the meaning and causes of emotions. Unfortunately, to date, few investigations have been conducted examining the ability of individuals with schizophrenia to understand and differentiate between various emotions, despite the relevance of such issues to social functioning and explaining broader deficits in emotion recognition. Rather, investigations have largely been circumscribed to individuals with schizotypal personality disorder, a schizophrenia-spectrum disorder that is

characterized by pervasive social disability and perceptual disturbances that border on, but do not strictly constitute the positive symptoms of schizophrenia (American Psychiatric Association, 2000). Such research has largely indicated that individuals with schizotypal personality disorder and high levels of schizotypy have significant difficulty in understanding their own emotions. A recent study by Kerns (2005) examined emotional understanding in 34 individuals with positive schizotypy and 56 healthy controls by using a self-report measure asking participants about how often they feel clear about the emotions they experience. This investigator found that compared to healthy controls, individuals with schizotypy were significantly less clear about their emotions. A similar study by Berenbaum, Valera, and Kerns (2003) examined the relationship between schizotypal characteristics and symptoms of alexithymia among a group 75 women with schizotypal personality symptoms. Here too, these investigators found a robust relationship between schizotypal personality characteristics and a lack of emotional understanding, providing further evidence of potential emotion understanding deficits in schizophrenia-spectrum disorders. In addition, research has extended these findings to non-clinical populations with low-continuum schizotypal personality characteristics. For example, Berenbaum and colleagues (2006) examined the relationship between emotional understanding and schizotypal personality characteristics, using the same measure of emotional clarity employed by Kerns, in a sample of 247 college students. They found that even among this non-clinical population, all facets of schizotypal personality disorder were related to poor understanding of emotion.

While most research on deficits in emotion understanding among individuals with schizophrenia-spectrum illnesses has focused on those with schizotypal personality characteristics, the few studies that have been conducted with individuals with psychosis have suggested similar results. In a small study of 44 male substance abusers, Taylor, Parker, and Bagby (1990) examined the presence of alexithymia among individuals with a variety of

psychiatric disorders diagnosed by the Minnesota Multiphasic Personality Inventory. While these investigators noted their results as preliminary, they found that individuals with alexithymia (i.e., difficulty in understanding their emotions) were significantly more likely to have a diagnosis of schizophrenia and comorbid substance dependence than those without alexithymia. In the only case-control study of alexithymia in schizophrenia, Cedro, Kokoszka, Popiel, and Narkiewicz-Jodko (2001) examined the presence of alexithymia among 50 outpatients with paranoid schizophrenia, compared to 50 healthy controls. They found that on average, individuals with schizophrenia exhibited significantly greater levels of alexithymia than healthy individuals. Such findings are congruent with studies of schizotypal personality disorder and provide further evidence of potential deficits in emotional understanding among persons with schizophrenia. As such, while current evidence regarding the presence of emotion understanding deficits in schizophrenia is limited, taken together these findings suggest that individuals with schizophrenia and other schizophrenia-spectrum illnesses may exhibit important deficits in their ability to identify and understand their own emotions.

Deficits in emotion facilitation. Another area of investigation regarding emotional intelligence deficits in schizophrenia has been concerned with the ability of such individuals to use emotion to enhance and direct cognition. While little research has been conducted examining the presence of emotion facilitation deficits among persons with schizophrenia, what findings do exist seem to suggest a relative absence of emotion facilitation deficits among this population. For example, perhaps one of the most common methods of examining emotion facilitation is by using a word recognition and recall paradigm, where individuals are presented with a list of neutral and emotionally-valanced words, asked to learn them, and then asked to recall them after a short or prolonged delay (Bock & Klinger, 1986). Research among healthy individuals has consistently indicated that recall is greater for affect-laden words compared to

neutral words, indicating a facilitative role of emotion on memory encoding and retrieval (Rusting, 1998). Few studies, however have examined this process among persons with schizophrenia. An early study by Koh, Kayton, and Peterson (1976) examined the ability of 18 individuals with schizophrenia to recall a series of 50 pleasant, unpleasant, or neutral words, compared to 15 psychiatric patients without schizophrenia and 19 healthy controls. Surprisingly, they found that similar to healthy persons and those with other psychiatric disorders, individuals with schizophrenia demonstrated significantly better recall for pleasant and unpleasant words, compared to neutral words, suggesting intact emotional facilitation abilities. More recent work by Mathews and Barch (2004) examined the ability of 27 individuals with schizophrenia and 28 healthy controls to learn affect-laden and neutral words. While persons with schizophrenia demonstrated significant deficits in the recall of learned words, these investigators also found evidence that, similar to healthy individuals, individuals with schizophrenia were able to recall more emotionally-valanced words than neutral words. In the context of the marked deficits in other domains of emotional intelligence that have been reported among persons with schizophrenia, these studies may suggest a surprisingly intact facilitative effect of emotion on memory function in this population.

While the weight of the limited evidence seems to suggest that individuals with schizophrenia may have intact emotion facilitation abilities, further research in this area is clearly warranted, as studies been limited in number and have only examined facilitation deficits in relation to working memory. To date, no study has examined the facilitative effects of emotion on higher-level cognitive functions, such as problem-solving ability. As discussed above, healthy individuals can receive a substantial benefit from emotion in problem-solving, as emotions have been shown to help guide social inference and social decision-making (Forgas, 1991; Forgas & Bower, 1987), as well as provide motivation for solving problems and

completing tasks (Mayer & Salovey, 1997). However, schizophrenia research has long highlighted the difficulties individuals with the disorder have in solving both social and non-social problems (Hogarty & Flesher, 1999a) and documented marked deficits in motivation and hedonic capacity (e.g., Berenbaum, Oltmanns, & Gottesman, 1990). While it has recently been recognized that deficits in emotion processing, such as emotion facilitation, may provide a promising avenue for understanding amotivation and problem-solving deficits among persons with schizophrenia (Barch, 2005), investigators have yet to examine the presence of emotion facilitation deficits in the context of problem-solving tasks among this population. Consequently, firm conclusions regarding a relative absence of emotion facilitation deficits in schizophrenia appear to be premature and await support from future research examining the facilitative effects of emotion on a broader array of cognitive constructs.

Deficits in emotion regulation. A final area of research examining emotional intelligence deficits in schizophrenia has focused on the ability of such individuals to regulate stress and negative emotions. Schizophrenia researchers have long hypothesized that individuals with schizophrenia tend to be overly sensitive to negative emotions and have difficulty in regulating such emotions. Indeed, the predominant biopsychosocial model of schizophrenia conceptualizes the illness itself as largely a dysregulated response to negative emotions and stress (Zubin & Spring, 1977). Providing support for this, a large body of research has indicated that individuals with schizophrenia tend to show more intense emotional responses to stressful and emotional stimuli than healthy individuals. For example, a unique study by Myin-Germeys, van Os, Schwartz, Stone, and Delespaul (2001) examined the in-vivo emotional reactivity of 42 individuals with schizophrenia to daily life stressors, compared to 47 of their first-degree relatives and 49 healthy controls. These investigators found that while all participants reacted negatively to stressful events, persons with schizophrenia presented the strongest level of

negative emotional reaction to stressful events compared to their relatives and healthy controls. A psychophysiological study by Kring and Neale (1996) examined electrodermal measures of arousal recorded during emotional video segments among 23 males with schizophrenia, compared to healthy controls. These investigators found that when compared to healthy controls, individuals with schizophrenia exhibited significantly greater skin conductance reactivity to the emotional videos, regardless of valance, providing biological evidence for a hypersensitivity to stress and emotion among this population.

Evidence concerning the emotion regulation skills of persons with schizophrenia has been congruent with these findings, by suggesting that such individuals not only have pronounced emotional reactions to some stressful and emotional situations, but that they also have significant difficulty in regulating such reactions. In one study by Macdonald, Pica, Mcdonald, Hayes, and Baglioni (1998), self-reported coping strategies and the perceived effectiveness of these strategies were examined in a sample of 50 outpatients with early course schizophrenia or related psychotic disorder, and compared to healthy controls. Results from this investigation indicated that individuals with schizophrenia tended to more frequently attempt to engage in emotion-focused coping than healthy controls, and they also perceived the effectiveness of their coping strategies in regulating their distress as significantly less effective than their healthy counterparts. Another study by Bellack, Mueser, Wade, Sayers, and Morrison (1992) examined the ability of 34 individuals with schizophrenia to cope with negative emotional confrontations in a role-playing task, compared to 19 healthy controls and 24 individuals with primary affective disorders. These investigators found that individuals with schizophrenia consistently engaged in ineffective and maladaptive coping strategies to regulate their distress (e.g., lying or denying errors asserted by critical confederates) in the negatively valanced emotional situation. Further, the investigators found that the ineffectiveness with

which individuals with schizophrenia engaged in emotion regulation and coping strategies tended to manifest through poor social skill, even in the presence of only modestly negative interactions.

Recent neurobiological investigations have complemented these findings by suggesting structural and functional anomalies in brain regions commonly implicated in emotion regulation among persons with schizophrenia. For example, Bogerts and colleagues (1993) examined anatomical volumes of the mesiotemporal regions (e.g., amygdala and hippocampus), which are commonly implicated in emotion regulation (Davidson, Jackson, & Kalin, 2000), of 19 males with schizophrenia and 18 healthy controls. They found that individuals with schizophrenia tended to have show significantly reduced bilateral hippocampal volumes, compared to healthy controls. Further, they found that these volumetric reductions were significantly related to psychopathology, providing evidence for the presence and significance of structural abnormalities in the emotion regulation areas of the brain. Additionally, functional neuroimaging studies have also shown abnormal patterns of neuronal activation in neurobiological regions associated with emotion regulation among persons with schizophrenia. For example, one study by Schneider and colleagues (1998) examined amygdala activation in 13 individuals with schizophrenia after a sad or happy mood-induction, compared to 13 healthy controls. Interestingly, while controls exhibited a normal pattern of activation in the amygdala during both happy and sad mood states, individuals with schizophrenia did not exhibit any significant amygdala activation when experiencing a sad mood state, suggesting amygdala disengagement in the presence of aversive emotional states. Taken together, these findings lend support to the presence of emotion regulation deficits among persons with schizophrenia, and begin to link such deficits to specific neurobiological abnormalities.

In summary, substantial evidence suggests that persons with schizophrenia have

significant deficits in emotional intelligence, particularly in emotion perception, understanding, and regulation. Evidence concerning the presence of emotion facilitation deficits among this population have tended to be limited in scope and yielded null results, indicating a need for future investigations of emotion facilitation deficits in persons with schizophrenia. Unfortunately, while deficits in a number of domains of emotional intelligence are clearly present among individuals with schizophrenia, the functional significance of these deficits is not clear. What follows is a review of the limited evidence-base examining the significance of deficits in emotional intelligence to social disability among individuals with schizophrenia.

3. Emotional Intelligence as a Contributor to Social Disability in Schizophrenia

Despite the clear relevance of the construct of emotional intelligence to adaptive social functioning and the vast array of evidence within schizophrenia research documenting the prevalence of deficits in emotional intelligence among this population, relatively little research has been conducted concerning the contributions of such deficits to social disability among persons with schizophrenia. In addition, the majority of investigations that do exist examining the relations between emotional intelligence deficits and social disability in schizophrenia have been limited by cross-sectional studies among inpatient samples that are largely circumscribed to examinations of deficits in emotion perception. Nonetheless, these investigations have highlighted the potential relevance of deficits in emotional intelligence to social disability among this population.

One such study by Mueser and colleagues (1996) examined the cross-sectional relations between emotion perception ability, social skill, and interpersonal behavior among 28 inpatients with schizophrenia. These investigators found that the ability to accurately perceive emotions in faces significantly predicted both social skill (r = .37) and interpersonal functioning (r = -.45). Another cross-sectional study by Hooker and Park (2002) examined the relations between

emotion perception and interpersonal functioning among a sample 20 inpatients with schizophrenia. Similar to Mueser and colleagues, they also found significant and moderate relationships between measures of facial emotion perception and impaired interpersonal functioning (r = -.59). In addition, they also examined the perception of emotion in affective prosody and found significant relations between vocal emotion perception ability and occupational (r = -.58), but not social dysfunction (r = -.10), suggesting a modality-specific association between emotion perception deficits and social disability in schizophrenia. A study by Penn, Spaulding, Reed, and Sullivan (1996) also examined the cross-sectional relations between emotion perception and interpersonal functioning in a sample of 27 inpatients with schizophrenia. Here too, these investigators found significant relations between measures of facial emotion perception ability and interpersonal functioning in the domain of social competence (r = .37). Such early investigations of individuals living on inpatient units have provided preliminary evidence of the association between emotion perception deficits (particularly deficits in facial affect recognition) and social disability in schizophrenia. It should be noted, however, that none of these investigations examined the unique relationship between emotion perception and social disability beyond psychopathological and neurocognitive confounds.

In one of the few studies to examine the relationship between emotion perception and social disability among individuals with schizophrenia living in the community, Poole, Tobias, and Vinogradov (2000) examined the cross-sectional relations between emotion perception and interpersonal function in a sample of 40 outpatients with schizophrenia. They found that facial emotion perception ability had a significant and moderate relationship to interpersonal functioning (r = .36). Furthermore, they observed that this relationship remained significant and only marginally attenuated (r = .34) when removing shared variance with neurocognitive

dysfunction, providing some evidence for the unique association between deficits in emotion perception and social disability among outpatients with schizophrenia. In a study of the relations between emotion perception and social skill in individuals with schizophrenia living in the community, Ihnen, Penn, Corrigan, and Martin (1998) found similar results when examining these relations among 26 outpatients with schizophrenia. Specifically, they found that the ability to accurately identify emotion in faces was significantly and moderately, cross-sectionally related to overall social skill (r = .44), particularly communication skills (r = .50). Another recent study of the relationship between emotion perception and social skill by Pinkham and Penn (2006) examined the cross-sectional relations between facial emotion perception and role-play based measures of social skill in a sample of 49 outpatients with schizophrenia. These investigators also found significant relationships between several different measures of facial emotion perception and overall social skill (r = .32 - .38). However, in contrast to findings reported by Poole and colleagues, Pinkham and Penn found that measures of emotion perception no longer significantly predicted in social skill, after removing shared variance with neurocognitive ability and psychopathology. These findings provide some initial support for the relationship between emotion perception deficits and social disability among individuals with schizophrenia living in the community, in addition to those living on inpatient wards. However, evidence concerning the unique relationship between these deficits and social disability, beyond psychopathology and neurocognitive dysfunction remains mixed.

With regard to other deficits in emotional intelligence, very few studies have examined the relations between emotion understanding, facilitation, or management and social disability among persons with schizophrenia, despite several investigations indicating the presence of such deficits among this population. A recent pilot program by Hodel, Kern, and Brenner (2004) examined the effects of emotion management training among 11 inpatients with schizophrenia.

These investigators found that not only did their novel emotion management treatment improve psychopathology, but that it also improved ratings of social disability among their participants, suggesting that improving emotion management skills may significantly improve social disability among persons with schizophrenia. Such conclusions are highly speculative, however, as no measure of the effects of this program on actual emotion management were reported. To date, this exists as the only known study to begin to examine the relations between emotion regulation and social disability among persons with schizophrenia. In addition, no studies are known to have examined the relations between deficits in emotion understanding and emotion facilitation, and social disability among this population. As a consequence, evidence concerning how deficits in the domains of emotional intelligence, beyond those in emotion perception, are related to social disability in schizophrenia is extremely limited, highlighting an important need for future investigations of these promising, yet largely overlooked potential correlates of social disability among this population.

As can be seen by this review of the literature examining the relationship between emotional intelligence deficits and social disability in schizophrenia, some preliminary evidence exists suggesting that such deficits may be important correlates of social disability among this population. However, firm conclusions have yet to be drawn regarding the significance of deficits in emotional intelligence, as work in this area has suffered from several important limitations that are readily apparent from this review of the literature. In particular, no studies have directly examined the relations between the broader dimensions of emotional intelligence (i.e., emotion understanding, facilitation, and regulation) and social disability among this population. Investigations of both healthy and other psychiatric populations have repeatedly highlighted the significant role each of these broader domains of emotional intelligence can play in guiding adaptive social functioning, or in producing social disability if they go awry

(Davidson, Putnam, & Larson, 2000; Forgas, 1991; Forgas & Bower, 1987; Spitzer, Siebel-Jurges, Barnow, Grabe, & Freyberger, 2005; Vanheule, Desmet, & Meganck, 2007). Further, evidence exists suggesting that individuals with schizophrenia have deficits in a number of these domains of emotional intelligence (see Section C.2), leading to speculation about their relevance to social and functional outcome (Green, Olivier, Crawley, Penn, & Silverstein, 2005). As such, elucidating the significance of domains of emotional intelligence, beyond that of emotion perception, to social disability in this population represents a promising and important area of investigation.

In addition to the limited scope of studies examining the relations between emotional intelligence deficits and social disability in schizophrenia, studies have also been limited by their predominant use of cross-sectional designs. Although it has been hypothesized that emotional intelligence deficits *predict* social disability among persons with schizophrenia, the absence of longitudinal investigations regarding this question leaves the direction of this relationship in inevitable ambiguity. To date, only two longitudinal investigations concerning the relationship between emotional intelligence deficits and social disability in schizophrenia have been conducted. In the first study, Brekke and colleagues (2005) examined the longitudinal predictive association between deficits in emotion perception and social disability in 100 persons with schizophrenia living in the community. These investigators found that deficits in the ability to accurately identify emotions in faces at baseline significantly predicted interpersonal functioning at 1-year follow-up, lending support to directional hypotheses regarding emotional intelligence and social disability. In a subsequent study, Brekke and colleagues (2007) followed 102 outpatients with schizophrenia and used hierarchical linear modeling to examine the relative contribution of initial neurocognitive and social-cognitive function to changes in functional outcome over the course of 1 year. Here too, these investigators found that emotion perception

was a significant predictive of change in social and major role functioning over the course of the study. However, while these investigators accounted for shared variance with neurocognitive dysfunction in their investigation, they did not account for shared variance with psychopathology, which may limit their results. Further, consistent with other investigations of emotional intelligence in schizophrenia, these investigators limited their examination to deficits in emotion perception. Clearly future investigations are needed to replicate these findings, while accounting for important potential confounds and examining the broader domains of emotional intelligence.

The limitations inherent in Brekke and colleagues' (2005, 2007) work highlights another important limitation inherent in much of the research examining the significance of emotional intelligence deficits to social disability in schizophrenia, by exemplifying the common inattention of such research to important and relevant confounders of the relationship between emotional intelligence and social disability. Despite a large body of evidence indicating the presence of relationships between social disability, psychopathology (particularly negative symptoms), and neurocognitive dysfunction in this population (see section B.3); as well as some evidence concerning an overlap between social cognition, neurocognition, and psychopathology, no known study, to date has examined the relationship between emotional intelligence deficits and social disability in the context of these confounding factors. As some have suggested (Vauth, Rusch, Wirtz, & Corrigan, 2004), evidence presented thus far could be an artifact of relations between neurocognitive and psychopathological confounds and social disability. Consequently, while some evidence does implicate emotional intelligence deficits in social disability among persons with schizophrenia, whether these deficits make any unique contribution to social functioning beyond neurocognitive dysfunction and psychopathology is largely unknown.

Finally, previous research regarding the relationship between emotional intelligence and social disability in schizophrenia has been limited by its narrow focus on inpatient samples. With the introduction of social policies favoring deinstitutionalization and atypical antipsychotic medications, the majority of individuals with schizophrenia now live in the community, yet most studies examining the relevance of emotional intelligence deficits to social disability among this population have focused on individuals living on inpatient units. While this evidence has led to important insights regarding the functional significance of emotional intelligence, such research may be less applicable to those individuals who live in the community. By definition, social functioning on an inpatient unit is both restricted and guided by very different principles than those that govern the non-institutionalized world of normal society. For example, the social functioning of individuals who live on an inpatient unit is substantially guided by explicit rules for behavior, whereas many of the social norms of society are often very subtle and implicit, yet key to adaptive social functioning. These differences in the range and nature of social functioning among individuals living on inpatient units may not only attenuate correlations with measures of emotional intelligence, but may also represent a different form of social disability when compared to those individuals living in the community (i.e., functioning based on explicit vs. implicit rules of behavior). As a consequence, the majority of schizophrenia research on the social significance of emotional intelligence may not generalize to the majority of individuals with schizophrenia, and thus more research on those individuals living in the community is needed.

In summary, a limited body of evidence suggests that deficits in emotional intelligence (mostly in emotion perception) may be related to social disability among persons with schizophrenia. However, a review of these investigations highlights several important limitations, including (1) a heavy emphasis on emotion perception, (2) a lack of longitudinal

investigations, (3) frequent inattention to the potential confounding effects of psychopathology and neurocognition, and (4) a narrow focus on inpatient samples. Such limitations illustrate the need for future longitudinal investigations of the unique contributions of the broader dimensions of emotional intelligence to social disability among individuals with schizophrenia living in the community. What follows is a brief overview of the current state-of-the-art in the assessment of emotional intelligence, which offers insight into the how difficulties experienced in the measurement of this construct have contributed to the limited investigation of the relevance of emotional intelligence to social disability among persons with schizophrenia.

D. ISSUES IN THE MEASUREMENT OF EMOTIONAL INTELLIGENCE

Emotional intelligence has been a widely studied construct among social psychologists for nearly a century, and recently been recognized as a potential key construct for understanding social disability among persons with schizophrenia. Unfortunately, despite its lengthy history of study, the measurement of emotional intelligence has not proven to be easy, particularly in psychiatric populations. This section presents a brief and critical overview of the measurement of emotional intelligence, which both identifies the limitations that have frequently plagued traditional measurement techniques and points to the promise of a performance-based measure of emotional intelligence that has been rigorously validated among healthy individuals, for assessing this construct among persons with schizophrenia.

1. Limitations of Traditional Measurement Strategies

Despite a broad array of literature signifying emotional intelligence as a key social-cognitive construct in understanding the adaptive behavior of healthy individuals, as well as a growing body of evidence highlighting the promise of this construct for understanding social disability among persons with schizophrenia, strategies to measure the different components of

emotional intelligence among persons with schizophrenia and other psychiatric disabilities are in their infancy. Currently, there exists a wide variety of self-report and performance-based assessments of emotional intelligence that have been generated from psychiatry and the broader field of social psychology, many of which have limited validity when applied to healthy and/or psychiatric populations. Perhaps the most commonly used measure of emotional intelligence is Ekman and Friesen's (1976) pictures of facial expressions that assess emotion perception. During this performance-based assessment, individuals are presented with different pictures of facial expressions of emotion, and are asked to identify the corresponding emotion expressed in the picture. This measure has been shown to be a reliable and valid assessment emotion perception among both healthy and clinical populations (Ekman & Friesen, 1976), and widely used in schizophrenia research. However, while research has suggested that Ekman's pictures have favorable psychometric properties in several populations, the instrument measures only a limited range of the emotional intelligence construct, in that it is circumscribed to the area of emotion perception. This limitation has served as an impetus for more recent instrument development efforts attempting to assess the broader components of emotional intelligence.

To date, the majority of measures attempting to assess the broader domains of emotional intelligence have made use of self-report measurement strategies. Social psychology, in particular, has contributed significantly to this area of measurement, by generating a number of self-report measures that reflect the different aspects of this construct. For example, the Emotional Quotient Inventory (Bar-On, 1997; EQ-i) has been developed as a self-report measure to assess emotional understanding in social relationships as well as the use of emotions to facilitate problem-solving; both key social-cognitive constructs outlined in Mayer and Salovey's (1997) model of emotional intelligence. Although the measure has been primarily used in academic settings and no research has examined the relation between EQ-i scores and social

functioning, available research does suggest that this measure is predictive of academic achievement (Bar-On, 1997). Unfortunately, to date, there have been no applications of the EQ-i to individuals with psychiatric disabilities, however investigations of this instrument among healthy populations has suggested that it is largely reflective of personality, rather than emotional ability (Davies, Stankov, & Roberts, 1998; Lopes, Salovey, & Straus, 2003; O'Connor & Little, 2003; Warwick & Nettelbeck, 2004).

Another popular self-report measure, the Trait Meta-Mood Scale (TMMS; Salovey, Mayer, Goldman, Turvey, & Palfai, 1995), was designed to measure a person's awareness and ability to regulate his/her own emotions, which also captures several key components of emotional intelligence. The scale has been applied in a somewhat wider context than the EQ-i, with studies indicating that the ability to understand and discriminate between emotions is positively related to quality of life (Palmer, Donaldson, & Stough, 2002). Further, the ability to adequately manage emotions seems associated with effective leadership styles (Palmer, Walls, Burgess, & Stough, 2001) and adaptive stress management (Salovey, Stroud, Woolery, & Epel, 2002). A recent study (particularly relevant to schizophrenia) found that individuals with positive schizotypy were less clear about their emotions and tended to be emotionally overwhelmed (Kerns, 2005). Unfortunately, here too, the TMMS appears to be largely redundant with personality, yielding little unique information about emotional abilities beyond the Big Five personality traits (Davies, Stankov, & Roberts, 1998; Lopes, Salovey, & Straus, 2003; O'Connor & Little, 2003; Warwick & Nettelbeck, 2004).

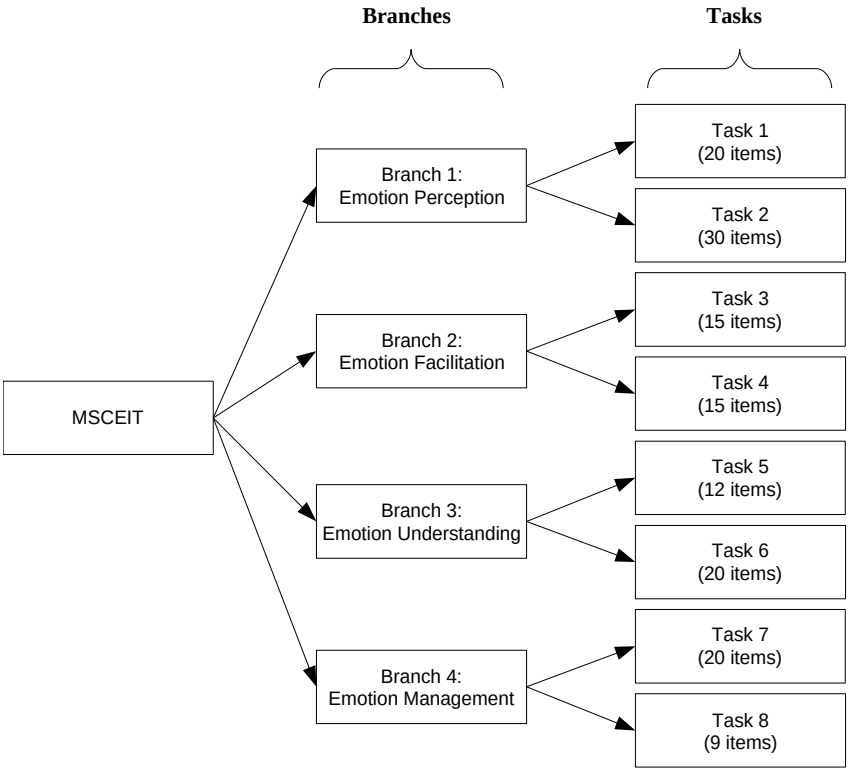
A recent review of existing self-report measurement strategies for assessing emotional intelligence suggested that such strategies (of which the EQ-i and TMMS are representative) are limited on two fronts (Mayer, Salovey, & Caruso, 2004). First, since emotional intelligence is conceptualized as an *ability*, its optimal measurement is thought to be theoretically based in

measures that require the performance of that ability, rather than a self-report of one's own ability. Unfortunately, with the exception of Ekman's pictures of facial expressions, all other emotional intelligence measures make use self-reports. Just as general IQ is conceptualized as a constellation of abilities that are measured by a person's performance in solving logical and analytic problems, emotional intelligence is also thought of as an ability that necessitates the measurement of a person's performance in solving emotional problems. Indeed, a large body of literature has suggested little relationship between performance-based and self-report measures of intelligence (e.g., Paulhus, Lysy, & Yik, 1998), highlighting the need for performance-based, rather than self-report assessment strategies of abilities. Second, as highlighted above, all self-report measurements of emotional intelligence share a substantial amount of variance with personality characteristics. As a consequence, these instruments offer little more than standardized assessments of the Big Five personality traits, and largely represent proxies for personality assessment rather than emotional intelligence. As such, traditional measurement strategies have either been limited in scope or method, which has significantly limited investigations of emotional intelligence among persons with schizophrenia. However, recently the decade-long development and validation of a unique, performance-based measure of emotional intelligence has come to fruition with the Mayer-Salovey-Caruso Emotional Intelligence (Mayer, Salovey, Caruso, & Sitarenios, 2003; MSCEIT), which holds significant promise for providing comprehensive and valid assessments of emotional intelligence among persons with schizophrenia.

2. Promise of the Mayer-Salovey-Caruso Emotional Intelligence Test

Over the past decade, the arbiters of the leading scientific model of emotional intelligence have dedicated their efforts to the development of a reliable and valid performance-based measure of the construct, the Mayer-Salovey-Caruso Emotional Intelligence (Mayer, Salovey,

Figure 1. Mayer-Salovey-Caruso Emotional Intelligence Test



Caruso, & Sitarenios, 2003; MSCEIT). This 8-task (141-item) instrument provides performance-based assessments of the four branches of emotional intelligence (see Figure 1), and as such represents a significance advance to a field that was for so long stuck only with a performance-based measure of emotion perception. Results from Mayer and colleague's developmental work on the MSCEIT has produced a body of evidence across over 1000 healthy persons suggesting the instrument has strong levels of reliability and validity (reviewed in detail below in Chapter 3). Briefly, evidence to date has indicated that the MSCEIT offers high levels of internal consistency and retest reliability (Mayer, Salovey, Caruso, & Sitarenios, 2003). In addition, the MSCEIT has been shown to possess adequate discriminant validity from measures of personality and general intellectual ability (Brackett & Mayer, 2003; Caruso, Mayer, & Salovey, 2002; Day & Carroll, 2004; Mayer, Caruso, & Salovey, 1999; Warwick & Nettelbeck, 2004), a factor structure consistent with the underlying theoretical framework of emotional intelligence (Gignac,

2005; Mayer, Salovey, Caruso, & Sitarenios, 2003; Palmer, Gignac, Manocha, & Stough, 2005), and adequate predictive validity, with regard to interpersonal functioning and behavior (Brackett, Rivers, Shiffman, Lerner, & Salovey, 2006; Brackett & Mayer, 2003; Lopes, Brackett, Nezlek, Schutz, Sellin, & Salovey, 2004). These findings, which have been verified by independent investigators, provide strong support for the ability of the MSCEIT to assess emotional intelligence in a reliable and valid manner among healthy individuals. In fact, evidence concerning the psychometric properties of this instrument have been so strong, that the National Institute of Mental Health Measurement and Treatment Research to Improve Cognition in Schizophrenia committee has recommended this assessment to be the key measure of social cognition in schizophrenia research. Unfortunately, to date, no investigation has confirmed these psychometric properties among individuals with schizophrenia, although a limited initial investigation by Nuechterlein and colleagues (2008) recently found adequate retest reliability when examining a subset of the MSCEIT subscales. As such, any investigation of emotional intelligence in schizophrenia research that makes use of this promising measure must first confirm previous evidence regarding its psychometric properties among this population.

E. PROPOSED STUDY AND HYPOTHESES

This study sought to conduct a longitudinal investigation of the relationship between deficits in emotional intelligence, as measured by the MSCEIT, and social disability in schizophrenia, using secondary data collected from an ongoing randomized clinical trial of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) for individuals living in the community with early course of schizophrenia (n = 57 at baseline, n = 47 followed-up at 1 year).

1. Study Context

This research was conducted within the context of an ongoing experimental examination of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006), a cognitive rehabilitation program for persons with schizophrenia that has been shown to be highly effective at improving emotional intelligence and social disability among this population (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007; Hogarty et al., 2004; Hogarty, Greenwald, & Eack, 2006). Individuals in this experiment were randomly assigned to receive either CET or an active, supportive control condition, both of which are described in detail in Chapter 3, and treated for 2 years. This research makes use of baseline (n = 57) and 1 year follow-up (n = 47) data from this clinical trial to conduct a unique examination of the relationship between emotional intelligence and social disability in schizophrenia. The experimental design and known efficacy of CET are purposely capitalized on in this research to ultimately investigate the relationship between experimentally manipulated (improved) levels of emotional intelligence and social disability in schizophrenia. This experimental manipulation comes from the random assignment of persons in the trial to either a treatment condition which has known effects on emotional intelligence (i.e., CET) or a treatment condition which has no known effects on emotional intelligence (i.e., the supportive control). In this regard, this research includes and ultimately moves beyond cross-sectional correlational examinations of the relationship between emotional intelligence and social disability in schizophrenia, by beginning to test the causal impact of longitudinal changes in emotional intelligence to changes in social disability within the context of experimental manipulation.

2. Aims and Hypotheses

Using data from this clinical trial of CET, this research aimed to first confirm the psychometric properties of the MSCEIT among persons with schizophrenia and then examine the

longitudinal relationship between emotional intelligence and social disability in schizophrenia. The specific aims and concomitant hypotheses of this research were to:

Aim #1: Confirm the psychometric properties of the MSCEIT when applied to persons with schizophrenia. Baseline data (n = 57) were used to confirm the internal consistency and discriminant validity (from neurocognitive function and psychopathology) of the MSCEIT. Additionally, given the modest sample size, an exploratory investigation of the factor structure of the instrument's 8 tasks was also conducted. It was expected that the MSCEIT would demonstrate adequate internal consistency ($\alpha \geq .80$) when applied to persons with schizophrenia, and would share no more than a small to moderate amount of variance ($r < .50$) with measures of neurocognitive function and psychopathology. It is also expected that the optimal factor solution for the 8 MSCEIT tasks would be a 4-factor oblique solution consistent with that presented in Figure 1 and reported in previous research with healthy individuals (Mayer et al., 2003).

Aim #2: Examine the unique cross-sectional relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology. Baseline data (n = 57) were used to compute correlation matrices and hierarchical linear regression analyses to examine the zero-order and unique associations (beyond neurocognitive function and psychopathology) between emotional intelligence and social disability. Hypotheses include:

H_{1a}: Emotional intelligence is significantly, negatively correlated with social disability at baseline.

H_{1b}: Emotional intelligence is significantly, negatively correlated with social disability at baseline, after removing shared variance with neurocognitive function and psychopathology.

Aim #3: Examine the unique longitudinal relationship between changes in emotional

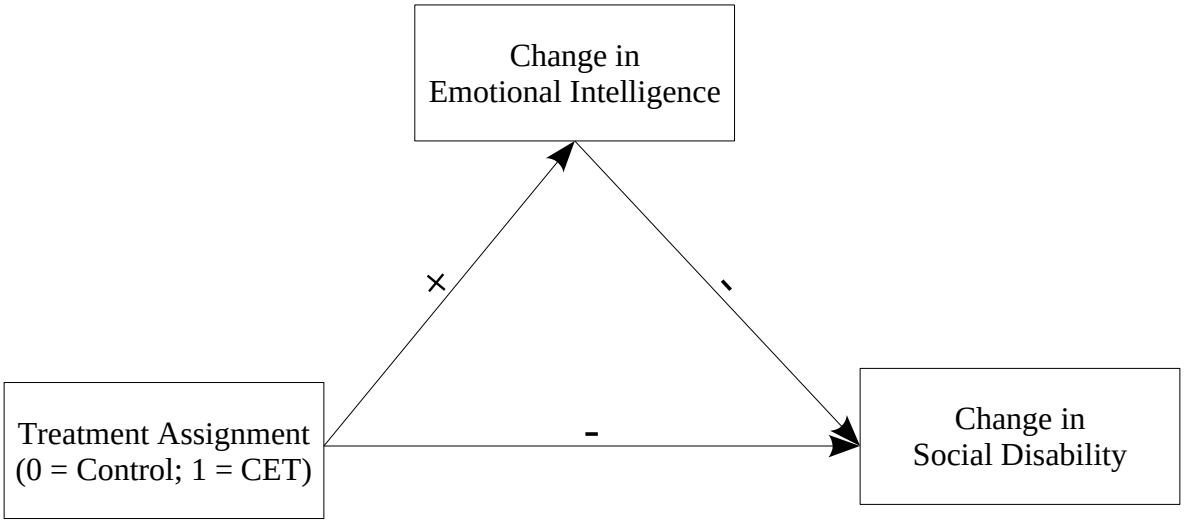
intelligence and changes in social disability, above and beyond changes in neurocognitive function and psychopathology. A mediator-analytic framework for clinical trials that capitalizes on the experimental design employed in this dataset is used with complete follow-up data (n = 47) to examine the unique relationship between experimentally manipulated (i.e., treatment-induced) changes in MSCEIT scores and changes in social disability through a series of multiple regression analyses (see Figure 2). Hypotheses include:

H_{2a}: Treatment assignment (0 = control; 1 = CET) is significantly correlated with improvements in emotional intelligence and reductions social disability.

H_{2b}: Changes in emotional intelligence are significantly, negatively correlated with changes in social disability, both before and after adjusting for improvements in neurocognitive function and psychopathology.

H_{2c}: Changes in emotional intelligence are partial, but significant mediators of the negative relationship between treatment assignment (0 = control; 1 = CET) and social disability, both before and after adjusting for the relationship between treatment assignment and changes in neurocognitive function and psychopathology.

Figure 2. Proposed Mediational Model of Emotional Intelligence and Social Disability



III. METHOD

This research makes use of secondary data analysis using a longitudinal, clinical and neuropsychological dataset from an ongoing clinical trial of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) for persons with schizophrenia or schizoaffective disorder. This unique dataset is queried to answer several questions concerning the significance of emotional intelligence deficits to social disability in schizophrenia. Specifically, this dataset is used to engage in an experimental, longitudinal investigation of the relationship between deficits in emotional intelligence and social disability in schizophrenia at both univariate and multivariate levels, while accounting for shared variance with neurocognitive function and psychopathology. Additionally, these data are used to confirm the reliability and validity of a unique measure of emotional intelligence that has yet to be validated among persons with schizophrenia. This chapter describes the design, participants, and measurement techniques used in this ongoing clinical trial that bear relevance to these questions, as well as the analytic techniques used for addressing the aims of this research.

A. STUDY DESIGN AND PARTICIPANTS

This research is embedded in an existing longitudinal study evaluating neurobiological and functional outcomes of individuals in the early course of schizophrenia or schizoaffective disorder participating in a clinical trial of social and neurocognitive rehabilitation (CET). This study makes use of a longitudinal, randomized-controlled design where study participants are randomly assigned to receive either CET or Enriched Supportive Therapy (EST) (Hogarty et al., 2004; described below). These two treatments generally consist of a cognitive remediation program (CET) and an illness management program (EST). Participants were recruited from the

inpatient and outpatient units of the Western Psychiatric Institute and Clinic in Pittsburgh, Pennsylvania, as well as from community mental health centers in the surrounding area, to participate in a clinical trial of CET. Potential participants between the ages of 16 and 65 were recruited for this clinical trial if they are currently living in the community and have been diagnosed with schizophrenia or schizoaffective disorder within the past eight years. This research included individuals diagnosed with both schizophrenia and schizoaffective disorder, as both disorders are phenotypic representations of the schizophrenia-spectrum of illnesses, with the latter experiencing more affective symptoms during acute psychotic exacerbations than the former (American Psychiatric Association, 2000). Individuals younger than 16 years were excluded from this research, as there is still some question as to whether schizophrenia can be detected at such an early age (American Psychiatric Association, 2000); and individuals older than 65 years were also excluded to avoid potential confounds in physical and neurocognitive deterioration that can occur in elderly patients with schizophrenia (Bowie, Reichenberg, Patterson, Heaton, & Harvey, 2006). Individuals diagnosed with an active comorbid substance abuse disorder within the past 2 months were also excluded from this research, in order to avoid the admittedly common, but confounding impact of substance use on cognitive functioning and social disability. Individuals with an Intelligence Quotient less than 80 have been excluded from this clinical trial, due to theoretical and empirical evidence suggesting the diminished efficacy of CET among patients with mental insufficiency. Finally, only individuals demonstrating social and cognitive disability on the Cognitive Style and Social Cognition Eligibility Interview (Hogarty et al., 2004) were eligible to participant in this research, given that this trial focused on remediating social and cognitive impairments commonly, but not always inherent to schizophrenia. No further exclusion criteria were used in this study in order to optimize the diversity of the sample and allow for adequate variability in cognitive and social dysfunction.

A total of 57 persons with schizophrenia or schizoaffective disorder living in the community were recruited for participation, 47 of whom have completed at least one year in the ongoing clinical trial of CET. This research makes use of complete baseline (n = 57) and 1-year follow-up (n = 47) datasets from this clinical trial. A complete description of the baseline and follow-up samples is provided in Chapter 4.

B. TREATMENTS

1. Cognitive Enhancement Therapy

Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006), the experimental condition in this research, is a unique, developmental approach to the remediation of neurocognitive and social-cognitive deficits in schizophrenia. The treatment is developmental in that it views cognitive dysfunction in schizophrenia has a byproduct of an early neurodevelopmental insult (Keshavan & Hogarty, 1999), and uses evidence regarding the neuroplasticity of regions implicated in impaired cognition to posit that enriched cognitive experiences can "jump start" this developmental delay and thereby improve cognitive function. CET provides these enriched cognitive experiences through targeted neurocognitive training and secondary socialization opportunities, so that individuals can develop the social and non-social cognitive abilities needed to succeed in complex interpersonal interactions. In this respect, the program incorporates an individual neurocognitive training program involving 60 hours of training in cognitive exercises designed to enhance attention, memory, and problem-solving abilities; and a 45 session, social-cognitive group that focuses on improving the social-cognitive abilities that underlie effective interpersonal behavior in unrehearsed social situations, such as taking the perspective of others, reading non-verbal cues, and adjusting knowledge about the rules and norms of behavior based on the social context (Hogarty & Greenwald, 2006). To date,

evidence from a previous randomized-controlled trial of 121 long-term patients with schizophrenia indicated that CET was highly effective at improving both neurocognitive (within-group d = 1.46, between-group d = .46) and social-cognitive (within-group d = 1.50, between-group d = .72) function in schizophrenia, and that these effects produced sizable and lasting benefits on overall social adjustment (Hogarty et al., 2004; Hogarty, Greenwald, & Eack, 2006). In addition, preliminary evidence from the current early course trial of CET has also indicated that the program can produce marked benefits in social cognition and social adjustment among individuals with early course schizophrenia (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007; Keshavan, 2005).

2. Enriched Supportive Therapy

Enriched Supportive Therapy (EST), the supportive control condition in this research, consists of components from the basic and intermediate phases of Personal Therapy (Hogarty, 2002), which is broadly a stress management and psychoeducation program for persons with schizophrenia. The focus of EST is on reducing the late (2nd and 3rd year, post-discharge) relapses that frequently occur among persons with schizophrenia, by providing such individuals with stress management and affect regulation techniques that are linked to the stage of recovery from an exacerbation of the illness (e.g., earlier stages of recovery receive techniques from the basic phases of the treatment). The management of stress, in particular, is a key component to this approach, based on earlier observations that the prodromal signs of a psychotic relapse tend to manifest as symptoms of dysregulated arousal, rather than the positive signs of psychosis (Carr, 1983). Based on these observations, EST incorporates an array of both novel (e.g., managing and responding to criticism) and traditional strategies (e.g., diaphragmatic breathing, progressive muscle relaxation) for managing stress and arousal, and flexibly provides individuals with stress management techniques designed to prevent the progression of a schizophrenia

prodrome into a full psychotic relapse (Hogarty, 2002). A unique aspect of EST is its sensitivity to individuals' need for different levels of treatment intensity at different phases of the illness, and as such is divided into basic and intermediate phases to accommodate a wide range of individuals, from the recently discharged to those living in the community for several years (Hogarty, 2002). The treatment is provided in one-hour, weekly individual sessions over the course of two years. Personal Therapy, the expanded predecessor of EST, has been shown to be highly effective at reducing late, post-discharge relapse among persons with schizophrenia (Hogarty et al., 1997a). Specific studies of EST have shown it to be effective at improving some aspects of social cognition (Hogarty et al., 2004), however no effects have been observed on overall emotional intelligence (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007). Minor effects have only been observed with regard to improved emotion understanding (within-group d = .14).

C. MEASUREMENTS

To achieve the aims of this research and *examine the unique relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology*, a combination of cognitive tests and clinician-rated instruments were used to assess emotional intelligence (independent/intervening variable), social disability (dependent variable), and neurocognitive functioning and psychopathology (confounding variables). The independent variable in Aim 3, treatment, was assessed by treatment group assignment. Instruments used to assess these variables included the Mayer-Salovey-Caruso Emotional Intelligence Test (Mayer, Salovey, Caruso, & Sitarenios, 2003); the Wechsler Adult Intelligence Scale (Wechsler, 1981); the third version of the Wechsler Memory Scale (Wechsler, 1987); the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987); the Wisconsin Card

Sorting Test (Heaton, Chelune, Talley, Kay, Curtiss, 1993); two measures of simple and choice reaction time (Ben-Yishay, Piasetsky, & Rattok, 1985; Bracy, 1994); the Brief Psychiatric Rating Scale (Overall & Gorham, 1962); and the Social Adjustment Scale-II (Schooler, Weissman, & Hogarty, 1979). Due to the large number of complex measures included in this research, an overview of variables garnered from each measure is provided in Table 1. The psychometric properties and use of these measures among individuals with schizophrenia is discussed in detail in the following sections.

Table 1. *Study Variables and Measurement Sources*

Variable	Source (Items)	Calculation
Total Emotional Intelligence	MSCEIT total score (141 items)	NA[a]
Emotion Perception	MSCEIT branch 1 score (50 items)	NA
Emotion Facilitation	MSCEIT branch 2 score (30 items)	NA
Emotion Understanding	MSCEIT branch 3 score (32 items)	NA
Emotion Management	MSCEIT branch 4 score (29 items)	NA
Total Social Disability	SAS-II total (26 items)	Mean of items 23, 24, 29-31, 33, 41-44, 46-51, 53-60, 63, 64
Interpersonal Anguish	SAS-II work, household, external family, social leisure, and personal well-being areas (12 items)	Mean of items 23, 24, 41-44, 46-48, 57, 63, 64
Sexual Relations	SAS-II social leisure area (3 items)	Mean of items 58-60
Family Relations	SAS-II household area (4 items)	Mean of items 29-31, 33
Non-Family Relations/Social Leisure Participation	SAS-II social leisure area (7 items)	Mean of items 49-51, 53-56
Total Neurocognitive Function	Ben-Yishay simple reaction time: Fixed and variable inter-stimulus interval reaction time (2 items, reverse scored) Bracy choice reaction time: dominant and non-dominant hand reaction time (2 items, reverse scored) WAIS-R: Digit-span test (1 item) WMS-R: Stories A and B, immediate/delayed recall (2 items) CVLT: List A, trials 1-5 total immediate and delayed recall (2 items) WCST: Number of perseverative errors (1 item, reverse scored)	Mean of scaled items 1-10
Total Psychopathology	BPRS total (18 items)	Mean of items 1-18

Note. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale II, WAIS-R = Wechsler Adult Intelligence Scale-Revised, WMS-R = Wechsler Memory Scale-Revised, CVLT = California Verbal Learning Test, WCST = Wisconsin Card Sorting Test, BPRS = Brief Psychiatric Rating Scale

[a]MSCEIT scores were calculated by the test distributer based on consensus scores from normative samples.

1. Independent/Intervening Variable: Emotional Intelligence

To examine the cross-sectional (Aim #2) and longitudinal (Aim #3) relationship between emotional intelligence and social disability, emotional intelligence is assessed using the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; Mayer, Salovey, Caruso, & Sitarenios, 2003). The MSCEIT is a performance-based test of emotional processing ability or emotional intelligence, that assesses the four domains of emotional intelligence described above (i.e., emotion perception, emotion understanding, emotion facilitation, and emotion regulation). The instrument is performance-based in that rather than asking participants to self-report on their emotional abilities, the MSCEIT requires participants to solve emotionally-laden problems (Mayer, Caruso, & Salovey, 2000), much like tests of mathematical or verbal intelligence. The MSCEIT consists of 8 tasks (each containing several multiple choice items), which form the 4 branches of emotional intelligence. These 8 tasks include tasks requiring participants to identify emotion in human faces, scenery, and artwork; match emotions to sensations; judge which emotions facilitate certain thoughts and behaviors; identify how emotions are combined to form other emotions; identify how emotions change across intensities; and identify strategies to manage one's own emotions and the emotions of others. Two tasks comprise a branch score in the Mayer and Salovey (1997) four-factor model of emotional intelligence (see Figure 1). For example, the MSCEIT contains a task asking participants to identify specific emotions in faces and another task asking participants to identify specific emotions in different pictures of scenery and artwork. These two tasks form one "branch" of their emotional intelligence model called emotion perception. Items are rated on a variable 5-point scale, are scored by consensus norms from a large healthy sample, and scaled with a mean of 100 (*SD* = 15), with lower scores reflecting poorer emotion processing.

Recent psychometric evaluations of the MSCEIT indicate that it has adequate levels of

internal consistency reliability among its branches (range of α = .79-.91), and has lent some support to the structural validity of the instrument by supporting Mayer and Salovey's four-factor model of emotional intelligence (Mayer, Salovey, Caruso, & Sitarenios, 2003; see Figure 1). Furthermore, unlike self-report attempts to measure the emotional components of social cognition, the MSCEIT has shown good discriminant construct validity by showing little to modest overlap with measures of personality (range of r = -.18 to .39) and general neurocognitive ability (range of r = .14 to .36) (Brackett & Mayer, 2003; Caruso, Mayer, & Salovey, 2002; Day & Carroll, 2004; Mayer, Caruso, & Salovey, 1999; Warwick & Nettelbeck, 2004; see Mayer, Salovey, & Caruso, 2004 for a review). Additionally, the MSCEIT has been shown to predict measures of the quality of interpersonal interactions (Lopes, Brackett, Nezlek, Schutz, Sellin, & Salovey, 2004) and measures of psychological well-being and academic achievement (Brackett & Mayer, 2003) beyond commonly used assessments of personality, lending some support to its incremental and predictive criterion validity. All of this psychometric evidence suggests that the emotion processing abilities assessed by the MSCEIT are constructs that are unique from other cognitive abilities and personality, that these constructs are internally consistent, contain a theoretically driven and empirically supported factor structure, and that they predict theoretically relevant outcomes among healthy populations. Unfortunately, to date, no evidence has been put forth comprehensively describing the psychometric properties of this instrument when used with persons with schizophrenia, although a limited initial investigation by Nuechterlein and colleagues (2008) recently found adequate retest reliability when examining the emotion perception and management subscales of the MSCEIT.

2. Dependent Variable: Social Disability

Social disability is conceptualized in this research as deficiencies in the quality and/or

quantity of a person's interpersonal relations, relationships with family members, and participation in leisurely social activities. To examine the relationship between emotional intelligence and social disability (Aims #2 and #3), these markers of social disability were assessed using the Social Adjustment Scale-II (SAS-II; Schooler, Weissman, & Hogarty, 1979). The SAS-II is a structured interview-based measure that assesses social disability in the areas of work (e.g., economic adequacy), household life (e.g., getting along with other household members, conjugal sexual adjustment), family life outside the household (e.g., getting along with relatives not living with the participant), social leisure (e.g., quantity and quality of interpersonal relations with non-family members, engagement in social leisure activities), and personal well-being (e.g., ability to care for oneself, life satisfaction). The instrument consists of 45 items covering the aforementioned domains, each of which is rated on a 5-point scale, with higher scores representing more social disability. Five global ratings are also provided with regard to work, household life, family life outside the household, social leisure, and general social adjustment, based on the entire interview. Global items are rated on a 7-point scale, with higher ratings reflecting more social disability.

The SAS-II has been shown to be a reliable and valid measure of social adjustment in schizophrenia, and has been widely applied to measure social disability in this population (Schooler, Weissman, & Hogarty, 1979). The SAS-II has demonstrated interrater reliability coefficients within acceptable ranges (range of κ = .64 to .90), adequate internal consistency (range of α = .92 to .99), and been shown to correspond with adjustment data provided by community informants (Bellack, Morrison, Mueser, Wade, 1990; Davies, Bromet, Schulz, Dunn, & Morgenstern, 1989; Glazer, Aaronson, Prusoff, & Williams, 1980; Schooler, Weissman, & Hogarty, 1979). In addition, the SAS-II has been shown to converge with in-vivo measures of social skill (r = .56 to .72), other measures of social adjustment (r = .49 to .86), and employment

status (r = -.32 to -.24) among persons with schizophrenia (Bellack, Morrison, Mueser, Wade, 1990; Jaeger, Berns, & Czobor, 2003; Munroe-Blum, Collins, McCleary, & Nuttall, 1996; Mueser, Salyers, & Mueser, 2001). Importantly, because the measure does not include any assessment of symptomatology, the SAS-II has generally been shown to have adequate discriminant validity from positive (r = -.09 to .39) and negative symptoms (r = .01 to .32) (Cohen, Forbes, Mann, & Blanchard, 2006; Sayers, Curran, & Mueser, 1996). Factor-analytic studies of the SAS-II indicate that the version of the instrument included in this research assesses 8 factors of social disability (Schooler, 1981, unpublished report). These include (1) interpersonal anguish (e.g., distress and friction in social relationships across domains), (2) sexual relations, (3) child-parental relations, (4) relationships with primary household members and family, (5) relationships with members outside the home and participation in social leisure activities, (6) affect toward working, (7) major role performance (e.g., economic independence, work performance), and (8) self-care.

Since the SAS-II assesses a number of other domains of social adjustment (e.g., self-care, affect toward working), and this study focuses explicitly on social disability with regard to the quantity and quality of interpersonal relations with family and friends and engagement in social leisure activities, only the interpersonal anguish, sexual relations, household/family relations, and non-familial relations/social leisure activities factors of the SAS-II were used in this research. In addition, because very few participants were parents (n = 4), the child-parental factor was not included in this research. Further, because few participants were married (n = 6), items related to conjugal sexual relations (item 34-36) were combined with those related to non-conjugal sexual relations (item 58-60) to form 3 items representing sexual relations, instead of 6 with many missing data points. For example, if participant A is married, they would have data for items 34-36, but not 58-60, so their scores on items 34-36 were carried over to items 58-60, so that all

information on sexual relations, whether conjugal or not, was located in one set of items. Aside from the context in which these different items are rated (e.g., conjugal versus non-conjugal), their content and rating scales are identical. Subscale and total scores were computed by averaging across items. For individuals who were systematically missing data on SAS-II subscales (e.g., if they are not living with someone and therefore cannot be rated using the household relations subscale), subscale scores remained missing, but total scores continued to be computed by averaging across non-missing items.

3. Confounding Variables: Neurocognition and Psychopathology

Neurocognition. To examine the *unique* relationship between emotional intelligence and social disability, above and beyond neurocognitive functioning (Aims #2 and #3), neurocognitive ability was assessed. The key neurocognitive deficits in attention, memory, and executive function that have presented in schizophrenia and been shown to be linked to social disability (see Chapter 2, section B.3.) were examined by a comprehensive neuropsychological battery assessing processing speed (a proxy for attention), working memory, verbal memory, and executive functioning. Processing speed was assessed using two tests of simple reaction time. The first test, developed by Ben-Yishay, Piasetsky, & Rattok (1985), asks participants to respond to the presentation of a critical stimulus under fixed and variable inter-stimuli intervals by pressing a computer space bar upon the presentation of the critical stimulus on a computer screen. Participants' reaction time is measured over a series of trials and averaged to provide a general measure of processing speed during the test. The second test, developed by Bracy (1994), uses a similar protocol, but asks participants to respond to critical stimuli using either their dominant or non-dominant hand, depending on the location of the stimulus on the computer screen. Participants' reaction time is again averaged over a series of trials. Both of these measures of processing speed have been widely used with general and psychiatric populations.

Considerable psychometric evidence supports their reliability among individuals with traumatic brain injury (Ben-Yishay, Piasetsky, & Rattok, 1985), and a recent study among persons with schizophrenia found 1-year retest reliability rates to be within acceptable ranges (range of r = .49 to .68) (Hogarty et al., 2004).

Working memory was assessed using the digit-span test of the revised Wechsler Adult Intelligence Scale (WAIS-R; Wechsler, 1981), a commonly used neuropsychological test for examining various forms of intellectual functioning. The digit-span test requires participants to remember several series of numbers and repeat these series both forwards and backwards. Participants are given points for each series they recall correctly. Scaled scores normed against healthy samples were used in this research, which range from 1 to 19, with higher scores indicating better working memory performance. Previous research has found the WAIS-R to have adequate reliability and validity when used with both healthy and psychiatric populations. Wechsler has demonstrated that the complete WAIS-R battery has adequate split-half internal consistency among healthy populations (r = .95), which others have found to continue to be strong among clinical populations (range of r = .73 to .97) (Zhu, Tulsky, Price, & Chen, 2001). The measure has also shown adequate factorial validity (Dickinson, Iannone, & Gold, 2002) and discriminant validity from separate, but related neurocognitive constructs among persons with schizophrenia (Green et al., 2002; Nuechterlein et al., 2004).

Verbal memory was assessed using the Stories A and B components of the Revised Wechsler Memory Scale (Wechsler, 1987; WMS-R) and the List A components of the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987). The WMS-R stories task asks participants to listen to a short story and remember and recall its details either immediately or after a short delay. Participants receive points for each detail they recall correctly when reconstructing the story. The sum of raw scores from stories A and B from the WMS-R was

used in this research, which range from 0 to 50, with higher scores indicating better verbal memory performance. The CVLT List A task asks participants to learn a 16-item grocery shopping list and proceed through a series of trials learning and recalling the list, while also being asked to learn a similar list of grocery items. Upon five trials of learning and recall, participants are asked to then recall the list after a 20 minute delay. Raw scores were used in this research, which range from 0 to 16 for each of the immediate and delayed recall trials, with high scores reflecting better verbal memory performance. The verbal memory components of the WMS-R have been shown to have adequate short-term retest reliability (r = .77) and internal consistency (range of r = .74-.75), as well as sufficient factorial and discriminant construct validity, in both normal and clinical populations (see Elwood, 1991 for review). The CVLT has also shown adequate retest reliability (range of r = .61 to .84) and construct validity (Elwood, 1995; Woods, Delis, Scott, Kramer, & Holdnack, 2006).

Executive functioning was assessed using the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993; WCST), perhaps the most common measure of executive function among persons with schizophrenia (Green, Kern, Braff, & Mintz, 2000). The WCST asks participants to sort a stack of cards based on an unknown and shifting sorting algorithm reinforced by the experimenter. The test generates several scores indicating the number of cards correctly sorted, number of perseverative and non-perseverative errors, and percentage of conceptual level (consecutively correct) responses. Frequency of correct and error responses range from 0 to 64, with higher correct responses and lower error rates indicating better executive functioning. Raw perseverative error rates were the only score used in this research. The psychometric properties of the WCST have been extensively examined among healthy and psychiatric populations (Heaton, Chelune, Talley, Kay, & Curtiss, 1993). Recent psychometric evidence among persons with schizophrenia indicate that the test has adequate retest reliability

(range of r = .52 to .76) and construct validity (Bell, Greig, Kaplan, & Bryson, 1997; Hogarty et al., 2004). Previous research has shown that persons with schizophrenia exhibit particularly significant perseverative error rates (e.g., Franke, Maier, Hain, & Klingler, 1992), which have been linked to frontal lobe pathology (Weinberger, Berman, & Zec, 1986).

To conserve power in this study, scores from each of these measures were converted to a common metric (z-scale) and averaged together to create a composite index of neurocognitive functioning. Neurocognitive scores for processing speed (various measures of simple reaction time) and executive functioning (number of perseverative errors on the WCST) were reverse coded, so that higher scores on the neurocognitive composite indicate better neurocognitive performance. Due to the longitudinal nature of these data, standardized scores were computed by scaling both baseline and year 1 data at the same time using a stacked dataset with multiple subject by time point records. This has the effect of centering participant responses around the mean neurocognitive levels during the entire course of the study, which does not obscure neurocognitive change unlike methods that isolate baseline from follow-up data during standardization. In addition, since this procedure is a linear transformation, this method allows the variance distribution to remain the same across both time points, such that if the variance at year 1 is larger than the variance at baseline, this characteristic of these data will remain the same. The result places all neurocognitive measures on a standard metric so they can be averaged and a composite index can be computed, but at the same time preserves individual differences in baseline and follow-up levels of neurocognitive function so that change can be examined through change scores.

Take for example, when neurocognitive instrument A is standardized using this method, where M_A = 1 at baseline and M_A = 2 at follow-up, indicating some improvement in the measure over time (standard deviations are not given for simplicity). Using this method, scores are

standardized with M_A = 1.5, such that M_A = -.5 at baseline and M_A = .5 at follow-up. The absolute difference between the measures at baseline and follow-up remains the same (1), even though the measure was placed on a common metric. Consequently, due to the longitudinal nature of these data, this method of standardization was used in order to preserve changes that occur over time on the neurocognitive composite.

Psychopathology. To examine the *unique* relationship between emotional intelligence and social disability, above and beyond psychopathology (Aims #2 and #3), psychopathology was assessed using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962). The BPRS is a short 18-item, clinician-rated measure of the positive and negative symptoms of schizophrenia, as well as symptoms of general psychopathology. Symptom items are rated on a Likert scale ranging from 1 ("not present") to 7 ("extremely severe") and averaged, with higher scores representing increasing degrees of psychopathology. The instrument has been widely used among persons with schizophrenia and other psychiatric disabilities for several decades, and has amassed a large body of psychometric validation (Faustman & Overall, 1999). Recent examinations have indicated that the BPRS has adequate internal consistency (α = .76), retest reliability (r = .52), and interrater reliability (range of κ = .67 to .75) (Hafkenscheid, 2000; Lachar et al., 2001). Additional investigations have also shown the BPRS to contain adequate factorial construct validity and to be predictive of length of hospitalization stay among persons with schizophrenia and other severe mental illnesses (Anderson, Crist, & Payne, 2004; Shafer, 2005). Total level of psychopathology was estimated in this research by averaging across all BPRS items.

D. PROCEDURES

Upon recruitment and study enrollment, participants in this research were randomly

assigned to receive two years of either CET or EST. Prior to the initiation of treatment, participants were assessed using the aforementioned measures of emotional intelligence, neurocognition, psychopathology, and social disability. Participants then began either CET or EST and were assessed yearly for two years using the same battery of instruments. This research only makes use of baseline and one-year follow-up data, as two-year assessments are currently still being completed. No data are available on the number or characteristics of participants who refused to participate in this research, although detailed information is available on those who began the study and did not complete a full two years of treatment. Medication compliance, dosage, and side-effects were closely monitored in all participants throughout the course of treatment by a research psychiatrist and clinical nurse specialist. Clinician-rated medication compliance indicated that less than 5% of patients showed any gross irregularities (i.e., frequently missing multiple daily dosages of medication) in compliance during the course of the study. All participants were maintained on Food and Drug Administration approved antipsychotic medications throughout the course of this research. All participants provided written, informed consent prior to participation in this research, and this study was monitored annually by the University of Pittsburgh Institutional Review Board.

E. DATA ANALYSIS

The data analytic plan for this research tested the hypotheses outlined within the specific aims above to (1) confirm the psychometric properties of the MSCEIT when applied to persons with schizophrenia; (2) examine the unique cross-sectional relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology; and (3) examine the unique longitudinal relationship between changes in emotional intelligence and changes in social disability, above and beyond changes in

neurocognitive function and psychopathology. This section will provide a detailed description of the analyses used to accomplish these aims, as well as a power analysis outlining the feasibility of this research with the available sample size.

1. Preliminary Analyses

Prior to investigating the primary analytic aims of this research, four preliminary analyses were conducted to verify internal consistency among study measures, check assumptions associated with the statistical tests proposed for this research, and inform subsequent analyses about the potential effects of demographic heterogeneity and patient attrition on estimates obtained from subsequent analyses. First, the internal consistency of SAS-II, BPRS, and neurocognitive composite were checked to ensure measurement reliability (the internal consistency of the MSCEIT is examined separately in Aim #1). Second, the distributions of continuous variables were examined for skewness and transformed using non-linear transformations and outliers were winsorized when appropriate, to meet the assumptions of parametric testing.

Third, to examine the effects of patient attrition on longitudinal treatment effects and relationship estimates, baseline differences in demographics, illness chronicity, medication compliance, emotional intelligence, neurocognitive function, psychopathology, and social disability outcome were examined between individuals who completed 1 year of treatment and those who did not using independent *t* or Fisher's exact tests, as appropriate. It was expected that no systematic differences would exist between those who completed 1 year of treatment and those who did not, as both treatment groups have had equal dropout rates.

Finally, since emotional intelligence may be related to age, gender, ethnicity and education (Mayer, Salovey, & Caruso, 2004), and these demographic characteristics, as well as illness chronicity, the presence of schizoaffective disorder, and medication compliance may all

bear some relation to social disability, psychopathology, and neurocognitive ability among this sample, variation in these demographic and clinical characteristics may need to be accounted for in subsequent analyses. To examine the degree to which these characteristics needed to be accounted for in subsequent analyses, Pearson or point-biserial correlation matrices were computed between the primary variables of interest in this research (emotional intelligence, social disability, neurocognitive ability, and psychopathology) and these demographic and clinical variables. Significant relationships between demographic/clinical characteristics, and two or more of the primary study variables prompted the need to remove shared variance between these variables using partial correlation and multiple regression in subsequent analyses.

2. Approach to Missing Data

Missing data when examining the primary aims of this research was handled using two approaches. The majority of missing data in this investigation comes from those participants who dropped out of the study before 1 year of treatment (n = 10). Such individuals cannot be assumed to be missing at random, and therefore the first approach to handling missing data in this research was to continue to count the data of these participants at 1 year follow-up as missing. The second primary source of missing data in this research concerns the 11 individuals at baseline and 1 year follow-up who were not living with other persons. Consequently, their data on the SAS-II household relations subscale are missing, which also cannot be considered to be missing at random. Therefore, these data were handled using the first approach to missing data, and were counted as missing.

The remaining missing data in this research comes from three participants, one who is missing complete data on the SAS-II and BPRS at baseline, and another two who are missing information on the sexual relations subscale of the SAS-II at baseline and year 1. These participants are not missing any other data on the primary study variables at baseline, and

therefore their scores on the SAS-II and/or BPRS are assumed to be missing at random. Recent research suggests that when data are missing at random, the current best approach for handling missing data, even with modest sample sizes, is to impute using the expectation-maximization algorithm (Schafer & Graham, 2002), which takes a maximum likelihood approach to estimating values for missing data. Consequently, for these three cases of missing data, the expectation-maximization approach was used to estimate the SAS-II and/or BPRS scores of these participants from available data on the other primary study variables, as well as demographic and clinical characteristics.

3. Aim #1: Confirm the Psychometric Properties of the MSCEIT

Investigate the internal consistency of the MSCEIT.

To evaluate the internal consistency of the MSCEIT, baseline data were pooled across treatment groups to calculate Cronbach's alpha coefficient for the full-scale MSCEIT, as well as its tasks and four branches. Using reliability guidelines suggested by Nunnelly (1978), alpha coefficients greater than .80 suggested adequate internal consistency.

Investigate the discriminant validity of the MSCEIT with regard to its divergence from measures of neurocognitive function and psychopathology.

The ability of the MSCEIT to assess a construct that is unique from general neurocognition and psychopathology was examined by calculating Pearson correlation coefficients between the MSCEIT branch and total scores, and the neurocognitive composite and BPRS. In addition, true score correlations correcting for unreliability were also estimated to gage the potential upper-bounds of these relations within this sample (Spearman, 1904b). These correlation matrices were computed at baseline only. The presence of no more than a small to moderate amount of shared variance ($r < .50$; Cohen, 1988) between MSCEIT and neurocognitive and psychopathology scores would suggest that the MSCEIT assesses a construct

that is sufficiently unique from neurocognition and psychopathology.

Investigate the factor structure of the MSCEIT.

A preliminary investigation of the factor structure of the MSCEIT was conducted using an oblique rotated, exploratory factor analysis on the 8 MSCEIT tasks that make up its branch and subscale scores. These tasks were used instead of the 141 MSCEIT items, due to the large sample size needed to estimate a reliable factor structure from such a large covariance matrix. The size and significance of the correlations among the factors were also examined. Previous research has found support for a four-factor, oblique solution in large factor-analytic studies (Mayer et al., 2003). As such, the presence of a four-factor solution (see Figure 1) for the MSCEIT using exploratory factor analysis of baseline data, with oblique rotation, would provide preliminary support for the factor structure of the MSCEIT when applied to persons with schizophrenia. It was expected that these four factors would be correlated, which was assessed by examining the correlations among the factors.

4. Aim #2: Examine the Cross-sectional Relationship Between Emotional Intelligence and Social Disability.

Hypothesis 1a. Emotional intelligence is significantly, negatively correlated with social disability at baseline.

The zero-order, cross-sectional relationship between emotional intelligence and social disability at baseline was examined by computing Pearson correlation coefficients between baseline MSCEIT total and branch scores and baseline SAS-II total and subscale scores. The presence of significant, negative relationships between these measures would indicate that emotional intelligence is negatively associated with social disability. In addition, due to the large body of evidence suggesting significant differences in social disability between males and females with the disorder, a series of exploratory analyses was conducted using moderated

multiple regression to explore whether the relationships between emotional intelligence and social disability across SAS-II and MSCEIT measures differ by gender.

Hypothesis 1b. Emotional intelligence is significantly, negatively correlated with social disability at baseline, after removing shared variance with neurocognitive function and psychopathology.

The unique, cross-sectional relationships between emotional intelligence and social disability at baseline was examined through a series of hierarchical linear regression analyses. These analyses predicted baseline SAS-II total and subscale scores from baseline MSCEIT total and branch scores, separately, after first removing shared variance with baseline neurocognitive composite and BRPS scores. The presence of significant increments in variance explained in SAS-II scores by MSCEIT scores after entering neurocognitive composite and BPRS scores into the model would indicate that emotional intelligence is significantly related to social disability, independent of neurocognitive function and psychopathology. In addition, emotional intelligence by gender interactions were again explored in these hierarchical models using moderated multiple regression.

5. Aim #3: Examine the Longitudinal Contribution of Changes in Emotional Intelligence to Social Disability.

This aim was investigated using a series of regression models designed to test the criteria for mediation outlined by the seminal work of Baron and Kenny (1986). According to Baron and Kenny, mediation can be considered to be present if (1) variations in the independent variable (treatment) account for variations in the dependent variable (changes in social disability); (2) variations in the independent variable (treatment) account for variations in the mediator (changes in emotional intelligence); (3) variations in the mediator (changes in emotional intelligence) account for variations in the dependent variable (changes in social

disability), when adjusting for the effects of the independent variable (treatment); and (4) when the relationships between the independent variable (treatment) and the mediator (changes in emotional intelligence), and the mediator (changes in emotional intelligence) and the dependent variable (changes in social disability) are controlled, the relationship between the independent variable (treatment) and the dependent variable (changes in social disability) is reduced (Baron and Kenny, 1986). These assumptions are tested with the following regression equations outlined by MacKinnon, Lockwood, Hoffman, West, and Sheets (2002) that are taken directly from Baron and Kenny's (p. 1177) framework,

$$\Delta\text{Social Disability} = \beta_{0(1)} + \tau(\text{Treatment}) + \epsilon_{(1)} \quad (1)$$

$$\Delta\text{Emotional Intelligence} = \beta_{0(2)} + \alpha(\text{Treatment}) + \epsilon_{(2)} \quad (2)$$

$$\Delta\text{Social Disability} = \beta_{0(3)} + \tau'(\text{Treatment}) + \beta(\Delta\text{Emotional Intelligence}) + \epsilon_{(3)} \quad (3)$$

where τ represents the relationship between the treatment and changes in social disability, α represents the relationship between the treatment and the changes in emotional intelligence, β represents the relationship between changes in emotional intelligence and changes in social disability adjusting for the effects of treatment on changes in social disability, τ' represents the relationship between treatment and changes in social disability adjusting for the effects of changes in emotional intelligence on changes in social disability, and $\beta_{0(1)}$, $\beta_{0(2)}$, $\beta_{0(3)}$ and $\epsilon_{(1)}$, $\epsilon_{(2)}$, $\epsilon_{(3)}$ represent the respective intercept and error terms in the models. Therefore, according to Baron and Kenny's framework, mediation is present when (1) $\tau \neq 0$, (2) $\alpha \neq 0$, (3) $\beta \neq 0$, and (4) $\tau' < \tau$. It should be noted that by meeting criteria 1 through 3, criterion 4 is mathematically assumed.

These four criteria were tested in the following hypotheses, where hypothesis 2a tests criteria 1 and 2, and hypothesis 2c tests criteria 3 and 4. In addition, since this research was particularly concerned with the relationship between emotional intelligence and social disability, an additional intermediate step (hypothesis 2b) was added to independently examine the relationship between changes in emotional intelligence and changes in social disability, which is usually necessary, but never sufficient for showing mediation (Baron & Kenny, 1986).

Hypothesis 2a. Treatment assignment (0 = control; 1 = CET) is significantly correlated with improvements in emotional intelligence and reductions social disability.

The relationship between treatment assignment and changes in emotional intelligence and social disability was examined using multiple regression by separately predicting changes in MSCEIT total and branch scores from treatment, and predicting changes in SAS-II total and subscale scores from treatment. Change scores for MSCEIT and SAS-II scores were computed by taking the residuals of year 1 scores predicted by baseline scores, to account for baseline individual differences in emotional intelligence and social disability. The presence of a significant, negative relationship between treatment assignment and SAS-II total and subscale change scores would confirm the previously documented beneficial effects of CET on social disability, and fulfill criterion 1 of Baron and Kenny's (1986) criteria for showing mediation. The presence of a significant, positive relationship between treatment assignment and MSCEIT total and branch change scores would confirm the previously documented beneficial effects of CET on emotional intelligence, and fulfill criterion 2 of Baron and Kenny's criteria for showing mediation.

Hypothesis 2b. Changes in emotional intelligence are significantly, negatively correlated with changes in social disability, both before and after adjusting for improvements in neurocognitive function and psychopathology.

The relationship between longitudinal changes in emotional intelligence and changes in social disability was examined through a series of hierarchical linear regression analyses. While these analyses are not by themselves sufficient for showing mediation in Baron and Kenny's (1986) framework, they are central to the aims of this research, where the relationship between changes emotional intelligence and changes social disability is key, regardless of the mediating effects of changes in emotional intelligence on the relationship between treatment and changes in social disability. These analyses began by separately predicting changes in SAS-II total and subscale scores from changes in MSCEIT total and branch scores in Pearson correlation analyses. After performing these analyses, the same series of analyses was performed, but first accounting for changes in neurocognitive composite and BPRS scores using hierarchical linear regression. All of these analyses made use of the residualized change scores for the SAS-II and MSCEIT created in hypothesis 2a, and the latter series of analyses created residualized change scores for neurocognitive composite and BPRS scores using the same method outlined in hypothesis 2a. The presence of significant, negative relationships between changes in MSCEIT scores and changes in SAS-II scores would indicate that improvements in emotional intelligence are related to reductions in social disability. The presence of significant, negative relationships between changes in MSCEIT scores and changes in SAS-II scores after accounting for changes in neurocognitive composite and BPRS scores would indicate that improvements in emotional intelligence are uniquely related to reductions in social disability, above and beyond changes in neurocognitive function and psychopathology. Changes in emotional intelligence by gender interactions were also explored in these analyses using moderated multiple regression to identify any significant differences in these relationships between genders.

Hypothesis 2c: Changes in emotional intelligence are partial, but significantly mediators of the negative relationship between treatment assignment (0 = control; 1 = CET) and

social disability, both before and after adjusting for the relationship between treatment assignment and changes in neurocognitive function and psychopathology.

The final criteria to satisfy Baron and Kenny's criteria for mediation were examined using a series of multiple regression analyses. Tests of hypothesis 2a have prepared for this final analysis by indicating a relationship between treatment assignment, and changes in emotional intelligence and changes in social disability. This analysis now examined whether the direct relationship between treatment assignment and changes in social disability identified in hypothesis 2a could be indirectly accounted for by the relationship between treatment and changes in emotional intelligence identified in hypothesis 2a, and the relationship between changes in emotional intelligence and changes in social disability identified in hypothesis 2b. Consequently, the degree to which longitudinal improvements in emotional intelligence serve as a *mechanism* for improving social disability was assessed.

This analysis was accomplished through a series of multiple regression analyses, separately predicting changes in SAS-II total and subscale scores from changes in MSCEIT total and branch scores and treatment assignment. These analyses were also repeated after adjusting for changes in neurocognitive composite and BPRS scores in hierarchical models, in order to examine the unique mediational effects of improvements in emotional intelligence on improvements in social disability, above and beyond changes in neurocognitive function and psychopathology. Further, emotional intelligence by gender interactions were also explored using moderated multiple regression to identify gender differences in mediational effects.

If a mediational effect is present, the presence of significant relations between changes in MSCEIT scores and changes in social disability in this model (Baron and Kenny [1986] criterion 3) would reduce the direct effect of treatment assignment on SAS-II scores from that observed in hypothesis 2a (Baron and Kenny criterion 4), and this would suggest that improvements in

emotional intelligence mediate the effect of treatment assignment on social disability. If changes in MSCEIT scores are significantly related to changes in social disability in this model, after adjusting for changes in neurocognitive composite and BPRS scores, then the direct effect of treatment assignment on SAS-II scores, after adjusting for changes in neurocognitive composite and BPRS scores would be further reduced, suggesting that improvements in emotional intelligence uniquely mediate the effect of treatment assignment on social disability, above and beyond changes in neurocognitive function and psychopathology. The size and significance of these indirect effects was evaluated using MacKinnon, Lockwood, Hoffman, West, and Sheets' (2002) asymptotic test of indirect effects. In addition, to examine the possibility of reverse mediation, this series of analyses was repeated using SAS-II scores as the mediator and MSCEIT scores as the independent variable. Taken together, *these analyses were used to identify whether social disability in schizophrenia can be reduced through manipulations (improvements) in emotional intelligence*; a robust test of the association between these constructs.

6. Power Analysis

Statistical power to detect relationships among the constructs discussed above is based upon the 57 individuals who have completed baseline assessments and, for specific tests, the subset of 47 individuals who have complete one-year follow-up assessments. While a moderate number of significance tests were conducted during the course of this analytic plan, the conventional Type I error rate is *not* adjusted in these power analyses to account for inflation of the experiment-wise Type I error rate that can come from excessive inference testing. This constitutes a limitation of the proposed research, as false positive results may occur. However, given the modest sample size, reduction of the conventional Type I error rate is likely to exceedingly increase the Type II error rate, and therefore obscure the significance of true relationships which can be confirmed in future studies employing larger samples.

All power analyses were conducted *a priori* using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007), with the exception of estimating power for MacKinnon and colleagues' (2002) asymptotic *z*-test for the significance of indirect effects, which was derived from an empirically estimated power table by Fritz and MacKinnon (2007). Table 2 presents the results of these analyses. Power analyses are not reported for exploratory analyses of the moderating effect of gender on relationships between emotional intelligence and social disability.

Table 2. *Power Analyses for Study Aims and Hypotheses*

Aim/Hypothesis	Analytic Test	1 - β	α	*n*	df	Minimum Effect Size
1	Pearson correlation	.80	.05	57	56	$r = .36$
2/1a	Pearson correlation	.80	.05	57	56	$r = .36$
2/1b	Multiple regression	.80	.05	57	1,53	$\Delta R^2 = .125$
3/2a	Multiple regression	.80	.05	47	1,45	$R = .39$
3/2b	Multiple regression	.80	.05	47	1,42	$\Delta R^2 = .15$
3/2c	Asymptotic *z*-test	.80	.05	47	46	Indirect effect = .35

Aim #1: Confirm the psychometric properties of the MSCEIT

Correlational analyses presented in the psychometric aims of this research depend upon testing the significance of Pearson correlation coefficients on complete baseline data. As can be seen in Table 2, using power analytic methods outlined by Cohen (1988), with 57 participants, given power = .80 and alpha = .05, adequate statistical power was available to detect medium ($r = .36$) relationships sizes in correlational analyses examining the MSCEIT's divergence from neurocognitive function and psychopathology. The examination of the MSCEIT's factor structure in this aim is admittedly exploratory and somewhat underpowered. While optimal guidelines for sample sizes required to estimate a reliable factor structure continue to be controversial and dependent upon a number of different parameters, recent Monte Carlo

examinations have found that reliable factor structures can be estimated with as small as a 5:1 subject to item ratio (Maccallum, Widaman, Zhang, & Hong, 1999). With 57 participants at baseline and 8 MSCEIT tasks, this research was able to meet these minimum (although perhaps not optimal) requirements for examining the factor structure of the MSCEIT.

Aim #2: Examine the cross-sectional relationship between EI and social disability.

The zero-order relationship between emotional intelligence and social disability in hypothesis 1a was examined by computing Pearson correlation matrices on the baseline data. As can be seen in Table 2, with 57 participants at baseline, power = .80, and alpha = .05, adequate power was available to detect medium (r = .36) relationship sizes between MSCEIT and SAS-II scores. The examination of the unique relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology in hypothesis 1b, relied upon a series of hierarchical linear regression analyses with 3 predictors. Also illustrated in Table 2, with 57 participants at baseline, power = .80, alpha = .05, and k = 3, adequate power was available to detect medium-sized (ΔR^2 = .125) increments in variance explained in SAS-II scores by MSCEIT scores, after first accounting for shared variance with neurocognitive composite and BPRS scores.

Aim #3: Examine the longitudinal contribution of changes in EI to social disability.

The relationship between treatment and changes in emotional intelligence and changes in social disability examined in hypothesis 2a relied upon a series of multiple regression analyses with a single predictor (treatment). As can be seen in Table 2, with 47 participants followed-up over 1 year, power = .80, alpha = .05, and k = 1, adequate power was available to detect medium to large (R = .39) relationship sizes between treatment and changes in MSCEIT and SAS-II scores. The relationship between changes in emotional intelligence and social disability in the context of treatment, changes in neurocognitive function, and changes in psychopathology tested

in hypothesis 2b relied upon a series of hierarchical linear regression models with a maximum of 4 *a priori* predictors. As shown in Table 2, with 47 participants followed-up over 1 year, power = .80, alpha = .05, and $k = 4$, adequate statistical power was available to detect medium-sized ($\Delta R^2 = .15$) effects for the unique relationship between changes in emotional intelligence and changes in social disability. Finally, as can be seen in Table 2, when estimating the significance of the indirect effect of treatment on changes in social disability through changes in emotional intelligence with MacKinnon and colleagues' (2002) asymptotic test of indirect effects in hypothesis 2c, adequate power was available to detect large indirect effects (.35 and greater) with power = .80, alpha = .05, and 47 participants followed-up over 1 year.

IV. RESULTS

This chapter presents a series of statistical analyses designed to answer the primary analytic questions of this research. These questions focus on (1) evaluating the psychometric properties of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) among persons with schizophrenia, (2) examining the cross-sectional relationship between emotional intelligence as assessed by the MSCEIT and social disability, and (3) investigating the association between longitudinal changes in emotional intelligence and changes in social disability. This chapter begins with a presentation of the demographic and clinical characteristics of the sample ascertained for this research, and then proceeds by presenting the results of a series of preliminary analyses designed to check the internal consistency of study measures, verify that study data meets criteria for parametric statistical testing, examine potential attrition bias in the sample, and investigate potential demographic and clinical confounds with primary study variables. Subsequent to these preliminary analyses, the results from the primary study aims are presented.

A. SAMPLE CHARACTERISTICS

A total of 57 persons with early course schizophrenia or schizoaffective disorder participated in this research, 47 of whom completed 1 year of treatment. As can be seen in Table 3, the majority of participants were diagnosed with schizophrenia and had been ill for less than four years. In addition, most participants were male, European American, and in their mid-twenties. Few had completed a college degree and were employed, although on average, participants had normal levels of intelligence. Importantly, treatment groups did not differ significantly with regard to any of these characteristics at baseline, suggesting that participants in

CET and EST could be safely combined at baseline. Unfortunately, no data are available regarding the amount of treatment individuals in this research had received prior to study participation.

Table 3. *Participant Demographics*

	M (*SD*)/% (*N*)			
	Baseline			
Variable	CET (*N* = 29)	EST (*N* = 28)	Combined (*N* = 57)	*p*[a]
Age	25.33 (5.73)	26.07 (6.16)	25.7 (5.91)	.641
Male	66% (19)	75% (21)	70% (40)	.565
European American	69% (20)	68% (19)	68% (39)	1.000
Completed College	31% (9)	32% (9)	32% (18)	1.000
Employed[b]	31% (9)	25% (7)	28% (16)	.770
Schizophrenia[c]	69% (20)	64% (18)	67% (38)	.783
Illness Length[d]	3.63 (3.12)	3.51 (2.44)	3.57 (2.78)	.875
IQ[e]	96.96 (10.45)	97.75 (12.82)	97.36 (11.59)	.802

Note. CET = Cognitive Enhancement Therapy (experimental group), EST = Enriched Supportive Therapy (control group), WAIS-R = Wechsler Adult Intelligence Scale-Revised.

[a]Fisher's exact test or independent *t*-test, two-tailed, for significant differences between CET and EST participants.

[b]Based on any paid employment.

[c]Remaining participants have schizoaffective disorder.

[d]Years since first psychotic episode.

[e]Based on the full Wechsler Adult Intelligence Scale-Revised. IQ data are only available for 56 individuals.

B. PRELIMINARY ANALYSES

1. Internal Consistency of the SAS-II, BPRS, and Neurocognitive Composite

Preliminary analysis of study data began by first performing a series of analyses to check the internal consistency of the primary study measures, with the exception of the MSCEIT which will be examined in the psychometric aim of this research (Aim #1), using baseline data combined across treatment groups. These analyses were conducted to estimate the reliability of

study measures, but were not the sole basis for including or excluding items within measures. Cronbach's α was used as the measure of internal consistency, with estimates of $\alpha \geq .80$ considered to be indicative of a highly internally consistent scale, and estimates $\alpha \geq .70$ considered to be indicative of a minimally adequate internally consistent scale (Nunnelly, 1978). Internal consistency estimates for scales with missing data were calculated using the expectation-maximization algorithm, which has been shown to be more accurate than listwise or pairwise deletion when computing Cronbach's α (Enders, 2003).

SAS-II. Table 4 presents internal consistency estimates of the total scale and four subscales of the SAS-II at baseline in the combined treatment group sample. The internal consistencies of the SAS-II total, interpersonal anguish, sexual relations, and household family relations subscales were all within acceptable ranges. The internal consistency of the social leisure subscale was somewhat lower, due to the small number of items assessing a broad array of social leisure participation and non-family relationships, but was still within acceptable ranges. In particular, the item "Leisure Activities" referring to degree of participation in social leisure activities demonstrated a low item-total correlation with the overall subscale. Nonetheless, given the conceptual importance of this item to tapping a range of social leisure and non-family relations domains, it has been retained as part of the overall subscale. Intercorrelations among SAS-II scales are presented in Table 5. As can be seen in the table, most SAS-II scales were highly correlated, with the exception of the sexual relations subscale, which was not significantly related to either the total or remaining subscales.

Table 4. *Social Adjustment Scale-II Internal Consistency*

Item	Alpha	Item Total	Alpha Without
Total	.82	-	-
Interpersonal Anguish	.83	-	-
Friction - Work (23)		.76	.82
Distress - Work (24)		.85	.77
Worry - Household (41)		.46	.81
Guilt - Household (42)		.38	.82
Wronged - Household (43)		.62	.80
Friction - External (44)		.40	.81
Worry - External (46)		.35	.82
Guilt - External (47)		.28	.82
Wronged - External (48)		.68	.80
Sensitivity (57)		.53	.80
Loneliness (63)		.51	.81
Social Self-Appraisal (64)		.43	.81
Social Leisure/Non-Family Relations	.71	-	-
Leisure Activities (49)		.14	.75
Social Contacts - Frequency (50)		.24	.72
Social Contacts - Degree of Activity (51)		.51	.66
Social Comfort (53)		.52	.67
Interpersonal Contacts (54)		.54	.65
Communication (55)		.58	.64
Friction (56)		.54	.64
Household/Family Relations	.75	-	-
Friction (29)		.60	.66
Adaptability (30)		.60	.66
Communication (31)		.58	.68
Expressed Feelings (33)		.46	.75
Sexual Relations	.82	-	-
Sexual Frequency (58)		.79	.64
Sexual Interest (59)		.48	.94
Sexual Problems (60)		.78	.63

Table 5. *Correlations Among Social Adjustment Scale-II Scales*

Variable	1	2	3	4	5
1. SAS-II Total	-				
2. Interpersonal Anguish	.80*	-			
3. Sexual Relations	.16	-.10	-		
4. Household Relations	.61*	.37*	.12	-	
5. Social Leisure	.71*	.36*	-.03	.32*	-

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57). SAS-II = Social Adjustment Scale-II

* $p < .05$, two-tailed.

BPRS. Internal consistency estimates for the BPRS at baseline using the combined treatment sample are presented in Table 6. While the BPRS has been shown to have a number of different factor analytic solutions (e.g., Shafer, 2005), this research was only focused on accounting for the confounding effects of psychopathology in multivariate analyses, and thus BPRS total scores were used. As can be seen in Table 6, the internal consistency of the BPRS total scale score was within acceptable ranges, with most items displaying item-total correlations above .15. It is of some note that the negative symptom items (i.e., emotional withdrawal, motor retardation, and blunted affect) showed little to no item-total correlation with the overall subscale, likely reflecting the tendency of these items to form their own domain of psychopathology (as shown in previous factor analytic studies [Shafer, 2005]). Nonetheless, given the conceptual relevance of negative symptoms to schizophrenia and social disability, these items have been retained in the computation of BPRS total scores.

Table 6. *Brief Psychiatric Rating Scale Internal Consistency*

Item	Alpha	Item Total	Alpha Without
Total	.77	-	-
Somatic Concern (1)		.27	.77
Anxiety (2)		.58	.74
Emotional Withdrawal (3)		.03	.78
Conceptual Disorganization (4)		.64	.74
Guilt Feelings (5)		.19	.77
Tension (6)		.38	.76
Mannerisms/Posturing (7)		.42	.76
Grandiosity (8)		.38	.76
Depressive Mood (9)		.33	.76
Hostility (10)		.32	.76
Suspiciousness (11)		.66	.73
Hallucinatory Behavior (12)		.52	.75
Motor Retardation (13)		-.03	.79
Uncooperativeness (14)		.48	.76
Unusual Though Content (15)		.67	.73
Blunted Affect (16)		.01	.79
Excitement (17)		.52	.75
Disorientation (18)		.10	.77

Neurocognitive composite. Finally, Table 7 presents internal consistency estimates of the neurocognitive composite measure at baseline using the combined treatment sample. The internal consistency of this composite was within acceptable ranges, however the neuropsychological test of simple reaction time at fixed inter-stimulus intervals demonstrated a low item-total correlation with the overall composite. In contrast to the items that demonstrated low item-total correlations in other measures employed in this research, this item does not have the weight of conceptual relevance to this study that would justify its inclusion in the overall composite to the detriment of its internal consistency. Further, there remains three additional measures of reaction time for assessing attention that all demonstrate adequate item-total

correlations with the overall composite. Consequently, the measure of reaction time at fixed inter-stimulus intervals was removed from the computation of the neurocognitive composite in favor of improving the reliability of this composite.

Table 7. *Neurocognitive Composite Internal Consistency*

Item	Alpha	Item Total	Alpha Without
Total	.77	-	-
Simple Reaction Time: Fixed Inter-stimulus Interval		.12	.79
Simple Reaction Time: Variable Inter-stimulus Interval		.36	.76
Choice Reaction Time: Dominant Hand		.22	.78
Choice Reaction Time: Non-Dominant Hand		.34	.76
WAIS-R: Digit-span test		.45	.75
WMS-R: Stories A and B, immediate recall		.59	.73
WMS-R: Stories A and B, delayed recall		.56	.73
CVLT: List A, trials 1-5 total immediate recall		.64	.72
CVLT: List A, trials 1-5 total delayed recall		.63	.72
WCST: Number of perseverative errors		.47	.75

Note. WAIS-R = Wechsler Adult Intelligence Scale-Revised, WMS-R = Wechsler Memory Scale-Revised, CVLT = California Verbal Learning Test, WCST = Wisconsin Card Sorting Test

2. Verifying Parametric Analytic Assumptions

After checking the internal consistency of the primary study measures, a series of analyses was conducted to examine the distributions of these measures and ensure they met the assumptions for parametric testing. These analyses were conducted by visually inspecting Box and Whisker plots of each of the measures to identify potential outliers, calculating skewness statistics to quantify skewed data distributions, and visually inspecting histograms of data distributions to identify potentially non-normal (e.g., bimodal) distributions. Individual cases were identified as outliers if their score on a single measure was 1.5 times the interquartile range

of the distribution of scores in the sample (Hoaglin, Iglewicz, & Tukey, 1986). Skewness statistics greater than .75 were considered indicative of moderately skewed distributions (McAweeney & Klockars, 1998). When only a couple of outlier cases were identified in an otherwise unskewed measurement distribution, a winsorization procedure was used to bring the outlier cases within 1.5 times the interquartile range of the data distribution, by setting the score value of the outlier to that of the next closest number within 1.5 times the interquartile range (Dixon & Tukey, 1968). Primary analyses were conducted on the resulting winsorized dataset, and secondary analyses (presented in Appendix A) were conducted with outlier case inclusion and deletion for completeness. No substantial differences in the interpretation of the results occurred with different analytic approaches to the treatments of outliers. For distributions that displayed significant skewness, non-linear transformation procedures were used to transform the data distribution to reduce skewness. In order to preserve comparability between baseline and follow-up measures, both baseline and 1 year data were identically transformed, even if the distribution was significantly skewed at only one time point.

Table 8 presents descriptive statistics and skewness information for the primary study variables. As can be seen, approximately half of the variables required a non-linear transformation or winsorization procedure to reduce skewness or remove outliers. Nonetheless, once transformed or winsorized, all study variables demonstrated acceptable ranges of skewness and contained no significant outliers. Four variables (SAS-II interpersonal anguish, household relations, social leisure subscales, and BPRS total scores) demonstrated significant skewness at 1 year follow-up that was not evident at baseline. After applying logarithmic transformations to these four variables, skewness was substantially reduced at 1 year follow-up, and baseline distributions continued to exhibit adequate levels of skewness. All subsequent analyses will make use of these transformed and winsorized variables, and any transformation applied to them

will not be referred to hereafter.

Table 8. *Descriptive and Skewness Statistics of Primary Study Variables*

Variable	$N_{missing}$[a]	*N*	*M*	*SD*	Min	Max	Skew (pre)	Transform	Skew (post)
				Baseline					
MSCEIT Total	0	57	86.03	15.72	54.09	121.70	.07		
Perceiving Emotions	0	57	91.46	16.19	50.53	131.82	.17		
Facilitating Emotions	0	57	93.59	17.52	46.59	123.24	-.30		
Understanding Emotions	0	57	87.23	12.24	58.03	115.01	-.06	win(2)	-.07
Managing Emotions	0	57	86.87	12.13	63.50	110.67	-.12		
SAS-II Total	1	57	1.48	.50	.46	2.50	.02		
Interpersonal Anguish	1	57	1.07	.64	.08	2.86	.61	$x^{1/2}$	-.03
Sexual Relations	2	57	3.54	1.18	.00	5.00	-1.31	x^2	-.51
Household/Family Relations[b]	11	47	1.08	.75	.00	3.00	.49	log(x)	-.05
Social Leisure/Non-Family Relations	1	57	1.45	.70	.14	3.20	.53	log(x) win(1)	.06
BPRS Total	1	57	39.91	10.40	15.05	64.00	.44	log(x) win(1)	.33
Neurocognitive Composite	0	57	-.05	1.02	-2.81	1.85	-.70	win(4)	-.18
				Year 1					
MSCEIT Total	0	47	88.52	15.15	57.87	117.82	-.07		
Perceiving Emotions	0	47	92.01	16.72	57.47	132.27	.33		
Facilitating Emotions	0	47	92.97	17.09	60.55	127.53	.02		
Understanding Emotions	0	47	91.49	11.25	63.65	115.38	-.11		
Managing Emotions	0	47	89.00	10.76	68.07	107.63	-.29		
SAS-II Total	0	47	1.22	.49	.15	2.35	.36		
Interpersonal Anguish	0	47	.79	.55	.08	2.14	.82	$x^{1/2}$	.23
Sexual Relations	1	47	3.38	1.33	.00	5.00	-1.28	x^2	-.54
Household/Family Relations[b]	11	36	.85	.68	.00	2.75	.96	log(x)	.33
Social Leisure/Non-Family Relations	0	47	1.14	.82	.00	3.43	1.00	log(x)	.36
BPRS Total	0	47	34.06	9.37	21.00	62.00	.89	log(x)	.26
Neurocognitive Composite	0	47	.05	.99	-2.24	1.80	-.27		

Note. Skew (pre) refers to skewness before non-linear transformation. Skew (post) refers to skewness after non-linear transformation. win(n) = winsorization procedure performed on n outliers.

[a]Missing data were imputed using the expectation-maximization approach.

[b]11 individuals at baseline and 11 at year 1 were systematically missing data due to living alone, and thus imputation was not performed for these cases.

3. Examining Potential Attrition Bias

Having winsorized outliers and transformed variables to reduce skewness, a series of analyses was then conducted to examine the possibility of systematic differences between those participants for whom data were available at 1 year follow-up (i.e., those who completed 1 year of treatment; $n = 47$) and those participants for whom data were only available at baseline (i.e., those lost to attrition; $n = 10$). These analyses were conducted by calculating descriptive statistics for the primary study variables, as well as demographic and clinical characteristics, between these two groups in the combined treatment sample at baseline, and then conducting independent *t* or Fisher's exact tests to identify significant between-group differences at baseline on these variables.

As can be seen in Table 9, the only statistically significant baseline difference between those participants with available 1 year follow-up data and those lost to attrition was with regard to psychopathology, in that participants who were lost to attrition demonstrated significantly higher levels of psychopathology at baseline than those with available 1 year follow-up data. In addition, while not statistically significant, a larger proportion of European American individuals had available 1 year follow-up data. These results suggest that baseline and follow-up samples are generally comparable on the majority of primary study variables, but systematic differences do exist with regard to initial levels of psychopathology and ethic group.

Table 9. *Comparison of Baseline Characteristics of Participants With and Without 1 Year Follow-up Data*

Variable	Completed (*N* = 47) *M* (*SD*)/% (*N*)	Dropped Out (*N* = 10) *M* (*SD*)/% (*N*)	*p*[a]
Age	3.23 (.22)	3.19 (.20)	.574
Male	68% (32)	80% (8)	.706
European American	74% (35)	40% (4)	.058
Completed College	34% (16)	20% (2)	.478
Employed	26% (12)	40% (4)	.443
Schizophrenia	68% (32)	60% (6)	.717
Illness Length	3.61 (2.58)	3.83 (3.77)	.808
IQ	96.65 (11.17)	100.6 (13.54)	.334
Medication Compliant	89% (42)	89% (8)	1.000
CET Treatment Assignment	49% (23)	60% (6)	.730
MSCEIT Total	85.47 (15.45)	88.66 (17.58)	.565
Perceiving Emotions	90.79 (16.62)	94.64 (14.32)	.499
Facilitating Emotions	94 (16.34)	91.66 (23.24)	.706
Understanding Emotions	87.21 (12.02)	87.25 (12.17)	.993
Managing Emotions	86.39 (11.51)	89.17 (15.19)	.515
SAS-II Total	1.44 (.50)	1.70 (.43)	.127
Interpersonal Anguish	.96 (.31)	1.10 (.34)	.240
Sexual Relations	14.13 (6.91)	12.68 (5.67)	.539
Household/Family Relations	.64 (.36)	.82 (.36)	.194
Social Leisure/Non-Family Relations	.84 (.27)	.95 (.33)	.256
BPRS Total	3.63 (.23)	3.8 (.25)	.044
Neurocognitive Composite	.01 (.90)	-.01 (.91)	.950

Note. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale II, BPRS = Brief Psychiatric Rating Scale

[a]Fisher's exact test or independent *t*-test, two-tailed, for significant differences between completed and dropped out participants.

4. Identifying Potential Demographic and Clinical Confounds With Study Variables

After examining potential systematic differences between participants with available 1 year follow-up data and those lost to attrition, a series of correlation analyses was conducted to examine the associations between the primary study variables (i.e., emotional intelligence, social disability, psychopathology, and neurocognitive function) and potential clinical and demographic confounders at baseline using the combined treatment sample. Based on previous research, these confounders included the demographic characteristics of age, gender, education and ethnicity, as well as the clinical characteristics of illness duration, diagnosis (i.e., schizophrenia or schizoaffective disorder), and clinician estimated medication compliance (i.e., compliant vs. non-compliant).

As can be seen by the correlation matrix presented in Table 10, both gender and illness length exhibited significant and moderate associations with a number of the primary study variables. In particular, these variables were associated with components of both emotional intelligence and social disability, indicating their potentially confounding influence on estimates of the relationship between these two constructs. In addition, both education and diagnosis also showed some significant association with emotional intelligence. As a consequence, subsequent analyses will adjust for gender, education, illness length and diagnosis when examining the relationship between emotional intelligence and social disability. Further, psychometric analyses of the MSCEIT will also take into account these associations to elucidate the effects of shared variance with these demographic and clinical characteristics on estimates of the psychometric properties of the instrument.

Table 10. *Associations Between Primary Study Variables and Potential Confounders at Baseline*

Variable	Age	Gender[a]	Ethnicity[b]	Education[c]	Illness Length	Diagnosis[d]	Medication Compliance[e]
MSCEIT Total[f]	-.13	.27*	.19	.27*	-.17	-.25	.07
Perceiving Emotions	-.21	.23	.19	.20	-.32*	-.04	.00
Facilitating Emotions	-.12	.19	.20	.20	-.10	-.28*	.05
Understanding Emotions	.02	.23	.11	.32*	-.10	-.16	.12
Managing Emotions	-.09	.24	.08	.16	.01	-.24	.08
SAS-II Total[g]	.10	-.40*	-.13	-.10	.16	.04	.05
Interpersonal Anguish	.24	-.27	-.13	-.02	.45*	-.08	.25
Sexual Relations	-.05	-.12	.04	-.07	-.19	.17	-.24
Household Relations	.02	-.25	-.11	-.07	-.07	.08	-.07
Social Leisure	-.08	-.31*	-.07	-.04	-.14	.06	-.20
BPRS Total[g]	.03	-.47*	-.18	-.29*	.37*	-.08	.11
Neurocognitive Composite[f]	-.04	.14	.22	.21	-.08	-.05	.15

Note. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale II, BPRS = Brief Psychiatric Rating Scale.
[a]1 = Male, 2 = Female
[b]1 = Non-European American, 2 = European American
[c]1 = No College, 2 = College
[d]1 = Schizoaffective Disorder, 2 = Schizophrenia
[e]1 = Complaint, 2 = Non-Complaint
[f]Higher scores indicate better performance
[g]Lower scores indicate better functioning/less symptomatology
* $p < .05$, two-tailed.

C. AIM #1: CONFIRM THE PSYCHOMETRIC PROPERTIES OF THE MSCEIT

1. Internal Consistency

Having conducted a series of preliminary analyses to investigate the quality of study data and potential attrition, demographic, and clinical confounds of the relationship between

emotional intelligence and social disability, the psychometric properties of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) were examined. This investigation began by first assessing the internal consistency of MSCEIT total, branch, and task scales on the combined treatment sample at baseline, to gain an understanding of the measure's reliability among persons with schizophrenia. As can be seen in Table 11, the MSCEIT total and branch scores demonstrated adequate levels of internal consistency. Estimates were somewhat lower for individual tasks, with the blends and emotion management tasks displaying suboptimal levels of internal consistency. These internal consistency estimates for MSCEIT total, branch, and task scores are quite similar to those found in a large sample of healthy individuals (Mayer, Salovey, Caruso, & Sitarenios, 2003).

Given that the MSCEIT shared significant amounts of variance with gender, education, illness duration, and diagnosis (see Table 10), adjusted internal consistency estimates were also computed from partial correlation matrices partialing out this shared variance to determine the degree to which sample heterogeneity in these characteristics may have contributed to inflated internal consistency estimates. As shown in Table 11, estimates of internal consistency did not change substantially when adjusting for shared variance among the MSCEIT, gender, education, illness duration, and diagnosis. The internal consistency of total and branch scores all remained within acceptable levels, and individual task scores continued to demonstrate attenuated levels of internal consistency. Taken together, these results suggest that the total and branch components of the MSCEIT possess adequate levels of internal consistency among persons with schizophrenia. However, consistent with evidence from healthy samples, it would appear that caution should be used before employing individual MSCEIT tasks, which demonstrate suboptimal levels of internal consistency.

Table 11. *Internal Consistency of The Mayer-Salovey-Caruso Emotional Intelligence Test*

Scale	Alpha	Adjusted Alpha[a]
MSCEIT Total	.94	.93
Branch 1 - Emotion Perception	.91	.89
Task 1 - Faces	.82	.81
Task 2 - Pictures	.91	.89
Branch 2 - Emotion Facilitation	.82	.81
Task 3 - Facilitation	.77	.74
Task 4 - Sensations	.73	.72
Branch 3 - Emotion Understanding	.78	.76
Task 5 - Changes	.70	.68
Task 6 - Blends	.52	.51
Branch 4 - Emotion Management	.81	.80
Task 7 - Management	.67	.67
Task 8 - Relationships	.73	.72

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57).
MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test

[a]Alpha adjusted for gender, education, illness length and diagnosis.

2. Discriminant Validity

After investigating the internal consistency of the MSCEIT at baseline, the discriminant validity of the instrument among persons with schizophrenia was assessed by examining its shared variance with two key potential correlates: neurocognitive ability and psychopathology. This was accomplished by computing Pearson correlation coefficients between MSCEIT, BPRS total and neurocognitive composite scores at baseline using the combined treatment sample. In addition, given that the MSCEIT shared significant amounts of variance with gender, education, illness duration, and diagnosis, partial correlation coefficients were also computed removing shared variance between these characteristics and MSCEIT, neurocognitive composite, and BPRS total scores to examine the degree to which neurocognitive ability and psychopathology contaminate assessments of emotional intelligence beyond these demographic and clinical characteristics. Partial, rather than semi-partial correlation coefficients were computed because

neurocognitive functioning and psychopathology also shared some variance with these demographic characteristics, although this shared variance was not always statistically significant (see Table 10). Finally, true score correlation estimates were computed for zero-order correlations by correcting these estimates for measurement error, to gain an understanding the potential upper bounds of these relationships.

As can be seen in Table 12, MSCEIT total and branch scores shared a modest, but significant amount of variance with neurocognitive functioning. The branch of the MSCEIT that showed the strongest relationship with neurocognitive functioning was the emotion understanding branch, which may reflect a heavier reliance on basic cognitive processes for accessing conceptual knowledge about emotions, although the emotion facilitation and understanding branches shared similar amounts of variance with neurocognitive ability. On the other hand, the emotion management branch, which conceptually may rely the least on basic cognitive processes, did not share a significant amount of variance with neurocognitive functioning. This pattern of results remained the same when adjusting for shared variance with gender, education, illness duration, and diagnosis, indicating that neurocognitive functioning does share some variance with emotional intelligence beyond these demographic and clinical characteristics. True score estimates of zero-order relationships adjusting for measurement error (Spearman, 1904b) also indicated the same pattern of results. Further, all estimates remained within the small to medium sized range, and were far from a perfect correlation of 1.00.

With regard to psychopathology, only the emotion perception and understanding branches of the MSCEIT shared significant amounts of variance with BPRS total scores (see Table 12). Further, little to no relationship was observed between MSCEIT and BPRS scores, after adjusting for shared variance with gender, education, illness duration, and diagnosis, suggesting little relationship between emotional intelligence and psychopathology above and

beyond these demographic and clinical characteristics. Finally, true score correlation estimates were all far from a perfect correlation of 1.00, generally within small to medium sized ranges, and continued to suggest only substantial relationships with emotion perception and emotion understanding.

Taken together, these findings suggest that the MSCEIT shares only small to moderate amounts of variance with neurocognitive functioning and psychopathology, which are attenuated to varying degrees after adjusting for shared variance with gender, education, illness duration, and diagnosis. The upper bounds of these relationships are also within the small to the medium-sized ranges defined by Cohen (1988), which points to emotional intelligence as a construct that is related to, but not necessarily the same as neurocognitive functioning or psychopathology in schizophrenia. These findings support the discriminant validity of the MSCEIT among this population.

Table 12. *Associations Between Emotional Intelligence, Neurocognition, and Psychopathology*

	Neurocognitive Composite[a]		BPRS Total[b]	
Variable	Correlation	True Score Relationship Estimate[c]	Correlation	True Score Relationship Estimate[c]
MSCEIT Total[a]	.39* (.33*)	.45	-.25 (-.05)	-.30
Emotion Perception	.34* (.28*)	.41	-.32* (-.07)	-.38
Emotion Facilitation	.34* (.30*)	.43	-.19 (-.08)	-.24
Emotion Understanding	.37* (.31*)	.48	-.29* (-.14)	-.37
Emotion Management	.26 (.22)	.32	-.08 (-.04)	-.10

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline ($N = 57$). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, BPRS = Brief Psychiatric Rating Scale

[a]Higher scores indicate better performance

[b]Lower scores indicate less psychopathology

[c]True score estimates were corrected for unreliability using Spearman's (1904b) method

* $p < .05$, two-tailed.

3. Factor Structure

Having found evidence for the reliability and discriminant validity of the MSCEIT among persons with schizophrenia, a preliminary investigation of the factor structure of the MSCEIT among this population was conducted. A series of exploratory factor analyses was conducted using principal axis factoring, with oblique oblimin rotation when multiple factors were present, on a correlation matrix of the 8 MSCEIT task scores using the combined treatment sample at baseline (see Table 13). An exploratory approach was taken for two reasons. First, the modest sample size in this research precludes confirmatory factor analysis. Second, prior work has employed only confirmatory techniques examining a limited number of theoretical models of emotional intelligence. As other psychometricians have cautioned (Tomarken & Waller, 2003), confirmatory factor-analytic approaches can overestimate the power of a theoretical model by potentially settling prematurely for models that only provide an adequate description of the

observed data and ignoring many alternative models that may provide an improved representation of the observed data. To date, previous research has suggested the presence of 1-, 2-, and 4-factor solutions for the MSCEIT among healthy populations (Mayer, Salovey, & Caruso, 2004), however alternative solutions may provide a better representation of the emotional intelligence construct among persons with schizophrenia that could be overlooked by confirmatory techniques. As can be seen by the screeplot presented in Figure 3, two of the MSCEIT components had eigen values greater than or equal to 1, suggesting at most a 2-factor solution. Nonetheless, 1-, 3-, and 4-factor solutions were examined from principal axis analyses, in addition to a 2-factor solution, to explore the degree to which the individual MSCEIT tasks fit different factor analytic solutions for the instrument identified among healthy populations.

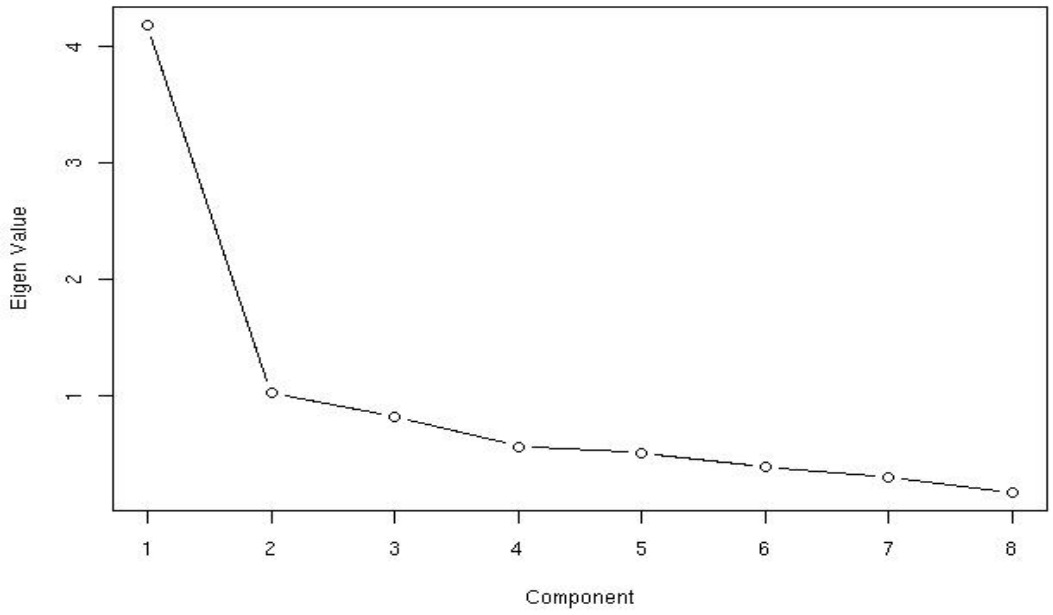
Table 13. *Correlations Among Mayer-Salovey-Caruso Emotional Intelligence Test Tasks*

MSCEIT Task	1	2	3	4	5	6	7	8
Branch 1 - Emotion Perception								
1. Task 1 - Faces	-							
2. Task 2 - Pictures	.42*	-						
Branch 2 - Emotion Facilitation								
3. Task 3 - Facilitation	.39*	.20	-					
4. Task 4 - Sensations	.20	.52*	.44*	-				
Branch 3 - Emotion Understanding								
5. Task 5 - Changes	.41*	.50*	.45*	.52*	-			
6. Task 6 - Blends	.43*	.45*	.38*	.50*	.61*	-		
Branch 4 - Emotion Management								
7. Task 7 - Management	.23	.37*	.55*	.61*	.35*	.53*	-	
8. Task 8 - Relationships	.28*	.35*	.44*	.62*	.55*	.54*	.69*	-

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57).
MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test

* $p < .05$, two-tailed.

Figure 3. *Screeplot of Eigen Values For The Mayer-Salovey-Caruso Emotional Intelligence Test*



As can be seen in Table 14, a 1 and 2-factor solution both provided an adequate fit fotor the observed MSCEIT data, with a 2-factor solution explaining a significant, but marginally greater amount of variance among the items than a 1-factor solution, $\Delta R^2 = .02$, $\Delta\chi^2(7, N = 57) = 19.80$, $p = .006$. As suggested by the screeplot, both 3- and 4-factor solutions were generally uninterpretable. The commonly found 4-factor solution for the MSCEIT did not converge and thus is not provided, and the 3-factor solution yielded many split loadings and did not result in a significantly better fit to the observed data than a 2-factor solution, $\Delta R^2 = .03$, $\Delta\chi^2(6, N = 57) = 8.16$, $p = .227$, and therefore is also not presented. Similar patterns of results were observed when estimating the factor structure of the MSCEIT using a partial correlation matrix that removed shared variance with gender, education, illness duration, diagnosis, neurocognitive functioning, and psychopathology.

Table 14. *Factor Structure of the Mayer-Salovey-Caruso Emotional Intelligence Test*

	1-Factor Solution[a]	2-Factor Solution[b]	
Variable	Factor 1	Factor 1	Factor 2
	Standardized Factor Loadings		
Branch 1 - Emotion Perception			
Task 1 - Faces	**.47**	-.16	**.72**
Task 2 - Pictures	**.59**	.07	**.59**
Branch 2 - Emotion Facilitation			
Task 3 - Facilitation	**.60**	**.42**	.23
Task 4 - Sensations	**.75**	**.62**	.20
Branch 3 - Emotion Understanding			
Task 5 - Changes	**.72**	.11	**.70**
Task 6 - Blends	**.74**	.27	**.55**
Branch 4 - Emotion Management			
Task 7 - Management	**.73**	**.92**	-.09
Task 8 - Relationships	**.76**	**.72**	.13
	Factor Correlations		
Factor	Factor 1	Factor 2	
2-Factor Solution			
Factor 1	-		
Factor 2	.62	-	

Note. Factor loadings greater than .30 appear in boldface. Analyses were conducted on the combined treatment sample at baseline (N = 57).

[a]R^2 = .46, $\chi^2(20, N = 57) = 41.18$, p = .004

[b]R^2 = .48, $\chi^2(13, N = 57) = 21.38$, p = .066

Interestingly, although a 2-factor solution appeared to best represent the observed data, the pattern of results observed for this solution was not congruent with 2-factor solutions found among healthy populations. Whereas factor-analytic studies of the MSCEIT among healthy populations have found Branches 1 and 2 to load on the same factor and Branches 3 and 4 to load on the same factor (Mayer, Salovey, Caruso, & Sitarenios, 2003), this current research found Branches 1 and 3 to load on the same factor and Branches 2 and 4 to load on the same factor. As can be seen in Table 15, zero-order correlations among the raw MSCEIT branch

scores also lent some support to this 2-factor solution, in that Branch 1 correlated strongly with Branch 3, and Branch 2 correlated most strongly with Branch 4 (although inter-correlations with other branches were also moderate to large).

Table 15. *Correlations Among Mayer-Salovey-Caruso Emotional Intelligence Test Branches*

MSCEIT Branch	1	2	3	4
1. Branch 1 - Emotion Perception	-			
2. Branch 2 - Emotion Facilitation	.48*	-		
3. Branch 3 - Emotion Understanding	.58*	.61*	-	
4. Branch 4 - Emotion Management	.38*	.70*	.59*	-

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57).
MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test

* $p < .05$, two-tailed.

Taken together, these results suggest a factor-analytic solution that is at variance with all existing factor-analytic studies of the MSCEIT among healthy populations and indicate the need for further examination of the factor structure of this instrument among persons with schizophrenia. Although these results are tentative given the small sample size employed in this research, this evidence clearly points to a simpler structure among persons with schizophrenia than the 4-factor structure proposed by Mayer and colleagues (2003). These results largely support an alternative 2-factor solution for the MSCEIT among this population, consisting of Branches 1 and 3 and Branches 2 and 4, although a single factor solution cannot be ruled out, as such a solution accounted for nearly as much variance among the items as a 2-factor solution and both factors were strongly correlated (see Table 14). Nonetheless, a 2-factor solution did produce a significant, although modest improvement in model fit over a single factor solution.

To date, factor-analytic studies of the MSCEIT among healthy individuals have all employed confirmatory approaches, so it is possible that the alternative factor solution uncovered

in this current research exists in previous studies of healthy populations, but has merely gone unexamined due to the sole use of *a priori* models and confirmatory factor-analytic techniques. Further, while at variance with Mayer and Salovey's (1997) theoretical model of emotional intelligence, the 2-factor solution identified in this research does make some clear conceptual sense, in that emotion perception and understanding both rely heavily on emotional knowledge, whereas emotion facilitation and management can be thought of as "hot" social-cognitive processes that may better reflect an ability to regulate emotions through cognitive facilitation or inhibition, than recall and process declarative knowledge about emotions. Consequently, these two factors can largely be conceptualized as *emotional knowledge* and *emotional regulation* factors, and will be referred to as such throughout the remainder of this research. Additionally, the single factor derived from the 1-factor solution can be conceptualized as a *general ('g') emotional intelligence* factor, analogous to the general intelligence factor identified in cognitive research (Spearman, 1904a), and will be referred to as such.

Given these results, a number of different scoring methods for the MSCEIT seem logical for subsequent analyses. An empirical approach for primary analyses that focuses on the 1- and 2-factor analytic solutions identified for the MSCEIT will be used for addressing subsequent questions in this research. To enhance generalizability, raw scores instead of factor scores will be used. To represent the 2-factor solution, two subscales will be formed by averaging across the MSCEIT tasks that loaded the highest on their respective factors. As such, the emotional regulation factor will be computed by averaging across MSCEIT tasks 3, 4, 7, and 8; and the emotional knowledge factor will be computed by averaging across MSCEIT tasks 1, 2, 5, and 6 (see Table 14). MSCEIT total scores will be used to represent the 1-factor solution identified in this research. In addition, secondary analyses are also conducted with conventional MSCEIT branch scores and presented in Appendix B, to examine the relationship between MSCEIT scores

and social disability using traditional scoring methods for the instrument.

D. AIM #2: EXAMINE THE CROSS-SECTIONAL RELATIONSHIP BETWEEN EMOTIONAL INTELLIGENCE AND SOCIAL DISABILITY

1. Bivariate Relationship Between Emotional Intelligence and Social Disability

After examining the psychometric properties of the MSCEIT, the first step was taken in elucidating the relationship between emotional intelligence and social disability by examining the bivariate, cross-sectional relationships between MSCEIT and SAS-II scores at baseline. As can be seen in Table 16, only small and non-significant relationships were observed between MSCEIT scores and SAS-II total and subscale scores. Relationship estimates were largest for the relationship between the MSCEIT general emotional intelligence and emotional regulation scores and SAS-II total and interpersonal anguish scores, although all these relationships were also quite small and not statistically significantly different from zero. After adjusting for the potential demographic confounds of gender, education, illness length and diagnosis using partial correlation analysis, most relationship estimates approached zero and all remained statistically non-significant. Such findings surprisingly suggest little to no cross-sectional relationship between emotional intelligence and social disability among persons with schizophrenia.

Table 16. *Associations Between Emotional Intelligence and Social Disability at Baseline*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
General Emotional Intelligence[a]	-.12 (.03)	-.14 (.04)	-.02 (.00)	-.07 (-.03)	.01 (.05)
Emotional Regulation	-.10 (-.01)	-.13 (-.06)	-.03 (.01)	-.06 (-.03)	-.02 (.01)
Emotional Knowledge	-.08 (.10)	-.09 (.14)	.09 (.10)	.05 (.12)	.02 (.07)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline (N = 57). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

Similar patterns of results were obtained from exploratory analyses of intercorrelations at year 1 follow-up (see Table 17). The primary exception includes the domain of household relations, which was related to both general emotional intelligence and emotional regulation at year 1. These relationships remained significant or marginal after adjusting for demographic and clinical confounds. In addition, emotional knowledge was positively related to interpersonal anguish, after adjusting clinical and demographic for confounds, perhaps reflecting a relationship between heightened emotional awareness and recognition of one's social problems.

Table 17. *Associations Between Emotional Intelligence and Social Disability at Follow-up*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
General Emotional Intelligence[a]	-.03 (.03)	.07 (.16)	-.04 (-.10)	-.34* (-.27†)	-.08 (-.06)
Emotional Regulation	-.06 (-.03)	.07 (.10)	-.04 (-.09)	-.41* (-.38*)	-.09 (-.07)
Emotional Knowledge	.08 (.19)	.14 (.32†)	.05 (-.04)	-.12 (-.02)	-.01 (.05)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at follow-up ($N = 47$). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

†$p < .15$, * $p < .05$, two-tailed.

2. Relationship Between Emotional Intelligence and Social Disability, Adjusting For Neurocognition and Psychopathology

Having found that emotional intelligence was not significantly related to social disability among persons with schizophrenia at baseline, the relationship between emotional intelligence and social disability, after adjusting for neurocognitive function and psychopathology was investigated. While such an analysis may seem superfluous, given the null bivariate results, this approach affords at least two benefits beyond the previous bivariate analyses. First, the possibility of a masking suppression effect of neurocognitive function and psychopathology cannot be overlooked, given that the aspects of emotional intelligence that are most strongly associated with social disability may only be those that are independent of neurocognitive function and psychopathology. Further, the fact that a positive indirect effect of emotional intelligence on social disability through neurocognitive function and psychopathology (see Table 12) may exist in addition to a negative direct effect of emotional intelligence on social disability also suggests the possibility of suppression (Tzelgov & Henik, 1991). Second, this multivariate

approach also allows for the examination of the potential moderating effect of gender on the relationship between emotional intelligence and social disability.

Table 18 presents the results of a series of hierarchical linear regression models examining the relationship between MSCEIT scores and SAS-II total and subscale scores at baseline, as well as the moderating effect of gender on these relationship estimates, after adjusting for the effects of demographic and clinical confounds, neurocognitive function, and psychopathology. As can be seen in this table, no significant relationships were observed between MSCEIT general emotional intelligence, emotional regulation, or emotional knowledge scores and SAS-II total and subscale scores in this multivariate context. In addition, no significant interactions were observed between MSCEIT scores and gender, when predicting SAS-II total and subscale scores, indicating the absence of a substantive moderating effect of gender on relationship estimates. When combined with results from previous bivariate analyses, such results continue to suggest little to no cross-sectional relationship between emotional intelligence and social disability. As such, the hypotheses that a significant negative relationship would exist between MSCEIT and SAS-II scores in bivariate (Hypothesis 1a) and multivariate analyses adjusting for neurocognition and psychopathology (Hypothesis 1b) were not supported.

Table 18. *Associations Between Emotional Intelligence and Social Disability After Adjusting for Neurocognitive Function and Psychopathology*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2	ΔR^2 = .12*			ΔR^2 = .20*			ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .02		
Neurocognitive Composite	.05	.07	.09	.05	.04	.15	-.27	1.07	-.04	.03	.08	.07	-.03	.04	-.08
BPRS Total	.96	.32	.46*	.76	.18	.58*	.17	5.09	.01	.08	.30	.05	.16	.20	.14
General Emotional Intelligence															
Step 3	ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .01		
General Emotional Intelligence[b]	.00	.00	.02	.00	.00	.00	.00	.07	.01	-.00	.00	-.04	.00	.00	.10
Step 4	ΔR^2 = .02			ΔR^2 = .02			ΔR^2 = .00			ΔR^2 = .01			ΔR^2 = .01		
General Emotional Intelligence X Gender	.01	.01	.88	.01	.00	.81	.06	.13	.39	-.01	.01	-.73	.00	.01	.73
Emotional Regulation															
Step 3	ΔR^2 = .00			ΔR^2 = .01			ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .01		
Emotional[b] Regulation	.00	.01	-.04	-.00	.00	-.11	.02	.09	.03	-.00	.01	-.05	.00	.00	.04
Step 4	ΔR^2 = .01			ΔR^2 = .02			ΔR^2 = .00			ΔR^2 = .03			ΔR^2 = .00		
Emotional Regulation X Gender	.01	.01	.91	.01	.01	1.21	-.06	.19	-.40	-.01	.01	-1.69	.00	.01	.56
Emotional Knowledge															
Step 3	ΔR^2 = .01			ΔR^2 = .02			ΔR^2 = .01			ΔR^2 = .01			ΔR^2 = .01		
Emotional[b] Knowledge	.01	.01	.14	.00	.00	.16	.08	.10	.14	.00	.01	.16	.00	.00	.13
Step 4	ΔR^2 = .03			ΔR^2 = .01			ΔR^2 = .02			ΔR^2 = .01			ΔR^2 = .03		
Emotional Knowledge X Gender	.01	.01	1.34	.01	.01	.78	.17	.17	1.14	-.01	.01	-.81	.01	.01	1.39

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 16). Step 2 is only provided

once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

E. AIM #3: EXAMINE THE LONGITUDINAL CONTRIBUTION OF CHANGES IN EMOTIONAL INTELLIGENCE TO SOCIAL DISABILITY

1. Relationship Between Treatment and Changes in Emotional Intelligence and Social Disability

To begin examining the longitudinal contribution of changes in emotional intelligence to changes in social disability, and the mediating effects of improvement in emotional intelligence on treatment-related improvements in social disability, the relationship between treatment and changes in these domains was examined. These analyses were used to fulfill criteria 1 (that the independent variable [treatment] be related to the dependent variable [changes in social disability]) and 2 (that the independent variable [treatment] be related to the mediating variable [changes in emotional intelligence]) of Baron and Kenny's (1986) criteria for demonstrating statistical mediation, and were accomplished through the use of multiple regression models predicting residualized changes in emotional intelligence and social disability from treatment assignment (EST=0, CET=1). Residualized change scores were computed by taking the residual of year 1 follow-up scores predicted by baseline scores. Consistent with guidelines for the appropriateness of using residualized change scores, no appreciable differences were found between treatment groups for baseline to year 1 correlations when producing any of the residualized scores (Maxwell, Delaney, & Manheimer, 1985).

As can be seen in Table 19, treatment was significantly related to improvements in general emotional intelligence, as well as emotional regulation and knowledge, such that individuals receiving CET displayed significantly more improvements in these domains than those receiving EST. In addition, treatment was also significantly related to changes in overall social disability and social leisure, with individuals receiving CET demonstrating significantly greater reductions in SAS-II total and social leisure disability scores. Such findings support the

previously documented efficacy of CET on emotional intelligence (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007) and social disability (Hogarty et al., 2004; Hogarty, Greenwald, & Eack, 2006), and support Baron and Kenny's (1986) first and second criteria for showing statistical mediation, by demonstrating a significant relationship between treatment and changes in emotional intelligence and social disability. Consequently, the hypothesis that CET treatment will result in improvements in these domains (Hypothesis 2a) was supported.

Table 19. *Effect of Treatment on Changes in Emotional Intelligence and Social Disability*

	EST (*N* = 24)		CET (*N* = 23)				
	Baseline	Year 1	Baseline	Year 1	Relationship Between Changes and Treatment[c]		
Variable	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*F*	*p*[d]	*R*
MSCEIT[a]							
General Emotional Intelligence	85.41 (14.30)	84.83 (15.52)	85.53 (16.89)	92.38 (14.05)	8.07	.007	.39
Emotional Regulation	91.21 (9.85)	89.94 (10.48)	92.70 (11.32)	95.89 (8.97)	6.93	.012	.37
Emotional Knowledge	92.85 (12.43)	91.75 (12.05)	89.92 (11.04)	95.97 (10.39)	6.56	.014	.36
SAS-II[b]							
Total	1.42 (.41)	1.33 (.46)	1.46 (.59)	1.11 (.50)	4.47	.040	-.30
Interpersonal Anguish	.96 (.34)	.85 (.33)	.97 (.29)	.81 (.29)	.37	.544	-.09
Sexual Relations	15.37 (6.52)	15.17 (5.67)	12.83 (7.21)	10.99 (7.48)	1.59	.214	-.18
Household Relations	.68 (.38)	.64 (.34)	.59 (.34)	.47 (.34)	1.32	.259	-.19
Social Leisure	.79 (.21)	.74 (.32)	.89 (.31)	.65 (.40)	4.52	.039	-.30

Note. Analyses were conducted on the combined, follow-up treatment sample (*N* = 47). CET = Cognitive Enhancement Therapy; EST = Enriched Supportive Therapy; MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

[c]Results from regression models predicting residualized change scores from treatment assignment

[d]Two-tailed test.

2. Relationship Between Changes in Emotional Intelligence and Social Disability

After finding support for the efficacy of CET at improving emotional intelligence and reducing social disability, the relationship between improvements in these domains was examined. This was accomplished by first calculating zero-order and partial correlation coefficients, adjusting for demographics and clinical characteristics, between residualized change

scores for MSCEIT and SAS-II scales. While these analyses do not strictly satisfy the criteria outlined by Baron and Kenny (1986) for demonstrating statistical mediation, they are central to the primary aims of this research, which seeks to understand the longitudinal relationship between changes in emotional intelligence and social disability, in addition to the mediating effects of changes in emotional intelligence on the relationship between treatment and changes in social disability. Further, Baron and Kenny's third criterion for demonstrating statistical mediation is dependent upon a relationship between changes in emotional intelligence and social disability, when adjusting for the effects of treatment on changes in social disability, which is unlikely to exist if no zero-order relationships are present in these analyses. Consequently, the results from these analyses will both provide a test of the relationship between changes in emotional intelligence and social disability, and serve as an intermediate step toward satisfying Baron and Kenny's third criterion for statistical mediation.

Table 20 shows estimated zero-order and partial correlation coefficients of the relationship between changes in MSCEIT and SAS-II scores. As can be seen in this table, improvements in emotional regulation were significantly related to reductions in overall social disability, as well as reductions in social disability regarding household relations. Furthermore, these effects remained statistically significant after adjusting for demographic and clinical characteristics shown to be related to emotional intelligence and social disability. In addition, a non-significant trend was observed indicating a positive relationship between improvements in emotional knowledge and increases in interpersonal anguish, perhaps reflecting a relationship between heightened emotional awareness and recognition of one's social problems. No significant or trend-level patterns of relationships emerged between zero-order changes in general emotional intelligence and any domain of social disability. However, improvements in general emotional intelligence were negatively related to increased social disability in household

relations at a trend level, after demographic and clinical confounds were taken into account. No significant interactions between changes in MSCEIT performance and gender were found with regard to relations with changes in social disability. Taken together, these findings support the hypothesis that improvements in emotional intelligence are related to reductions in social disability (Hypothesis 2b) in some domains, with the strongest evidence pointing to a relationship between improved emotional regulation abilities and reduced overall social disability and problematic household relations.

Table 20. *Associations Between Changes in Emotional Intelligence and Social Disability*

Variable	ΔSAS-II Total[b]	ΔInterpersonal Anguish	ΔSexual Relations	ΔHousehold Relations	ΔSocial Leisure
ΔGeneral Emotional Intelligence[a]	-.13 (-.11)	-.04 (.00)	.02 (-.04)	-.19 (-.27†)	-.09 (-.08)
ΔEmotional Regulation	-.31* (-.31*)	-.18 (-.15)	.06 (-.04)	-.34* (-.45*)	-.20 (-.18)
ΔEmotional Knowledge	.11 (.11)	.22† (.24†)	.05 (-.00)	.08 (.03)	-.02 (-.00)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample (N = 47). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†$p < .15$, * $p < .05$, two-tailed.

It is interesting that improvements in emotional regulation were related to improved household relations, as this may reflect either a true relationship among these constructs or the existence of stronger and more pervasive set of relationships between changes in emotional intelligence and social disability among the subset of individuals (n = 36) who received scores on this domain because they were living with a family member or significant other. Table 21 supports the former explanation more than the latter, as the pattern of relationships between changes in emotional intelligence and social disability among the subsample of individuals living with a significant other was quite similar to the complete sample (see Table 20). However, changes in general emotional intelligence did exhibit a trend-level negative relationship with changes in SAS-II total scores that was not evident in the complete sample, and improvements in emotion regulation abilities were now more strongly associated with reductions in SAS-II total scores. Consequently, it appears that for those individuals living with significant others, improvements in emotional intelligence may have a somewhat stronger circumscribed effect on reductions in overall social disability.

Table 21. *Associations Between Changes in Emotional Intelligence and Social Disability Among Individuals Living With Significant Others*

Variable	Δ SAS-II Total[b]	Δ Interpersonal Anguish	Δ Sexual Relations	Δ Household Relations	Δ Social Leisure
Δ General Emotional Intelligence[a]	-.28† (-.28†)	-.09 (-.05)	.04 (-.03)	-.19 (-.27†)	-.19 (-.18)
Δ Emotional Regulation	-.42* (-.41*)	-.23 (-.18)	.06 (-.00)	-.34* (-.46*)	-.25 (-.24)
Δ Emotional Knowledge	-.02 (-.07)	.24 (.22)	.06 (-.01)	.08 (.03)	-.15 (-.13)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample of individuals who were living with a significant other (N = 35, a single participant was excluded due to missing data on the MSCEIT at follow-up). MSCEIT = Mayer-Salovey-Caruso

Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†$p < .15$, *$p < .05$, two-tailed.

Having demonstrated significant relations between changes in emotional intelligence and social disability in several domains, the presence of these relationships after adjusting for changes in neurocognitive function and psychopathology was examined through the use of hierarchical regression models. As can be seen in Table 22, while neurocognitive change was not related to changes in any domain of social disability, decreases in psychopathology were significantly related to decreases in all measures of social disability. Despite these sizable relations between changes in psychopathology and social disability, the relationships among improvements in emotional regulation, and reductions in total social disability and problematic household relations continued to persist after adjusting for changes in neurocognition and psychopathology. Conversely, the trend-level relationship identified between changes in emotional knowledge and interpersonal anguish was not longer present after accounting for neurocognitive and psychopathology change. However, the previous trend-level relationship between improvements in general emotional intelligence and household relations was statistically significant, after adjusting for neurocognitive function and psychopathology. No significant gender interactions were found for estimates of the relationship between changes in emotional intelligence and social disability in any domain. These findings point to the importance of longitudinal changes in emotional intelligence, particularly emotion management and cognitive facilitation abilities, to overall social disability and household relations, independent of neurocognitive and symptom change. Such findings continue to lend support to the hypothesis that improvements in emotional intelligence are uniquely related to reductions in

social disability (Hypothesis 2b).

Table 22. *Associations Between Changes in Emotional Intelligence and Social Disability After Adjusting for Changes in Neurocognitive Function and Psychopathology*

	Δ SAS-II Total[c]			Δ Interpersonal Anguish			Δ Sexual Relations			Δ Household Relations			Δ Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2	ΔR^2 = .30*			ΔR^2 = .24*			ΔR^2 = .11			ΔR^2 = .16			ΔR^2 = .26*		
ΔNeurocognitive Composite	-.04	.08	-.07	.04	.05	.13	-1.05	1.28	-.13	.02	.06	.06	-.09	.06	-.23
ΔBPRS Total	1.08	.25	.59*	.58	.16	.51*	9.62	4.29	.33*	.61	.24	.44*	.71	.20	.50*
General Emotional Intelligence															
Step 3	ΔR^2 = .03			ΔR^2 = .01			ΔR^2 = .00			ΔR^2 = .11*			ΔR^2 = .00		
ΔGeneral Emotional Intelligence[b]	-.01	.01	-.18	-.00	.00	-.10	-.01	.09	-.02	-.01	.01	-.38*	-.00	.00	-.08
Step 4	ΔR^2 = .00			ΔR^2 = .02			ΔR^2 = .01			ΔR^2 = .01			ΔR^2 = .01		
ΔGeneral Emotional Intelligence X Gender	-.00	.02	-.01	.01	.01	.16	-.19	.25	-.12	.01	.01	.11	-.01	.01	-.10
Emotional Regulation															
Step 3	ΔR^2 = .08*			ΔR^2 = .03			ΔR^2 = .00			ΔR^2 = .18*			ΔR^2 = .01		
ΔEmotional[b] Regulation	-.02	.01	-.32*	-.01	.01	-.18	.01	.14	.02	-.02	.01	-.48*	-.01	.01	-.13
Step 4	ΔR^2 = .00			ΔR^2 = .02			ΔR^2 = .00			ΔR^2 = .02			ΔR^2 = .00		
ΔEmotional Regulation X Gender	.00	.03	.02	.02	.02	.14	-.20	.43	-.07	.02	.02	.14	-.01	.02	-.06
Emotional Knowledge															
Step 3	ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .00			ΔR^2 = .02			ΔR^2 = .01		
ΔEmotional[b] Knowledge	-.00	.01	-.04	.00	.00	.08	-.03	.11	-.05	-.01	.01	-.15	-.00	.01	-.09
Step 4	ΔR^2 = .01			ΔR^2 = .02			ΔR^2 = .02			ΔR^2 = .01			ΔR^2 = .03		
ΔEmotional Knowledge X Gender	-.01	.02	-.11	.01	.01	.17	-.22	.25	-.15	.01	.02	.10	-.02	.01	-.20

Note. Analyses were conducted on the combined, follow-up treatment sample (N = 47). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented

to reduce visual clutter and avoid redundancy (see Table 20). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

3. Mediating Effect of Improved Emotional Intelligence on The Relationship Between Treatment and Social Disability

The previous series of analyses have provided support for the effects of CET on emotional intelligence and social disability (Baron & Kenny's [1986] first and second criteria for mediation), and pointed to the existence of a relationship between longitudinal changes in emotional regulation abilities and select domains of social disability. While such findings provide support for the malleability of emotional intelligence and the importance of changes in this domain to social disability, the question of whether experimentally manipulated changes in emotional intelligence have any effect on social disability remains. A series of mediation models was examined to investigate the degree to which improvements in emotional intelligence mediated the effects of CET on social disability to begin to answer this question.

Within the context of this research, Baron and Kenny's (1986) criteria for detecting statistical mediation rest upon the presence of (1) a relationship between treatment and changes in social disability, (2) a relationship between treatment and changes in emotional intelligence, and (3) a relationship between changes in emotional intelligence and social disability, when adjusting for the effects of treatment on changes in social disability. To these formal criteria, an intermediate step of first identifying zero-order relationships between changes in emotional intelligence and social disability can be seen as building evidence for Baron and Kenny's third criterion, as this criterion is unlikely to be satisfied if zero-order relationships between these constructs do not exist. Based on the previous series of analyses, of the 15 potential direct mediation models, 6 met Baron and Kenny's first and second criteria for showing statistical mediation (see Table 23). In addition, four models provided intermediate evidence for criterion 3, by demonstrating a significant or trend-level zero-order relationship between changes in emotional intelligence and social disability. However, only a single model (Model 6) satisfied both Baron and Kenny's first and second criteria, *and* demonstrated a zero-order relationship

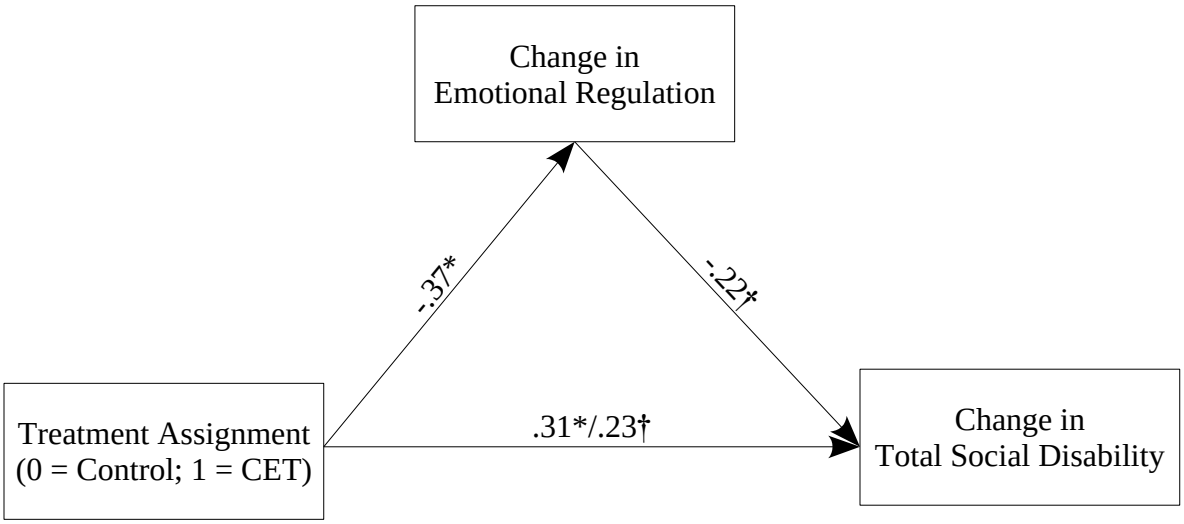
between changes in emotional intelligence and social disability. As such, this was the only model considered as a potentially successful mediation model, and therefore was the sole mediation model out of 15 possible models tested in the following analyses. This model consisted of the mediating effect of treatment assignment on changes in SAS-II total scores through changes in emotion regulation abilities. Corrections to the type I error rate were not performed for subsequent analyses, given the exploratory nature of this work. Although, it must be acknowledged at the outset that the possibility of detecting a false-positive finding is increased given the minority of mediational models (1 of 15) that met criteria for subsequent testing.

Table 23. *Summary of Possible Emotional Intelligence Mediational Models of the Relationship Between Treatment and Changes in Social Disability*

Mediation Model	Criterion 1: Relationship between treatment and Δsocial disability?	Criterion 2: Relationship between treatment and Δemotional intelligence?	Intermediate Criterion 3: Relationship between Δsocial disability and Δemotional intelligence?
1. Treatment → ΔGeneral Emotional Intelligence → ΔSAS-II Total	X	X	
2. Treatment → ΔGeneral Emotional Intelligence → ΔInterpersonal Anguish		X	
3. Treatment → ΔGeneral Emotional Intelligence → ΔSexual Relations		X	
4. Treatment → ΔGeneral Emotional Intelligence → ΔHousehold Relations		X	X
5. Treatment → ΔGeneral Emotional Intelligence → ΔSocial Leisure	X	X	
6. Treatment → ΔEmotional Regulation → ΔSAS-II Total	X	X	X
7. Treatment → ΔEmotional Regulation → ΔInterpersonal Anguish		X	
8. Treatment → ΔEmotional Regulation → ΔSexual Relations		X	
9. Treatment → ΔEmotional Regulation → ΔHousehold Relations		X	X
10. Treatment → ΔEmotional Regulation → ΔSocial Leisure	X	X	
11. Treatment → ΔEmotional Knowledge → ΔSAS-II Total	X	X	
12. Treatment → ΔEmotional Knowledge → ΔInterpersonal Anguish		X	X
13. Treatment → ΔEmotional Knowledge → ΔSexual Relations		X	
14. Treatment → ΔEmotional Knowledge → ΔHousehold Relations		X	
15. Treatment → ΔEmotional Knowledge → ΔSocial Leisure	X	X	

As can be seen in Figure 4, there was evidence of a reduction in the direct effect of treatment on changes in SAS-II total scores when accounting for changes in emotional regulation abilities. In particular, the effects of treatment on changes in overall social disability was reduced to only marginally significant (from β = .31 to .23), when accounting for changes in emotional regulation. However, the previously significant relationship between changes in emotional regulation abilities and changes in overall social disability, was also now only marginally significant. MacKinnon, Fritz, Williams, and Lockwood's (2007) asymmetric test of the indirect effect of treatment assignment on changes in overall social disability through changes in emotional regulation showed a non-significant trend ($z' = 1.30$, $p = .119$), possibly suggesting the potential mediating role of changes in emotion regulation. Given the possibility that changes in social disability mediate CET effects on emotion regulation (reverse mediation), a test of the indirect effect of treatment assignment on changes in emotion regulation through changes in overall social disability was also conducted. Results from this test also showed a non-significant trend supporting mediation ($z' = -1.23$, $p = .122$), suggesting that any mediating relationship between CET, emotional intelligence, and social disability may be bidirectional in nature. However, none of these results achieved statistical significance a conventional error rate levels, and thus they must be interpreted with caution.

Figure 4. Indirect Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation

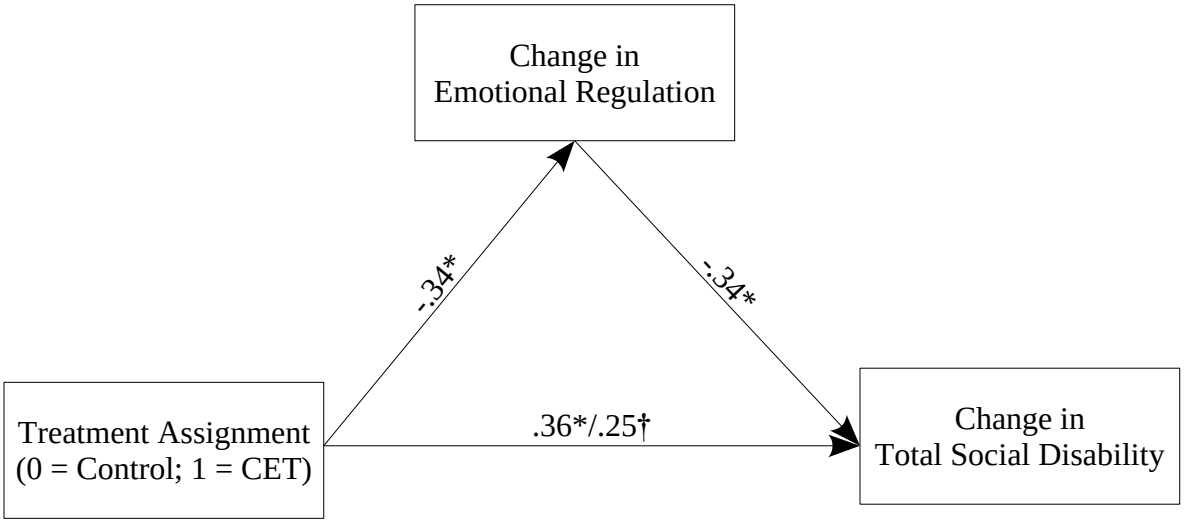


Note. Path parameters are standardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator.
CET = Cognitive Enhancement Therapy
†$p < .15$, *$p < .05$, two-tailed.

In addition to examining the mediating effect of changes in emotional regulation on the relationship between treatment assignment and changes in social disability in the complete follow-up sample of participants, this mediating effect was also investigated among those participants living with significant others, as the relationship between changes in emotional regulation abilities and social disability was particularly strong for this group of participants (see Table 21). As can be seen in Figure 5, the direct effect of treatment on changes in overall social disability was again reduced to marginally significant (from $\beta = .36$ to .25) when taking into account changes in emotional regulation abilities among this subsample of participants. In addition, the relationship between changes in emotional regulation abilities and social disability continued to remain statistically significant when accounting for the direct effect of treatment assignment on social disability. A test of the significance of the indirect effect of treatment

assignment on changes in social disability through changes in emotional regulation was significant ($z' = 1.47$, $p = .053$). However, a reverse test of the significance of the indirect effect of treatment assignment on changes in emotional regulation through changes in overall social disability was also significant ($z' = -1.52$, $p < .05$). Taken together, these findings provide partial and tentative support for the hypothesis that changes in emotional intelligence serve as potential mechanisms by which CET produces improvements in social disability (Hypothesis 2c), but also support reverse mediation and the bidirectionality of this effect. Unfortunately, given that the research design employed in this study consists of only a single post-test, the true degree of bidirectionality of this relationship cannot be tested and remains unclear. Further, this bidirectional mechanism may be the strongest among individuals living with family and/or significant others, as it was only among this subgroup did tests of statistical significance for mediation effects meet conventional error rate thresholds.

Figure 5. Indirect Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation Among Participants Living with Significant Others



Note. Path parameters are standardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator.
CET = Cognitive Enhancement Therapy
†$p < .15$, *$p < .05$, two-tailed.

Having found possible evidence for a bidirectional mediating effect of changes in emotional regulation abilities on the relationship between treatment assignment and changes in overall social disability, the degree to which this effect could be accounted for by demographic, neurocognitive, or psychopathological confounders was investigated. Inspection of mediational models accounting for gender, illness duration, education, diagnosis, as well as changes in neurocognitive function and psychopathology, yielded results largely similar to the previous series of mediation models. Results from these analyses indicated that treatment assignment continued to demonstrate marginally significant trends toward an indirect effect on changes in social disability through changes in emotion regulation abilities among the total follow-up sample ($z' = 1.47$, $p = .057$) and those living with family and/or significant others ($z' = 1.28$, $p =$

.096), after adjusting for the effects of demographics (gender, illness duration, education, and diagnosis) and changes in neurocognitive function and psychopathology. Further, reverse mediation models with changes in overall social adjustment mediating the effect of treatment on changes in emotional intelligence also showed non-significant and weaker trends among the total follow-up sample ($z' = -1.07$, $p = .179$) and those living with significant others ($z' = -1.13$, $p = .151$). Such findings continue to provide partial and tentative support for the hypothesis that changes in emotion regulation abilities are significant mediators of CET effects on overall social adjustment, independent of demographics and neurocognitive and psychopathological change (Hypothesis 2c), and suggest this effect may be bidirectional.

V. DISCUSSION

Social disability is a hallmark characteristic of schizophrenia that places major limitations on the ability of individuals who suffer from this disorder to recover and achieve a better quality of life. Despite the introduction of newer antipsychotic medications, individuals with schizophrenia often, though not always, continue to be severely socially disabled (Swartz et al., 2007). Research has increasingly pointed to the importance of cognitive deficits, particularly those associated with the processing and interpretation of socio-emotional information (Newman, 2001), as potential overlooked contributors to social disability in schizophrenia and novel areas for therapeutic intervention (Hogarty & Flesher, 1999a). Recently, exciting early research on the presence of deficits in emotional intelligence, or the ability to accurately perceive, understand, and regulate emotions (Mayer & Salovey, 1997), has documented pervasive deficits in this domain among individuals with schizophrenia (e.g., Cedro, Kokoszka, Popiel, & Narkiewicz-Jodko, 2001; Edwards, Jackson, & Pattison, 2002; Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001) and linked such deficits to social disability in several cross-sectional studies (e.g., Hooker & Park, 2002; Mueser et al., 1996). However, the study of emotional intelligence in schizophrenia and its contribution to social disability has been dramatically limited by measures circumscribed to the domain of emotion perception and a reliance on cross-sectional samples of long-term inpatients living in psychiatric hospitals. Consequently, the longitudinal role of the broader domains of emotional intelligence in predicting social disability among individuals with schizophrenia living in the community has largely gone unexamined.

This dissertation sought to begin to elucidate the role of emotional intelligence deficits in conspiring against social functioning in schizophrenia by examining the associations between a

novel performance-based measure of emotional intelligence (the MSCEIT) and a field standard measure of social disability (the SAS-II) among early course outpatients with the disorder. Secondary data were gathered from an ongoing randomized clinical trial of Cognitive Enhancement Therapy (CET; Hogarty & Greenwald, 2006) for individuals living in the community with early course of schizophrenia to (1) confirm the psychometric properties of the MSCEIT when applied to this population; (2) examine the unique cross-sectional relationship between emotional intelligence and social disability, above and beyond neurocognitive function and psychopathology; and (3) examine the unique longitudinal relationship between changes in emotional intelligence and changes in social disability, above and beyond changes in neurocognitive function and psychopathology. This chapter will provide a summary of the results of this research designed to address these aims, as well as a discussion of study limitations and implications for future research and social work practice.

A. SUMMARY OF FINDINGS

This investigation provided two important advances to the field of schizophrenia research. First, the measurement properties of a promising measure of emotional intelligence, the MSCEIT, have now been estimated in a sample of individuals with schizophrenia. Of particular importance, psychometric results uncovered compelling reasons to question the prevailing 4-factor structure of the instrument in this population, and perhaps healthy samples as well, which is considered to be a significant contribution of this research. Evidence was found for a general emotional intelligence factor and a novel 2-factor solution, but no factor-analytic findings could support the commonly found 4-factor structure for the instrument. As the reader can see, these results were compelling enough to inform the subsequent analytic approach used for examining the relations between emotional intelligence and social disability, resulting in a

restriction of the primary analysis of these relations to the 1- and 2-factor structures that provided the best fit to the sample under study. Potential reasons for an alternative factor structure for the MSCEIT and emotional intelligence in schizophrenia and their implications are discussed in detail below. In addition, psychometric analyses provided support for the reliability of the MSCEIT and its discriminant validity from demographic, neurocognitive, and psychopathological characteristics in this sample of individuals with schizophrenia.

The second major contribution of this investigation comes from the elucidation of the relationship between emotional intelligence and social disability in schizophrenia, which was admittedly weaker and less pervasive than expected. Evidence was found only supporting longitudinal relationships between improvements in emotional regulation abilities and improvements in overall social disability and household relations. Marginal and tentative support was also observed indicating that relations between improvements in emotion regulation and social disability might severe as a potential bidirectional mechanism by which CET may produce its effects on overall social disability. While these findings provide some exciting, albeit tentative support for the possibility of targeting emotional regulation abilities as a method of reducing social disability in schizophrenia, they also highlight the undeniable fact that evidence from this investigation could not support emotional intelligence as a strong and pervasive predictor of social disability. Cross-sectional analyses with social disability yielded few significant relations, and the longitudinal relations that did exist were small and sparse. Consequently, it seems likely that although emotional intelligence (particularly emotion regulation abilities) may play some role in social disability in schizophrenia, investigations of broader social-cognitive constructs relevant to functioning will also be needed.

The broader implications of the two major contributions of this research will be discussed below in detail within the study context in which they were conducted, along with a discussion

of a number of important study limitations, which require replication of these results before firm conclusions can be made regarding the factor structure of the MSCEIT and its relations to social disability. First, however, a detailed discussion of the findings of this investigation are provided.

1. Psychometric Properties of the MSCEIT

One of the major aims of this research, beyond investigating the relationship between emotional intelligence deficits and social disability in schizophrenia, was to first validate the psychometric properties of the MSCEIT among this population. As mentioned previously, research in this area has been substantially limited to circumscribed measures of emotion perception, as well-validated measures of the broader domains of emotional intelligence have thus far been lacking. The psychometric results of this research pointed to a number of strengths and limitations with regard to employing the MSCEIT to assess emotional intelligence in schizophrenia. Most notably, a series of exploratory factor analyses provided compelling reasons to challenge the validity of Mayer and Salovey's (1990) proposed 4-branch model of the MSCEIT when applied to persons with schizophrenia. While sample size limitations may have limited the results, clear evidence was found supporting an alternative factor structure than the common 4-factor branch (i.e., emotion perception, facilitation, understanding, and management) model demonstrated among healthy individuals.

Factor-analytic results pointed to the possibility of a single-factor, general emotional intelligence solution, which has been reported previously in the literature (Mayer, Salovey, Caruso, & Sitarenios, 2003); and at most a moderately correlated 2-factor solution that has never before been evidenced in any factor-analytic study of the MSCEIT with healthy individuals (Gignac, 2005; Mayer, Salovey, Caruso, & Sitarenios, 2003; Palmer, Gignac, Manocha, & Stough, 2005). No evidence was found from screeplots of eigenvalues for any additional factors beyond a 2-factor solution, and a forced 4-factor solution did not converge. The resulting 2-

factor solution found in this research consisted of a combination of the emotion perception and understanding branches and the emotion facilitation and management branches, to form what were tentatively interpreted as emotional knowledge and regulation factors, respectively. While this factor-analytic solution is at variance with those reported among healthy individuals, it does make some conceptual sense as the perception and understanding of emotion both rely largely upon emotional knowledge, whereas emotion facilitation and management can be thought of as "hot" social-cognitive processes that reflect either the up- or down-regulation of emotion.

That previous factor-analytic studies of the MSCEIT have yet to find the particular 2-factor solution elucidated in this research could point to a true shift in the construct of emotional intelligence among individuals with schizophrenia, compared to healthy persons. Another likely alternative, is that this 2-factor solution has simply gone overlooked by investigators committed to testing theoretical solutions with confirmatory approaches. It is important to note that not only is this the first factor-analytic study of the MSCEIT among individuals with schizophrenia, it is also the first study to use exploratory factor analysis to elucidate the factor structure of the instrument, as previous studies have exclusively employed confirmatory factor analysis to test *a priori* theoretical models. While such an approach certainly has its merits, it also has the unfortunate drawback of potentially overlooking conceptually distinct models not specified by frequently imperfect theoretical frameworks. Consequently, it is unclear whether the factor structure found for the MSCEIT in this research represents a true shift in the latent structure of emotional intelligence among individuals with schizophrenia, or is merely a potential alternative solution overlooked by a field wedded to more modern confirmatory techniques. Further, it also remains unclear whether a 2-factor solution provided a substantial improvement beyond a more parsimonious general emotional intelligence solution, as both factors were moderately correlated and such a solution only explained a modest, but statistically significant, 2% increase in variance

among MSCEIT tasks. What is clearer, is that a 4-factor solution cannot be assumed in persons with schizophrenia. Consequently, all subsequent analyses of the relationship between emotional intelligence and social disability were based upon the 1- and 2-factor solutions that provided the best fit to this sample.

Although psychometric results pointed to a potential alternative factor-analytic solution for the MSCEIT among persons with schizophrenia, analyses of the internal consistency and discriminant validity of the instrument provided strong evidence regarding the reliability and validity of the MSCEIT in schizophrenia samples. Estimates of internal consistency were found to be particularly strong among MSCEIT total and branch scores, even when adjusting for demographic heterogeneity; although estimates were lower and at times below acceptable minimum thresholds for individual MSCEIT tasks. These findings mirrored that of Mayer and colleagues (2003) with healthy individuals, and indicate that the total and branch scores of the MSCEIT can be reliably employed among individuals with schizophrenia, but leave questions about the reliable use of individual tasks. These findings are also consistent with recent results by Nuechterlein and colleagues (2008), who found that the emotion perception and management branches of the MSCEIT had adequate levels of retest reliability.

Discriminant validity estimates were also strong for the MSCEIT, as the instrument generally shared little variability with demographic, cognitive and clinical characteristics. MSCEIT total scores did tend to be higher among females and more educated participants, as reported with healthy individuals (Brackett, Rivers, Shiffman, Lerner, & Salovey, 2006). In addition, emotion perception and facilitation scores were greater among individuals with shorter illness durations and schizoaffective disorder, respectively. Not surprisingly, emotion understanding scores were also related to educational levels. Nonetheless, all of these relationships remained within the small to medium-sized range, indicating that demographic and

illness characteristics did not provide a substantial confound to MSCEIT performance.

Discriminant validity findings with regard to cognitive function and psychopathology, which might be expected to limit MSCEIT performance and correlate highly with MSCEIT assessments, were also strong. As expected, neurocognitive function did display consistent relations with MSCEIT total and branch scores, which remained after adjusting for demographic and illness characteristics. However, again these relations were small to moderate, with true score estimates of the relationship between MSCEIT and neurocognitive composite scores never moving beyond the medium-sized range. Associations with psychopathology were few, with only emotion perception and understanding branch scores relating significantly to psychopathology. Although statistically significant, these relationships were also small to moderate in size, and did not persist when adjusting for demographic and illness characteristics. In addition, true score estimates of the relationship between MSCEIT and BPRS total scores remained in the small to medium-sized range. Such findings largely support the discriminant validity of the MSCEIT when employed with individuals with schizophrenia, by indicating that MSCEIT assessments provide a measure of social-cognitive abilities beyond what can be gathered from standard neuropsychological and clinical assessments. This is critical, if the measure is to be widely employed in schizophrenia research, as suggested by the recommendations of the MATRICS committee (Green, Olivier, Crawley, Penn, & Silverstein, 2005).

2. Relationship Between Emotional Intelligence and Social Disability

The first aim of this investigation guided subsequent aims toward an appropriate scoring and factor-analytic solution for this sample of individuals with schizophrenia, and provided some confidence in the reliable and valid use of the MSCEIT in schizophrenia research. While such psychometric evidence is critical to the use of the MSCEIT among individuals with

schizophrenia, the primary focus of this study was admittedly on elucidating the relationship between emotional intelligence and social disability in this population. The critical findings from analyses of these relations in the second and third aims of this research pointed to a longitudinal relationship between changes in emotional intelligence and changes in social disability. Consistent with previous findings, CET was shown to have significant beneficial effects on changes in both emotional intelligence and social disability. Furthermore, evidence was found indicating that individuals who demonstrated more improvement in emotion regulation also tended to experience greater reductions in overall social disability and problems in household relationships. For example, the CET participant who experienced a 15 point (i.e., 1 *SD*) gain in emotion regulation ability throughout the course of the study, also experienced a 58% decrease in his/her overall social disability score, and an 84% decrease in his/her household relations disability score. Conversely, the participant not receiving CET who lost 11 points (i.e., .75 *SD*) in emotional regulation ability during the study, experienced a 76% and 29% increase in overall and household relations social disability scores, respectively. Relations between changes in emotion regulation and social disability were particularly prominent among individuals living with family members or significant others, and regardless of whether the whole sample or this subsample was examined, these relationships persisted after adjusting for demographic and illness characteristics, as well as neurocognitive functioning and psychopathology. Consequently, some support was found for Hypothesis 2b, indicating changes in emotional intelligence and social disability were indeed related within select domains, both before and after adjusting for neurocognitive functioning and psychopathology. These results laid the framework for the possibility of CET effects on social disability being mediated by improvements in emotion regulation.

The mediating effect of changes in emotional intelligence on the effects of CET on social

disability were nonetheless mixed, as partial and somewhat limited support was found for improved emotion regulation abilities mediating the effect of CET on overall social disability. Specifically, non-significant trends were observed indicating that improvements in emotional regulation abilities mediated CET effects on overall social disability, and these trends were the strongest and reached statistical significance among individuals living with family or significant others. That these effects were strongest among individuals living with family members and/or significant others may point to the possibility of improvements in emotion regulation having the largest impact when close interpersonal situations are prevalent, such as when living with a spouse or other loved ones.

Marginally significant trends ($p < .15$) were still observed supporting mediational effects of changes in emotion regulation on CET effects on social disability after adjusting for demographic and illness characteristics, as well as confounding changes in neurocognitive function and psychopathology. In fact, somewhat stronger support for mediation was found among the whole sample, after adjusting for these characteristics, indicating that the aspects of changes in emotion regulation that are unique from neurocognitive function and psychopathology may be the key components of the construct that produce changes in social disability. Given the trend-level associations observed from these analyses, firm conclusions cannot be made regarding CET mechanisms of social disability improvement, although these findings do provide tentative support for the mediating role of improvements in emotion regulation in partially explaining the effects of CET on overall social disability (Hypothesis 2c).

It should be noted, however, that the mediational model supported in this research represented but 1 of 15 potential mediational models, with the other 14 models not meeting Baron and Kenny's (1986) criteria for statistical mediation. Further, the longitudinal relations between emotion regulation and social disability were all small in size, and although changes in

emotion regulation were found to mediate CET effects on overall social disability, this mediational effect was only partial, as CET still demonstrated a small, but marginally significant direct effect on SAS-II total scores. In addition, some evidence was found for reverse mediation, as trends were also observed indicating that changes in overall social disability partially mediated CET effects on emotion regulation. Consequently, it seems clear that even if emotional intelligence is a mechanism by which social disability can be improved, this mechanism is likely to be bidirectional and this is clearly not the only, and perhaps not even the strongest, social-cognitive mechanism that exists for addressing social disability in schizophrenia.

While the longitudinal analyses employed in this investigation provided some support for a relationship between changes in emotional regulation and social disability over time, surprisingly, MSCEIT performance demonstrated little to no cross-sectional relationship with social disability at both baseline and follow-up. Further, these constructs remained largely unrelated regardless of analytic adjustments made for the confounding effects of demographic, clinical, and neurocognitive characteristics. The sole exception to this pattern of results were two significant relationships between household relations, and general emotional intelligence and emotion regulation abilities at year 1. However, these relationships were not present at baseline, and therefore should be interpreted with caution. Contrary to a large body of previous literature (Green, Kern, Braff, & Mintz, 2000), neurocognitive functioning also demonstrated little to no relation with social disability. Rather, the most consistent cross-sectional predictor of social disability was psychopathology, which was related to, as one might expect, interpersonal anguish scores and overall SAS-II total scores. Consequently, little support was found for Hypotheses 1a and 1b, specifying that emotional intelligence and social disability would be cross-sectionally related, both before (1a) and after (1b) adjusting for neurocognitive function and psychopathology.

Several factors could explain the disparate findings between cross-sectional and longitudinal relations of emotional intelligence and social disability. On the one hand, it is certainly possible that the two constructs may not be endogenously related on a cross-sectional level, even though changes in these constructs over time bear some relationship to one another. Cross-sectional associations are not necessary for either longitudinal or change associations, which ask a substantively different, and one might argue more informative question about how emotional intelligence and social disability are related. For example, research has repeatedly shown little relation between cross-sectional rates of vigorous physical activity and weight (e.g., Dowda, Ainsworth, Addy, Saunders, & Riner, 2001), yet programs designed to increase exercise are often associated with significant weight loss (e.g., Slentz et al., 2004). Such a finding exists because many factors influence current weight levels, of which exercise is only one, however relatively few factors can produce active changes in weight. Consequently, the strength the of association between exercise and weight differs between cross-sectional and change analyses because the universe of potential additional associative factors with weight differs for these two analytic questions.

The same could be said for social disability in schizophrenia, where extant levels of disability are determined by many genetic, biologic, and environmental factors (Thaker & Carpenter, 2001); however there may be relatively few mechanisms by which such disability can be improved. As such, it seems plausible that a true association between changes in emotional intelligence and social disability could exist, despite a lack of cross-sectional evidence. On the other hand, a number of important study limitations could also attenuate and/or preclude the detection of cross-sectional relations, which might reasonably explain the discrepancy between the cross-sectional and longitudinal findings of this research. As will be discussed in detail below, the modest sample size employed in this research and the possibility of restricted range in

social disability could both lead to conflicting results between cross-sectional and change analyses. Finally, the lack of cross-sectional relations could merely be indicative of a general small relationship between emotional intelligence and social disability, as even longitudinal relations, while significant, were not large. When coupled with the notable absence of cross-sectional relations, it seems increasingly likely that other social-cognitive factors could play an important role in social disability in schizophrenia, and subsequent efforts should be directed toward elucidating these factors.

In summary, the results of this research point to the internal consistency and discriminant validity of the MSCEIT for measuring emotional intelligence deficits in schizophrenia, but leave many questions regarding its factor structure and contribution to social disability among this population. Little to no evidence was found for a cross-sectional relationship between MSCEIT performance and social disability, but some support was found for a relationship between *changes* in select domains of MSCEIT performance and *changes* in social disability. More tentative mediational results suggest that these changes may serve as a bidirectional mechanism by which social disability can be improved in schizophrenia.

B. LIMITATIONS

Prior to any discussion of the implications of this research, it is important to note a number of limitations, which should both highlight the need for future research in this area and severe to temper substantive interpretations of this work and its implications for social work practice. Although specific hypotheses were developed based on previous evidence, this research, which is the first to examine the use of the MSCEIT among individuals with schizophrenia, was largely exploratory in nature. Hypotheses proposed a general relationship between emotional intelligence and social disability, but the degree to which the specific

domains of these constructs (of which there are many) were related remained largely unknown. Given the somewhat exploratory nature of this research and modest sample size employed, an analytic approach was adopted favoring power to detect significant relations among the constructs of interest. While this approach is appropriate for novel exploratory work, results that ensue must be interpreted with caution and cannot be taken as definitive, due to the increased likelihood of falsely rejecting the null hypothesis that comes with multiple inference testing, even when conventional alpha levels are used (Shaffer, 1995). To investigate the primary hypotheses of this research, many inferential tests of statistical significance were used, which could potentially inflate the experiment-wise error rate. While this makes little difference for the interpretation of the largely null cross-sectional relations between emotional intelligence and social disability (Hypotheses 1a and 1b), longitudinal relations between these constructs where some statistically significant results were found could be affected.

Primary analyses of the relationship between changes in emotional intelligence and changes in social disability relied of 15 tests of statistical significance, and found 2 significant relations between changes in emotional regulation and changes in overall social disability and household relations. Although these results were significant at the conventional .05 alpha level, none survived a Bonferroni corrected .003 (.05/15) alpha level adjusting for multiple inference testing. Subsequent mediator analyses and analyses of the relationship between changes in emotional intelligence and social disability, above and beyond changes in neurocognitive function and psychopathology, relied upon these initial analyses and thus could be affected by an inflated experiment-wise error rate as well. Even if these subsequent analyses were themselves considered isolated from initial correlational analyses, the multiple inference testing problem continues to potentially affect results. For the analyses of the incremental relationship between changes in emotional intelligence and social disability, above and beyond changes in

neurocognitive function and psychopathology, 15 primary tests of statistical significance were also conducted, and again none of the findings that demonstrated statistical significance at the conventional alpha level survived Bonferroni corrections. The same was true for mediator analyses, where 8 primary tests of statistical significance were conducted, none of which survived Bonferroni corrections. Consequently, although this research demonstrated some statistically significant relations between changes in emotional intelligence and social disability, the probability that these effects represent true relations beyond chance is potentially reduced due to an inflated experiment-wise error rate. As such, the interpretation of these relationships must be made with caution until evidence is available from future studies.

In addition to problems of multiple inference testing, this research is also limited by its somewhat modest sample size, which could have precluded the detection of small, but significant relations between emotional intelligence and social disability. Adequate power was only available in this research to detect small to medium-sized relations between these constructs, and as such smaller effect sizes may have gone overlooked. This limitation is most apparent by the presence of a number of non-significant trends indicating a relationship between changes in emotional intelligence and changes in social disability, as well as the mediating effect of changes in emotional regulation on CET effects on social disability. In addition, cross-sectional relations between emotional intelligence and social disability, which were largely null, could also have been overlooked due to power limitations. Currently, because of the modest sample size employed in this research, it is difficult to draw firm conclusions from such results. On the one hand, trend-level effects may represent true relationships that are merely masked by sample size limitations. On the other hand, such effects cannot be considered to exist beyond chance, due to their sub-threshold significance level and aforementioned issues regarding the inflation of the experiment-wise error rate. Aside from precluding the detection of smaller, trend-level relations,

the modest sample size employed in this research could have also precluded the survival of significant relations after corrections for multiple inference testing. Consequently, while some significant relations were found between changes in emotional intelligence and social disability, smaller but more pervasive effects may also exist that were overlooked due to sample size limitations.

The modest sample size employed in this research could have also precluded the detection of additional factors in factor analyses of the MSCEIT. As mentioned previously, this research found support for at most 2 factors, which were at variance with the reigning 4-factor solution for the instrument. When a 4-factor solution was tested, results did not converge, which may reflect a sample size limitation. Many psychometricians have indicated that the appropriate sample size for a factor analysis is dependent upon a number of different characteristics of the data and model to be tested (Maccallum, Widaman, Zhang, & Hong, 1999). One important characteristic is the size of the model examined, where models with more parameters (e.g., factor loadings, variances, correlations) generally require larger sample sizes. The 4-factor solution for the MSCEIT reported in the literature has 11 more parameters that require estimation than the most complex, 2-factor solution found in this research. Consequently, the sample size employed in this research may have allowed for the stable estimation of model parameters in a 2-factor solution, but may not have been sufficient for the estimation of more complex models, such as the 4-factor solution reported by Mayer and colleagues (2003). However, it should be noted that eigenvalue decomposition of the correlation matrix among MSCEIT tasks, which is not sensitive to sample size, indicated at most two eigenvalues greater than 1, which supports the more parsimonious solution found in this research.

Another limitation with this research also stems from the nature of the sample employed, in that the selection of individuals in the early course of schizophrenia for a controlled treatment

trial may have restricted the range of emotional intelligence and social disability experienced by the sample. This could have had particularly detrimental effects on cross-sectional relations between emotional intelligence and social disability, and may explain the surprising lack of cross-sectional relations found between these constructs. While a sufficient range of scores was seen with regard to emotional intelligence (e.g., range = 54.09 to 121.70 at baseline for overall emotional intelligence), this sample was characterized by marked social disability, with few individuals reaching the upper bounds of functioning based on SAS-II scores. Such individuals are most appropriate for a treatment trial aimed at improving cognition and social disability, but the restricted range of social disability scores limits this investigation due to a potential artificial attenuation of relations (Pearson, 1903). Such a range restriction could not only explain the surprising lack of relations between emotional intelligence and social disability at baseline, but could also explain why neurocognitive function also demonstrated no relationship with social disability, which is contrary to a large body of evidence (Green, Kern, Braff, & Mintz, 2000). Sufficient variability within constructs is an essential requirement for detecting shared variance between constructs, and while variability did exist on social disability in this sample, it may be less than that observed in more naturalistic samples.

In fact, the pattern of cross-sectional relations observed in this research suggest the real possibility of a range restriction problem with regard to social disability. Recall that no significant relations were observed between emotional intelligence and social disability at baseline, while some significant relations were present at year 1 follow-up. Inspection of the range of emotional intelligence scores at these time points reveals largely similar ranges (e.g., 54.09 to 121.70 at baseline and 57.87 to 117.82 at year 1 for overall emotional intelligence), however somewhat larger ranges of overall social disability scores exist at year 1 (range = .15 to 2.35) compared to baseline (range = .46 to 2.50). This additional variability in social disability

at follow-up is likely a result of CET effects on social disability diverging the two treatment groups, and this added variability could explain why some significant cross-sectional relations exist at year 1, whereas none exist at baseline. Consequently, the null findings of this research with regard to cross-sectional relations between emotional intelligence and social disability may be substantially affected by restrictions on the range of social disability experienced by this sample, and therefore studies employing larger, more heterogeneous samples may find more evidence for greater relations between these constructs.

Finally, this research is limited to some degree by its modest 1 year follow-up of participants. While the longitudinal nature of this research is a significant strength, the longitudinal design employed is also limited for answering some critical questions regarding the relationship between changes in emotional intelligence and changes in social disability. This is perhaps most evident when considering results surrounding the mediational effects of changes in emotion regulation abilities on CET effects on social disability. Recall that tentative evidence was found supporting changes in emotion regulation abilities as potential mechanisms of CET effects on reduced social disability. However, evidence was also found for reverse mediation, with changes in social disability serving as a potential mediator of CET effects on emotional intelligence. As such, evidence exists for the bidirectional nature of the relationship between changes in emotional intelligence and changes in social disability. Unfortunately, what this research is unable to answer with a single post-test, is the relative magnitude of each direction of this relationship.

For example, it is posited in this research that even in the likely case that the relationship between emotional intelligence and social disability is bidirectional, it is the early accrual of improvements in emotional intelligence that serve as the primary mediator of CET effects on long-term social disability. The answer to such a question cannot be arrived at without at least 1

additional follow-up point, so that relations between *early* improvements in emotional intelligence and *later* improvements in social disability can be elucidated and compared with tests of the relationship between early improvements in social disability and later improvements in emotional intelligence. Ideally, even larger longitudinal designs could be employed to estimate the effects of early improvements in emotional intelligence on later social disability, and the subsequent reciprocal effects of these later reductions in social disability on any additional improvement in emotional intelligence. Such evidence is critical for untangling the true mechanisms of CET effects and understanding the precise nature of the bidirectional relationship between emotional intelligence and social disability in schizophrenia.

C. IMPLICATIONS

1. Implications for Research

The results of this investigation have a number of important implications for future research, despite the extant limitations of this study. In fact, study limitations can been seen as one of the major motivating factors for future investigations in this area and provide fertile ground for a number of new research directions. An overall theme of the findings of this research was that contrary to biopsychosocial models of social disability in schizophrenia and the hypotheses of this investigation, the emotional intelligence components of social cognition assessed by the MSCEIT were at most only modestly related to social disability. While emotion regulation abilities seemed to hold the strongest relations to social disability, this was limited primarily to overall social disability and disability with regard to household/family relations, and appeared only in longitudinal analyses. Further, emotional knowledge appeared to have no relation to social disability, either cross-sectionally or longitudinally. Although the limitations discussed above may account for the sparse relations between MSCEIT performance and social

outcome, it also seems clear from the results of this research that emotional intelligence is not the only social-cognitive factor critical to functional disability in schizophrenia. A more plausible model of social disability in schizophrenia would likely consist of other components of the broad domain of social cognition, such as perspective-taking ability (Harrow & Miller, 1980; Hogarty & Flesher, 1999a), foresightfulness (Eack & Keshavan, 2008), social cue recognition (Corrigan & Green, 1993), and theory of mind (Premack & Woodruff, 1978).

To date, schizophrenia researchers have shown that individuals with the illness possess some deficits in social-cognitive domains other than emotional intelligence (e.g., Brune, 2005b; Corrigan & Green, 1993), however their link to functioning and social disability has yet to be fully examined. While some cross-sectional studies have shown relations between other domains of social cognition and social disability (e.g., Roncone et al., 2002), longitudinal studies have yet to be conducted and carefully constructed measures of perspective-taking, foresight, theory of mind, and social cue recognition have yet to be developed and validated among persons with schizophrenia. Consequently, given the findings of this research, there is an urgent need to both look beyond emotional intelligence for the social-cognitive contributors to social disability in schizophrenia, and carefully construct and validate additional performance-based measures of social cognition that may shed light on the rate-limiting factors to functional improvement among this population. At the heart of the neurodevelopmental theory of Cognitive Enhancement Therapy, is the (untested) assumption that social-cognitive deficits in perspective-taking are primary factors in social disability in schizophrenia. Given the efficacy of the treatment for improving both social cognition and social disability (Hogarty et al., 2004; Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007), future research might profitably focus measurement development efforts on perspective-taking as a step in elucidating the additional social-cognitive contributors to social disability in schizophrenia.

In addition to this research pointing to the need to look beyond emotional intelligence in future studies, the findings of this investigation pertaining to emotional intelligence also indicate the need for additional studies of this social-cognitive domain among individuals with schizophrenia. Although not all hypotheses in this research were supported, some domains of emotional intelligence did show consistent and persisting relations with critical domains of social disability, indicating the potential importance of this construct to social functioning in schizophrenia. Sample size limitations and the somewhat exploratory nature of this investigation preclude drawing firm conclusions from this study, but these results may provide promising leads for future research.

To begin, it will be critical for subsequent studies to first replicate the factor-analytic results of this investigation, to examine the degree to which these findings might be unduly influenced by sample size limitations or actually point to an alternative factor structure for the MSCEIT among individuals with schizophrenia. This is a critical issue for the next logical step of schizophrenia research employing the MSCEIT that will seek to make valid cross-group comparisons between those with schizophrenia, healthy individuals, and individuals with other psychiatric disabilities. Such comparisons will only be meaningful if there is a consistent latent structure to emotional intelligence across these groups, and the preliminary findings of this research call this point into question. Consequently, the degree to which individuals with schizophrenia demonstrate deficits in MSCEIT performance compared to other groups will remain tentative until such time as these factor-analytic questions are resolved. Any investigation in this domain will need to also revisit factor-analytic results for the MSCEIT among healthy populations, since the instrument has yet to be subjected to exploratory factor analysis with such individuals, which may have masked the factor structure elucidated in this investigation. Ideally, future studies will recruit both healthy samples and individuals with

schizophrenia in sufficient numbers, so that simultaneous estimates of measurement equivalence and factorial invariance can be provided.

Second, future studies will also need to replicate cross-sectional and longitudinal findings regarding relationships between MSCEIT performance and social disability. While no significant relations were found cross-sectionally between these constructs in this research, it is possible that small relations did exist that were beyond the statistical power of this study to detect. It is quite surprising that cross-sectional relations did not exist, as previous studies have been replete with cross-sectional associations between emotional intelligence domains (particularly emotion perception) and social functioning (see Couture, Penn, & Roberts, 2006 for review). As suggested above, this result may also stem from range restrictions on social cognition and social disability for individuals participating in this clinical trial of CET, which could have attenuated relationship estimates (Pearson, 1903). These two limitations make the findings of this investigation tentative, and clearly indicate a need for future studies employing larger and more heterogeneous samples to further investigate the association between emotional intelligence and social disability in schizophrenia.

Furthermore, given the somewhat exploratory nature of this work, even the more positive longitudinal and mediational findings of this investigation need to be interpreted with caution and beckon replication. As discussed above, no correction for inflated Type I error rates was used in this research, despite a large number of tests of statistical inference employed. This undoubtedly raises the possibility that some of these findings reflected false positive results stemming from inflated experiment-wise error rates. As such, this research should be seen as providing fertile ground for subsequent work, not a definitive answer to the longitudinal relations between emotional intelligence and social disability in schizophrenia or the mechanisms of CET effects. Based on the results of this investigation, studies focusing specifically on emotion

regulation abilities, overall social disability, and household/family relations are needed to replicate these findings and determine their veracity.

In addition, the presence of findings suggesting that some domains of emotional intelligence may be longitudinally predictive of social disability in schizophrenia also makes it increasingly important to examine the degree to which these domains are actually deficient in the disorder. Much research has been conducted showing deficits in emotion perception among individuals with schizophrenia (Edwards, Jackson, & Pattison, 2002), however the major contributors to social disability in this research were deficits in emotion regulation, which have not been extensively studied in this population. In fact, little is known about whether individuals with schizophrenia have deficits in either emotion facilitation, management, or both; although mean levels in this sample were certainly below those from the normative mean score of 100 (see Table 8). Nonetheless, in the absence of direct comparisons of MSCEIT performance between individuals with schizophrenia and healthy populations, the magnitude of this deficit, which this study suggests may be functionally relevant, is not clear. Studies of MSCEIT performance in individuals with schizophrenia and matched healthy controls are particularly important avenues for future research, given the findings of this investigation.

Finally, if the longitudinal relations between emotional regulation and social disability demonstrated in this research are with merit, and improvements in emotional regulation abilities indeed provide a mechanism for the amelioration of social disability, subsequent studies might profitably focus on providing clarification regarding the bidirectional nature of this relationship. As discussed above, while evidence was found pointing to improvements in emotional regulation abilities as a mechanism of CET effects on social disability, evidence for reverse mediation was also present. The adoption of a single follow-up period in this research precludes disentangling the bidirectional extent of this relationship, as it is impossible to tell whether improvements in

emotional intelligence preceded reductions in social disability. Long-term studies of CET with multiple follow-up periods will be needed to determine the degree to which early improvements in emotional intelligence are associated with later improvements in social disability, and vice versa, to come to a clear understanding of the nature of the mechanisms of CET effects in these domains. A 3-year, post-treatment follow-up is planned for the current trial of CET that is the focus of this research, which may provide an opportunity to more definitively answer these mediational questions.

In summary, the results of this investigation provide several promising directions for future research. These directions include broader measurement efforts to examine domains of social cognition relevant to schizophrenia beyond emotional intelligence, further examination of the factor structure and performance levels of the MSCEIT in healthy and psychiatric populations, and replication of longitudinal associations between emotional regulation abilities and social disability in studies employing larger and more heterogeneous samples. With such research, it is hoped that studies will begin to close in on the domains of social cognition most relevant to functional disability in schizophrenia that could ultimately provide promising targets for future treatment development efforts.

2. Implications for Social Work Practice

This research admittedly raises more questions than it answers. As such, at this time the implications of these findings for social work practice are limited compared to the implications for future research efforts, as this study represents a very early effort in the field of schizophrenia research to understand the contribution of emotional intelligence to social disability in schizophrenia. Nonetheless, some important, but tentative, implications of this research for social work practice are apparent if the results of this investigation are validated in future studies. Perhaps the strongest implication for social work practice evinced by this research concerns the

effects of CET on emotional intelligence and social disability. Although not a primary focus of this research, evidence from this investigation did provide substantial support for the efficacy of CET, a psychosocial intervention, for improving both emotional intelligence and social functioning among individuals in the early course of schizophrenia. Even independent of all the other results of this research, this finding alone has substantial implications for social work practice and schizophrenia treatment.

For quite some time, debates have ensued about the degree to which cognitive deficits in schizophrenia can be remediated (Green, 1993), and for many years the field remained agnostic, if not nihilistic about the possibility of real cognitive change among such disabled individuals. Such a view has done a great disservice to the individuals, families, and communities that suffer with this disorder by stymying focused intervention development designed to improve the lives of people with schizophrenia. Some dedicated scientists have nonetheless pioneered their own efforts in spite of wide skepticism and critique from the field, and Hogarty's CET is one of these efforts. The approach integrates a massive evidence-base on brain plasticity, cognitive rehabilitation, and schizophrenia neurodevelopment to provide a theoretically-grounded justification for optimism regarding the potential for cognitive enhancement in schizophrenia (Hogarty & Flesher, 1999a). This optimism has been well-justified in a large randomized-controlled trial of CET for individuals with long-term (15 years on average) schizophrenia. In this trial Hogarty and colleagues (2004) showed that not only can cognition be improved with CET among individuals with long-term schizophrenia, but that the program can also help individuals make meaningful gains in social functioning, such as in interpersonal relations, major role performance, and work potential. A subsequent 1-year follow-up study of treated patients indicated that these improvements in cognition and functioning were maintained even a year after the treatment had ended, and that early cognitive improvements in the program were

significant mediators of subsequent functional change (Hogarty, Greenwald, & Eack, 2006).

Still, despite this very convincing evidence of the benefits of CET, questions have remained about applicability of the approach to early course patients, as well as the replicability of the results in general. Serious and legitimate questions were raised in the previous study of CET about the validity of the unblinded measures of social cognition developed by the team exclusively for assessing the efficacy of the treatment, as well as the somewhat limited assessments of social disability and other functional outcomes. This research largely confirms the previous documented benefits of CET for individuals with long-term schizophrenia, and suggests that these benefits can be extended to individuals in the early course of the disorder. It is particularly compelling that significant improvements in social cognition were realized on a performance-based, independent measure that is not subject to the influences that plague unblinded clinician-rated instruments. Furthermore, the previously documented effects of CET on social disability were also realized with a highly structured measure of social disability that represents a standard for the field.

These two findings from this investigation provide substantial support for the use of CET with individuals with schizophrenia to improve both cognition and social disability, and make a significant advance to the field of schizophrenia treatment, which has for some time been stumped at providing significant cognitive and functional benefits to those in desperate need. Recent evidence indicates that currently available pharmacological approaches for the treatment of schizophrenia have had little impact on cognition or social disability (e.g., Sergi et al., 2007; Swartz et al., 2007). When coupled with findings from individuals who have had schizophrenia for many years, these provide some of the most definitive evidence to date about the benefits of cognitive rehabilitation for individuals in the early course of the disorder. Given the effectiveness of the treatment and that social workers are some of the primary providers of

psychosocial therapies for individuals with schizophrenia (Substance Abuse and Mental Health Services Administration, 2001), the results of this investigation suggest that social work practitioners should seriously consider CET as a key psychosocial adjunct to pharmacological treatments for individuals with schizophrenia.

Not only does this research have implications directly for CET, but more generally, these results provide important continued support for the psychosocial treatment enterprise. Pharmacotherapy has become the standard treatment in the field for individuals with schizophrenia, and while the use of appropriate prophylactic antipsychotic medication is certainly essential to schizophrenia treatment, it is clear that much is to be gained from offering additional services, supports, and therapies. Social workers have been pioneers in developing such services (e.g., Anderson, Reiss, & Hogarty, 1986; Hogarty, 2002; Stein & Test, 1980), and repeated evidence has shown that psychosocial treatments can improve lives by reducing symptomatology and the likelihood of relapse (Hogarty et al., 1991; Stein, Test, & Marx, 1975; Tarrier et al., 2004), improving employment outcomes (Bond, Drake, Mueser, & Becker, 1997), interpersonal functioning and adjustment (Hogarty et al., 1974b; Hogarty et al., 1997b), and now even cognition (Eack, Hogarty, Greenwald, Hogarty, & Keshavan, 2007; Hogarty et al., 2004). Yet, evidence from common community clinics indicate that few individuals with schizophrenia receive much more than pharmacotherapy, and almost none receive anything that could even be loosely construed as evidence-based psychosocial treatment (Lehman et al., 1998b). This research continues to add critical support for the efficacy of psychosocial treatment approaches that is need to influence policy decisions about what treatment modalities need to be prioritized in community clinics with dwindling resources. Consequently, the findings of this research have important applications for both social work practice and policy.

Finally, most directly related to the primary focus of this research is the implication that a

single component of emotional intelligence, emotion regulation, may be a promising bidirectional mechanism by which social disability can be improved. The limitations of this research notwithstanding, evidence provided by this investigation suggested that improvements in emotion regulation may serve as one potential bidirectional mechanism by which CET produces its beneficial effects on overall social disability. This provides critical support not only for CET, but also for targeting emotional regulation abilities in diverse treatment approaches. If emotion regulation is indeed a mechanism, even a small partial one, by which social disability in schizophrenia can be improved, this could provide social work practitioners and treatment developers with critical insights into how to best help people with schizophrenia recover from the functional impact of their disorder. To date mechanisms of social disability change in schizophrenia and many other severe mental disorders have remained largely elusive, and unresponsive to medication (Swartz et al., 2007). The elucidation of such mechanisms provides fertile ground for future treatment development that could have a substantial impact on social work practice with this population, and ultimately serve to improve the lives many individuals with the disorder. That emotion regulation appears to be one potential mechanism by which social disability can be improved suggests that treatments that focus explicitly on targeting problems in emotion regulation could be particularly effective for this population. This will be the real test of the merit of these results, and social work practitioners and researchers will need to collaborate to begin applying these findings and identify the utility of emotion regulatory treatment approaches for schizophrenia.

D. CONCLUSIONS

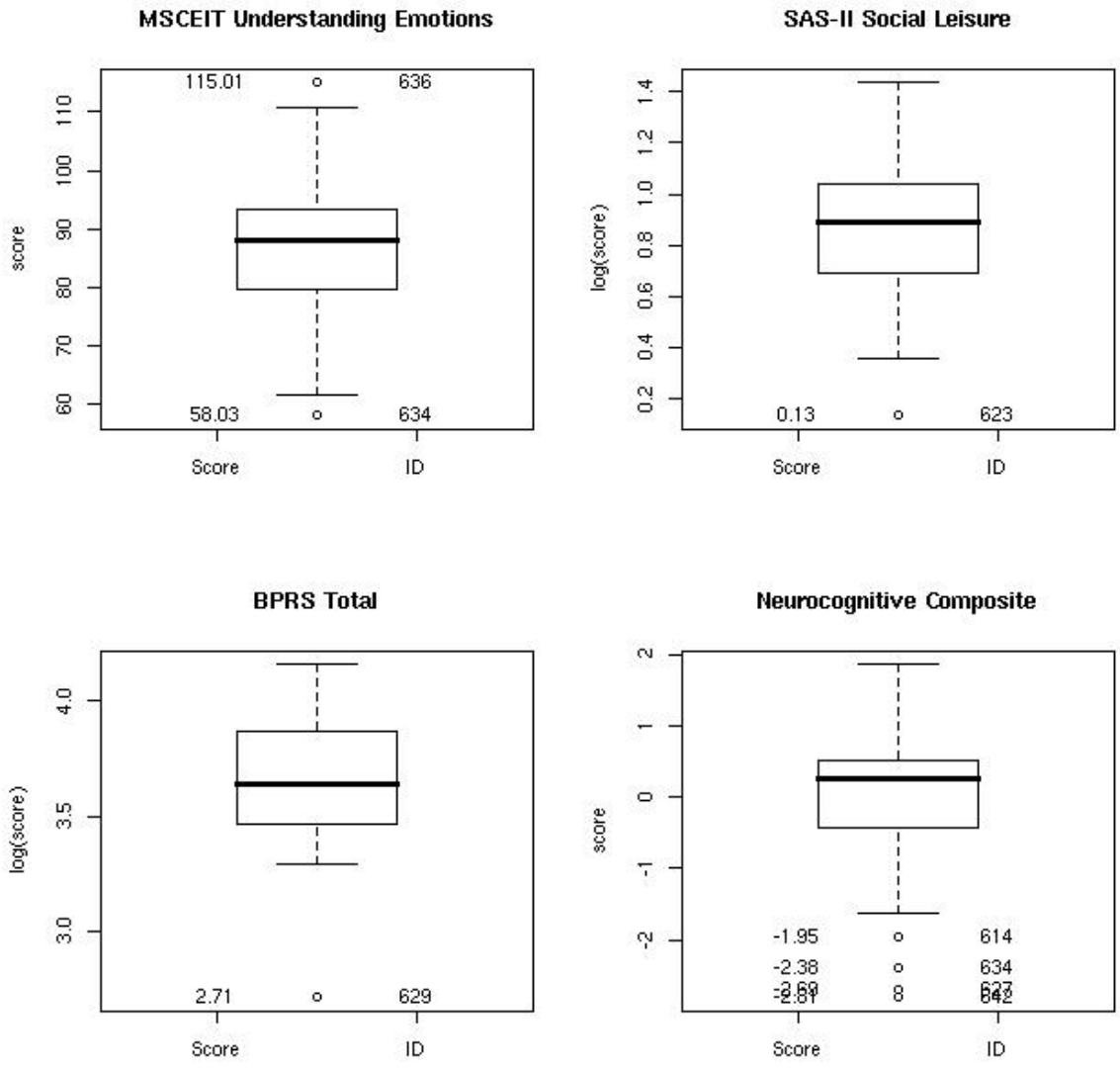
This research sought to conduct the first psychometric investigation of the MSCEIT among individuals with schizophrenia, and examine the unique, longitudinal contribution of the

emotional intelligence assessments it provides to social disability among population. Psychometric findings revealed serious challenges to the factor structure of the MSCEIT when applied to individuals with schizophrenia, but at the same time pointed to the reliability and discriminant validity of the instrument. Relations between MSCEIT performance and social disability were modest, with significant associations existing only between improvements in emotion regulation and reductions in overall social disability and household/family relationship problems. Tentative evidence pointed to the possibility of improvements in emotion regulation to serve as a mechanism by which CET achieves its effects on social disability, although reverse mediation could not be ruled out. Future research will need to replicate these findings with larger and more heterogeneous samples, and focus on the development of additional measures to study broader domains of social cognition, beyond emotional intelligence, that may bear relevance to social disability. The results of this investigation make two important contributions to the field by providing empirically-based information on the strengths and limitations of the MSCEIT and its factor structure as applied to schizophrenia, and elucidating the somewhat limited relations between emotional intelligence and social disability. By identifying these strengths and limitations of the MSCEIT and the relations between emotional intelligence and social disability, it is hoped that continued progress will be made by social work researchers and practitioners to identify additional contributors to social disability among this population, and ultimately develop and disseminate effective treatments to improve the lives of the many individuals who suffer from this disorder.

APPENDIX A - RESULTS WITH ALTERNATIVE TREATMENT OF OUTLIERS

A number of outliers were present at baseline across the primary study measures used in this research. As can be seen in Figure A1.1, 7 unique cases contributed to 8 different outliers across 4 primary study measures at baseline. Importantly, there were no outliers present on any measures at year 1 follow-up.

Figure A1.1. Study Outliers



Primary analyses in this research were conducted using a winsorization technique (Dixon & Tukey, 1968) to address case outliers, which uses a nearest neighbor algorithm to set the score on outlier to the next closest score within 1.5 times the interquartile range of the distribution of scores. While this approach conserves power by keeping the full sample of cases in the presence of outliers, given the appropriate treatment of outliers is still a subject of considerable debate (e.g., Ratcliff, 1993), it is usually recommended that multiple methods of addressing case outliers be used and compared. This appendix presents an examination of the key analyses of this research under two common alternative methods of addressing outliers: case deletion and case inclusion/no treatment. Results from these analyses under different outlier conditions are compared to primary analyses using winsorization. Findings are presented in identical tables to those that appear in the primary analyses, and a *Change Note* is provided in each table outlining the differences between the results in the given outlier condition and those in the primary analyses.

Overall, there are no changes to the interpretation of the primary study results under different outlier conditions, however some minor changes in significance levels and/or effect sizes are observed. The most common change in results was the reduction of statistically significant effects to trend-level results in the case deletion condition, which is expected due to the decrease in power associated with the deletion of cases from an already modest sample size. The details of the effects of alternative treatments of outliers are presented and discussed below for each specific aim of this research.

A. AIM #1: CONFIRM THE PSYCHOMETRIC PROPERTIES OF THE MSCEIT

1. Internal Consistency

Table A1.1 present internal consistency analyses for the MSCEIT under case deletion and

case inclusion outlier strategies. As can be seen in these tables, internal consistency levels for branch, total, and task scores were all similar and did not depart significantly from those observed when using winsorization.

Table A1.1. *Internal Consistency of The Mayer-Salovey-Caruso Emotional Intelligence Test*

	Winsorization			Case Deletion			Case Inclusion		
Scale	*N*	Alpha	Adjusted Alpha[a]	*N*	Alpha	Adjusted Alpha[a]	*N*	Alpha	Adjusted Alpha[a]
MSCEIT Total	57	.94	.93	50	.93	.92	57	.94	.93
Branch 1 - Emotion Perception	57	.91	.89	50	.90	.89	57	.91	.89
Task 1 - Faces	57	.82	.81	50	.82	.82	57	.82	.81
Task 2 - Pictures	57	.91	.89	50	.91	.90	57	.91	.89
Branch 2 - Emotion Facilitation	57	.82	.81	50	.81	.78	57	.82	.81
Task 3 - Facilitation	57	.77	.74	50	.76	.70	57	.77	.74
Task 4 - Sensations	57	.73	.72	50	.71	.69	57	.73	.72
Branch 3 - Emotion Understanding	57	.78	.76	50	.73	.71	57	.78	.76
Task 5 - Changes	57	.70	.68	50	.67	.65	57	.70	.68
Task 6 - Blends	57	.52	.51	50	.40	.39	57	.52	.51
Branch 4 - Emotion Management	57	.81	.80	50	.79	.78	57	.81	.80
Task 7 - Management	57	.67	.67	50	.65	.63	57	.67	.66
Task 8 - Relationships	57	.73	.72	50	.72	.71	57	.73	.72

Note. Analyses were conducted on the combined treatment sample at baseline. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test

Change Note. No significant changes.

[a]Alpha adjusted for gender, education, illness length and diagnosis.

2. Discriminant Validity

Table A1.2 present discriminant validity analyses of the MSCEIT through an examination of its associations with neurocognitive functioning and psychopathology. Relations between MSCEIT performance and neurocognitive function continued to remain modest in both outlier conditions, however emotion perception was no longer significantly related to neurocognitive function in the case deletion condition. In addition, in the case deletion condition, BPRS total scores were also no longer related to MSCEIT performance. In the case inclusion condition, BPRS total scores continued to be modestly related with MSCEIT performance, and MSCEIT total scores now showed a significant relations with BPRS total scores. Together, all 3 outlier analyses continued to indicate low levels of association between MSCEIT performance and neurocognitive function and psychopathology, supporting the discriminant validity of the instrument.

Table A1.2. *Associations Between Emotional Intelligence, Neurocognition, and Psychopathology*

Variable	Neurocognitive Composite[a] Correlation	Neurocognitive Composite[a] True Score Relationship Estimate[c]	BPRS Total[b] Correlation	BPRS Total[b] True Score Relationship Estimate[c]
	Winsorization (N = 57)			
MSCEIT Total[a]	.39* (.33*)	.45	-.25 (-.05)	-.30
Emotion Perception	.34* (.28*)	.41	-.32* (-.07)	-.38
Emotion Facilitation	.34* (.30*)	.43	-.19 (-.08)	-.24
Emotion Understanding	.37* (.31*)	.48	-.29* (-.14)	-.37
Emotion Management	.26 (.22)	.32	-.08 (-.04)	-.10
	Case Deletion (N = 50)			
MSCEIT Total[a]	.32* (.29*)	.37	-.17 (.03)	-.20
Emotion Perception	.26 (.24)	.31	-.23 (-.01)	-.28
Emotion Facilitation	.30* (.28*)	.38	-.14 (-.04)	-.17
Emotion Understanding	.32* (.27)	.43	-.21 (-.04)	-.27
Emotion Management	.19 (.16)	.25	-.03 (.06)	-.04
	Case Inclusion (N = 57)			
MSCEIT Total[a]	.41* (.35*)	.48	-.28* (-.10)	-.32
Emotion Perception	.37* (.30*)	.44	-.34* (-.12)	-.40
Emotion Facilitation	.36* (.32*)	.45	-.20 (-.10)	-.25
Emotion Understanding	.38* (.32*)	.49	-.30* (-.16)	-.39
Emotion Management	.29* (.26)	.37	-.09 (.01)	-.11

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, BPRS = Brief Psychiatric Rating Scale

Change Note. Emotion perception is now no longer related to neurocognitive composite scores, and symptomatology now shows no significant relations with any MSCEIT scores under case deletion. MSCEIT total scores are now related to BPRS total scores under case inclusion.

[a]Higher scores indicate better performance

[b]Lower scores indicate less psychopathology

[c]True score estimates were corrected for unreliability using Spearman's (1904b) method

* $p < .05$, two-tailed.

3. Factor Structure

Screeplots of eigen value decomposition among correlation matrices of the MSCEIT under case deletion and case inclusion outlier conditions continued to indicate the presence of at most 2 eigen values greater than or equal to 1, suggesting at most a 2-factor solution. As can be seen in Table A1.3., the factor structure and loadings continued to remain highly similar for the 1 and 2-factor solutions for the MSCEIT across different outlier conditions. The only notable exception to this was the emergence of a split factor loading for Task #4 in the 2-factor solution under the case deletion condition. It is suggested that this result stems from the removal of cases from the already modest sample of participants, which further precludes the detection of more complex factor-analytic solutions. Results from the case inclusion method support this suggestion, as the 2-factor structure is identical to that found with the winsorization approach. In addition, correlation matrices of the associations between MSCEIT branch scores under different outlier conditions also suggest the original 2-factor solution reported in the primary analyses, as Branches 1 and 3, and Branches 2 and 4 displayed strong relations with each other in both case deletion and case inclusion outlier conditions (see Table A1.4). Consequently, regardless of the method of addressing outliers, factor-analytic results largely remain the same and suggest a 1 or at most a 2-factor solution for the MSCEIT.

Table A1.3. *Factor Structure of the Mayer-Salovey-Caruso Emotional Intelligence Test*

	Winsorization			Case Deletion			Case Inclusion		
	N	1 Fact[a]	2 Fact[b]	*N*	1 Fact[c]	2 Fact[d]	*N*	1 Fact[e]	2 Fact[f]
Variable		F1	F1 F2		F1	F1 F2		F1	F1 F2
				Standardized Factor Loadings					
Branch 1 - Emotion Perception									
Task 1 - Faces	57	**.47**	-.16 **.72**	50	**.48**	-.12 **.63**	57	**.47**	-.16 **.72**
Task 2 - Pictures	57	**.59**	.07 **.59**	50	**.56**	-.04 **.65**	57	**.59**	.07 **.59**
Branch 2 - Emotion Facilitation									
Task 3 - Facilitation	57	**.60**	**.42** .23	50	**.60**	**.46** .22	57	**.60**	**.42** .23
Task 4 - Sensations	57	**.75**	**.62** .20	50	**.72**	**.38** **.43**	57	**.75**	**.62** .20
Branch 3 - Emotion Understanding									
Task 5 - Changes	57	**.72**	.11 **.70**	50	**.70**	-.01 **.77**	57	**.72**	.11 **.70**
Task 6 - Blends	57	**.74**	.27 **.55**	50	**.73**	.14 **.66**	57	**.74**	.27 **.55**
Branch 4 - Emotion Management									
Task 7 - Management	57	**.73**	**.92** -.09	50	**.71**	**.98** -.05	57	**.73**	**.92** -.09
Task 8 - Relationships	57	**.76**	**.72** .13	50	**.73**	**.52** **.31**	57	**.76**	**.72** .13
				Factor Correlations					
Factor		F1	F2		F1	F2		F1	F2
F1		-			-			-	
F2		.62	-		.58	-		.62	-

Note. Factor loadings greater than .30 appear in boldface. Analyses were conducted on the combined treatment sample at baseline.

Change Note. Tasks 4 and 8 now have split loadings with Factor 1 under the case deletion.

[a]R^2 = .46, $\chi^2(20, N = 57) = 41.18$, p = .004

[b]R^2 = .48, $\chi^2(13, N = 57) = 21.38$, p = .066

[c]R^2 = .44, $\chi^2(20, N = 50) = 39.03$, p = .007

[d]R^2 = .48, $\chi^2(13, N = 50) = 18.81$, p = .129

[e]R^2 = .46, $\chi^2(20, N = 57) = 41.18$, p = .004

[f]R^2 = .48, $\chi^2(13, N = 57) = 21.38$, p = .066

Table A1.4. *Correlations Among Mayer-Salovey-Caruso Emotional Intelligence Test Branches*

MSCEIT Branch	1	2	3	4
Winsorization (N = 57)				
1. Branch 1 - Emotion Perception	-			
2. Branch 2 - Emotion Facilitation	.48*	-		
3. Branch 3 - Emotion Understanding	.58*	.61*	-	
4. Branch 4 - Emotion Management	.38*	.70*	.59*	-
Case Deletion (N = 50)				
1. Branch 1 - Emotion Perception	-			
2. Branch 2 - Emotion Facilitation	.47*	-		
3. Branch 3 - Emotion Understanding	.59*	.60*	-	
4. Branch 4 - Emotion Management	.35*	.66*	.60*	-
Case Inclusion (N = 57)				
1. Branch 1 - Emotion Perception	-			
2. Branch 2 - Emotion Facilitation	.48*	-		
3. Branch 3 - Emotion Understanding	.57*	.61*	-	
4. Branch 4 - Emotion Management	.38*	.70*	.59*	-

Note. Analyses were conducted on the combined treatment sample at baseline. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test

Change Note. No significant changes.

* $p < .05$, two-tailed.

B. AIM #2: EXAMINE THE CROSS-SECTIONAL RELATIONSHIP BETWEEN EMOTIONAL INTELLIGENCE AND SOCIAL DISABILITY

1. Bivariate Relationship Between Emotional Intelligence and Social Disability

Table A1.5 present cross-sectional correlations between MSCEIT performance and social disability under different outlier conditions. Results from both analyses continue to indicate little to no cross-sectional relation between MSCEIT performance and social disability, as was demonstrated in the primary analyses. When analyses were restricted to those individuals living with household members (see Table A1.6, relations again became somewhat stronger with general emotional intelligence and emotion regulation showing significant negative associations with household relations. This pattern of results is identical to those observed in primary analyses under conditions of winsorization.

Table A1.5. *Associations Between Emotional Intelligence and Social Disability at Baseline*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
			Winsorization (N = 57)		
General Emotional Intelligence[a]	-.12 (.03)	-.14 (.04)	-.02 (-.00)	-.07 (-.03)	.01 (.05)
Emotional Regulation	-.10 (-.01)	-.13 (-.06)	-.03 (.01)	-.06 (-.03)	-.02 (.01)
Emotional Knowledge	-.08 (.10)	-.09 (.14)	.09 (.10)	.05 (.12)	.02 (.07)
			Case Deletion (N = 50)		
General Emotional Intelligence[a]	-.09 (.03)	-.10 (.05)	-.03 (-.02)	-.08 (-.06)	.06 (.06)
Emotional Regulation	-.04 (.03)	-.06 (.00)	-.09 (-.07)	-.11 (-.10)	.04 (.05)
Emotional Knowledge	-.03 (.11)	-.03 (.17)	.13 (.14)	.06 (.13)	.06 (.07)
			Case Inclusion (N = 57)		
General Emotional Intelligence[a]	-.12 (.03)	-.14 (.04)	-.02 (-.00)	-.07 (-.03)	.01 (.05)
Emotional Regulation	-.10 (-.01)	-.13 (-.06)	-.03 (.01)	-.06 (-.03)	-.02 (.01)
Emotional Knowledge	-.08 (.10)	-.09 (.14)	.09 (.10)	.05 (.12)	.02 (.07)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

Change Note. No significant changes.

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

Table A1.6. *Associations Between Emotional Intelligence and Social Disability at Follow-up*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
Winsorization (N = 47)					
General Emotional Intelligence[a]	-.03 (.03)	.07 (.16)	-.04 (-.10)	-.34* (-.27†)	-.08 (-.06)
Emotional Regulation	-.06 (-.03)	.07 (.10)	-.04 (-.09)	-.41* (-.38*)	-.09 (-.07)
Emotional Knowledge	.08 (.19)	.14 (.32†)	.05 (-.04)	-.12 (-.02)	-.01 (.05)
Case Deletion (N = 42)					
General Emotional Intelligence[a]	-.02 (-.01)	.06 (.10)	-.00 (-.11)	-.37* (-.31†)	-.04 (-.02)
Emotional Regulation	-.08 (-.09)	.01 (-.02)	-.02 (-.08)	-.50* (-.48*)	-.05 (-.04)
Emotional Knowledge	.11 (.18)	.18 (.32*)	.09 (-.05)	-.11 (-.02)	.02 (.09)
Case Inclusion (N = 47)					
General Emotional Intelligence[a]	-.03 (.03)	.07 (.16)	-.04 (-.10)	-.34* (-.27†)	-.08 (-.06)
Emotional Regulation	-.06 (-.03)	.07 (.10)	-.04 (-.09)	-.41* (-.38*)	-.09 (-.07)
Emotional Knowledge	.08 (.19)	.14 (.32†)	.05 (-.04)	-.12 (-.02)	-.01 (.05)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline. MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

Change Note. No significant changes.

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

2. Relationship Between Emotional Intelligence and Social Disability, Adjusting For Neurocognition and Psychopathology

As suggested by previous bivariate relations between emotion intelligence and social disability, emotion intelligence made little to no cross-sectional contribution to social disability after adjusting for neurocognitive function and psychopathology, regardless of the outlier method employed (see Tables A1.7a-c).

Table A1.7a. *Associations Between Emotional Intelligence and Social Disability After Adjusting for Neurocognitive Function and Psychopathology (Winsorization)*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
Neurocognitive Composite	.05	.07	.09	.05	.04	.15	-.27	1.07	-.04	.03	.08	.07	-.03	.04	-.08
BPRS Total	.96	.32	.46*	.76	.18	.58*	.17	5.09	.01	.08	.30	.05	.16	.20	.14
General Emotional Intelligence															
Step 3															
General Emotional Intelligence[b]	.00	.00	.02	.00	.00	.00	.00	.07	.01	-.00	.00	-.04	.00	.00	.10
Step 4															
General Emotional Intelligence X Gender	.01	.01	.88	.01	.00	.81	.06	.13	.39	-.01	.01	-.73	.00	.01	.73
Emotional Regulation															
Step 3															
Emotional[b] Regulation	-.00	.01	-.04	-.00	.00	-.11	.02	.09	.03	-.00	.01	-.05	.00	.00	.04
Step 4															
Emotional Regulation X Gender	.01	.01	.91	.01	.01	1.21	-.06	.19	-.40	-.01	.01	-1.69	.00	.01	.56
Emotional Knowledge															
Step 3															
Emotional[b] Knowledge	.01	.01	.14	.00	.00	.16	.08	.10	.14	.00	.01	.16	.00	.00	.13
Step 4															
Emotional Knowledge X Gender	.01	.01	1.34	.01	.01	.78	.17	.17	1.14	-.01	.01	-.81	.01	.01	1.39

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 16). Step 2 is only provided

once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

Table A1.7b. *Associations Between Emotional Intelligence and Social Disability After Adjusting for Neurocognitive Function and Psychopathology (Case Deletion)*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
Neurocognitive Composite	.04	.08	.07	.04	.04	.11	-.61	1.26	-.08	.03	.09	.06	-.01	.05	-.04
BPRS Total	.73	.34	.35*	.70	.18	.51*	-2.4	5.54	-.08	-.07	.34	-.04	.10	.23	.08
General Emotional Intelligence															
Step 3															
General Emotional Intelligence[b]	-.00	.00	-.00	-.00	.00	-.00	.00	.08	.00	-.00	.00	-.08	.00	.00	.08
Step 4															
General Emotional Intelligence X Gender	.01	.01	1.03	.01	.00	.97	.05	.14	.33	-.01	.01	-.77	.00	.01	.71
Emotional Regulation															
Step 3															
Emotional[b] Regulation	.00	.01	.00	-.00	.00	-.05	-.03	.11	-.05	-.00	.01	-.13	.00	.00	.06
Step 4															
Emotional Regulation X Gender	.01	.01	.80	.01	.01	1.08	-.02	.20	-.15	-.01	.01	-1.50	.00	.01	.31
Emotional Knowledge															
Step 3															
Emotional[b] Knowledge	.00	.01	.12	.00	.00	.16	.11	.10	.19	.01	.01	.16	.00	.00	.10
Step 4															
Emotional Knowledge X Gender	.02	.01	1.55	.01	.01	.92	.11	.18	.77	-.01	.01	-1.24	.01	.01	1.54

Note. Analyses were conducted on the combined treatment sample at baseline (N = 50). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

Change Note. No significant changes.

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not

presented to reduce visual clutter and avoid redundancy (see Table 16). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

Table A1.7c. *Associations Between Emotional Intelligence and Social Disability After Adjusting for Neurocognitive Function and Psychopathology (Case Inclusion/No Treatment)*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
Neurocognitive Composite	.03	.06	.06	.04	.03	.12	-.17	.93	-.03	.03	.07	.08	-.03	.04	-.11
BPRS Total	.79	.27	.43*	.62	.15	.52*	1.9	4.34	.08	.08	.26	.06	.12	.18	.11
General Emotional Intelligence															
Step 3															
General Emotional Intelligence[b]	.00	.00	.05	.00	.00	.04	.01	.07	.01	-.00	.00	-.04	.00	.00	.12
Step 4															
General Emotional Intelligence X Gender	.01	.01	.76	.00	.00	.67	.05	.13	.36	-.01	.01	-.76	.00	.01	.65
Emotional Regulation															
Step 3															
Emotional[b] Regulation	-.00	.01	-.01	-.00	.00	-.08	.02	.09	.03	-.00	.01	-.05	.00	.00	.06
Step 4															
Emotional Regulation X Gender	.01	.01	.82	.01	.01	1.13	-.07	.19	-.45	-.01	.01	-1.74	.00	.01	.49
Emotional Knowledge															
Step 3															
Emotional[b] Knowledge	.01	.01	.17	.01	.00	.20	.09	.10	.15	.01	.01	.16	.00	.00	.16
Step 4															
Emotional Knowledge X Gender	.01	.01	1.11	.00	.01	.50	.16	.17	1.06	-.01	.01	-.90	.01	.01	1.26

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

Change Note. No significant changes.

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not

presented to reduce visual clutter and avoid redundancy (see Table 16). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

C. AIM #3: EXAMINE THE LONGITUDINAL CONTRIBUTION OF CHANGES IN EMOTIONAL INTELLIGENCE TO SOCIAL DISABILITY

1. Relationship Between Treatment and Changes in Emotional Intelligence and Social Disability

Table A1.8 present relations between treatment assignment and changes in emotional intelligence and social disability under case deletion and case inclusion outlier conditions. As can be seen in these Tables, CET continued to demonstrate a significant beneficial effect on emotion intelligence, overall social disability, and social leisure in both outlier conditions. The only departure from the primary analyses in these results is under the case inclusion condition where CET now demonstrated only a marginally significant beneficial effect on social leisure (p = .051).

Table A1.8. *Effect of Treatment on Changes in Emotional Intelligence and Social Disability*

	EST		CET				
	Baseline	Year 1	Baseline	Year 1	Relationship Between Changes and Treatment[c]		
Variable	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*F*	*p*[d]	*R*
Winsorization (*N* = 47)							
MSCEIT[a]							
General Emotional Intelligence	85.41 (14.30)	84.83 (15.52)	85.53 (16.89)	92.38 (14.05)	8.07	.007	.39
Emotional Knowledge	92.85 (12.43)	91.75 (12.05)	89.92 (11.04)	95.97 (10.39)	6.56	.014	.36
SAS-II[b]							
Total	1.42 (.41)	1.33 (.46)	1.46 (.59)	1.11 (.50)	4.47	.040	-.30
Social Leisure	.79 (.21)	.74 (.32)	.89 (.31)	.65 (.40)	4.52	.039	-.30
Case Deletion (*N* = 42)							
MSCEIT[a]							
General Emotional Intelligence	85.58 (13.90)	84.17 (15.80)	86.03 (17.35)	93.66 (13.59)	10.68	.002	.46
Emotional Knowledge	93.62 (12.28)	91.78 (12.44)	90.02 (11.45)	96.91 (10.23)	8.27	.006	.41
SAS-II[b]							
Total	1.35 (.36)	1.30 (.47)	1.49 (.57)	1.12 (.51)	4.39	.042	-.31
Social Leisure	.77 (.21)	.76 (.34)	.91 (.30)	.64 (.40)	5.89	.020	-.35
Case Inclusion (*N* = 47)							
MSCEIT[a]							
General Emotional Intelligence	85.41 (14.30)	84.83 (15.52)	85.53 (16.89)	92.38 (14.05)	8.07	.007	.39
Emotional Knowledge	92.85 (12.43)	91.75 (12.05)	89.92 (11.04)	95.97 (10.39)	6.56	.014	.36
SAS-II[b]							
Total	1.42 (.41)	1.33 (.46)	1.46 (.59)	1.11 (.50)	4.47	.040	-.30
Social Leisure	.79 (.21)	.74 (.32)	.89 (.33)	.65 (.40)	4.00	.051	-.28

Note. Analyses were conducted on the combined, follow-up treatment sample (*N* = 47). CET = Cognitive Enhancement Therapy; EST = Enriched Supportive Therapy; MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

Change Note. CET effects on social leisure are now only marginally significant under case inclusion.

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

[c]Results from regression models predicting residualized change scores from treatment assignment

[d]Two-tailed test.

2. Relationship Between Changes in Emotional Intelligence and Social Disability

Relations between changes in emotional intelligence and social disability under different outlier conditions all remained the same, with significant relations existing between changes in emotion regulation and changes in overall social disability and household relations (see Table A1.9). As with the primary analyses, the same pattern of results was observed when the sample was restricted the individuals living with household members, where the same relations persisted and became somewhat stronger (see Table A1.10). In addition, as can be seen in Tables A1.11a-c, these relations between changes in emotion regulation and changes in overall social disability and household relations persisted after adjusting for changes in neurocognitive function and psychopathology, as they did in the primary analyses. However, under the case deletion condition, relations between changes in emotion regulation and overall social disability were now only marginally significant (p = .08). The consistency of these results with the primary analyses all continue to indicate a modest relation between improvements in emotion regulation and improved household relations and overall social functioning.

Table A1.9. *Associations Between Changes in Emotional Intelligence and Social Disability*

Variable	ΔSAS-II Total[b]	ΔInterpersonal Anguish	ΔSexual Relations	ΔHousehold Relations	ΔSocial Leisure
Winsorization (N = 47)					
ΔGeneral Emotional Intelligence[a]	-.13 (-.11)	-.04 (.00)	.02 (-.04)	-.19 (-.27†)	-.09 (-.08)
ΔEmotional Regulation	-.31* (-.31*)	-.18 (-.15)	.06 (-.04)	-.34* (-.45*)	-.20 (-.18)
ΔEmotional Knowledge	.11 (.11)	.22† (.24†)	.05 (-.00)	.08 (.03)	-.02 (-.00)
Case Deletion (N = 42)					
ΔGeneral Emotional Intelligence[a]	-.13 (-.10)	-.02 (.02)	.02 (-.05)	-.20 (-.28†)	-.08 (-.06)
ΔEmotional Regulation	-.32* (-.30†)	-.19 (-.17)	.08 (.00)	-.39* (-.51*)	-.20 (-.17)
ΔEmotional Knowledge	.12 (.12)	.26† (.27†)	.05 (-.03)	.10 (.05)	-.02 (.02)
Case Inclusion (N = 47)					
ΔGeneral Emotional Intelligence[a]	-.13 (-.11)	-.04 (.00)	.02 (-.04)	-.19 (-.27†)	-.08 (-.08)
ΔEmotional Regulation	-.31* (-.31*)	-.18 (-.15)	.06 (-.04)	-.34* (-.45*)	-.18 (-.17)
ΔEmotional Knowledge	.11 (.11)	.22† (.24†)	.05 (-.00)	.08 (.03)	-.03 (-.00)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample (N = 47). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

Change Note. No significant changes.

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†p < .15, * p < .05, two-tailed.

Table A1.10. *Associations Between Changes in Emotional Intelligence and Social Disability Among Individuals Living With Significant Others*

Variable	ΔSAS-II Total[b]	ΔInterpersonal Anguish	ΔSexual Relations	ΔHousehold Relations	ΔSocial Leisure
Winsorization (N = 47)					
ΔGeneral Emotional Intelligence[a]	-.28† (-.28†)	-.09 (-.05)	.04 (-.03)	-.19 (-.27†)	-.19 (-.18)
ΔEmotional Regulation	-.42* (-.41*)	-.23 (-.18)	.06 (-.00)	-.34* (-.46*)	-.25 (-.24)
ΔEmotional Knowledge	-.02 (-.07)	.24 (.22)	.06 (-.01)	.08 (.03)	-.15 (-.13)
Case Deletion (N = 32)					
ΔGeneral Emotional Intelligence[a]	-.29† (-.27†)	-.08 (-.01)	.03 (-.04)	-.20 (-.29†)	-.19 (-.18)
ΔEmotional Regulation	-.45* (-.42*)	-.25 (-.18)	.07 (.00)	-.39* (-.51*)	-.26 (-.25)
ΔEmotional Knowledge	.00 (-.06)	.29† (.27†)	.04 (-.05)	.10 (.05)	-.15 (-.13)
Case Inclusion (N = 47)					
ΔGeneral Emotional Intelligence[a]	-.28† (-.28†)	-.09 (-.05)	.04 (-.03)	-.19 (-.27†)	-.19 (-.18)
ΔEmotional Regulation	-.42* (-.41*)	-.23 (-.18)	.06 (-.00)	-.34* (-.46*)	-.23 (-.22)
ΔEmotional Knowledge	-.02 (-.07)	.24 (.22)	.06 (-.01)	.08 (.03)	-.15 (-.13)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample (N = 35). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

Change Note. No significant changes.

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†p < .15, * p < .05, two-tailed.

Table A1.11a. *Associations Between Changes in Emotional Intelligence and Social Disability After Adjusting for Changes in Neurocognitive Function and Psychopathology (Winsorization)*

	Δ SAS-II Total[c]			Δ Interpersonal Anguish			Δ Sexual Relations			Δ Household Relations			Δ Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
ΔNeurocognitive Composite	-.04	.08	-.07	.04	.05	.13	-1.05	1.28	-.13	.02	.06	.06	-.09	.06	-.23
ΔBPRS Total	1.08	.25	.59*	.58	.16	.51*	9.62	4.29	.33*	.61	.24	.44*	.71	.20	.50*
General Emotional Intelligence															
Step 3															
ΔGeneral Emotional Intelligence[b]	-.01	.01	-.18	-.00	.00	-.10	-.01	.09	-.02	-.01	.01	-.38*	-.00	.00	-.08
Step 4															
ΔGeneral Emotional Intelligence X Gender	-.00	.02	-.01	.01	.01	.16	-.19	.25	-.12	.01	.01	.11	-.01	.01	-.10
Emotional Regulation															
Step 3															
ΔEmotional[b] Regulation	-.02	.01	-.32*	-.01	.01	-.18	.01	.14	.02	-.02	.01	-.48*	-.01	.01	-.13
Step 4															
ΔEmotional Regulation X Gender	.00	.03	.02	.02	.02	.14	-.20	.43	-.07	.02	.02	.14	-.01	.02	-.06
Emotional Knowledge															
Step 3															
ΔEmotional[b] Knowledge	-.00	.01	-.04	.00	.00	.08	-.03	.11	-.05	-.01	.01	-.15	-.00	.01	-.09
Step 4															
ΔEmotional Knowledge X Gender	-.01	.02	-.11	.01	.01	.17	-.22	.25	-.15	.01	.02	.10	-.02	.01	-.20

Note. Analyses were conducted on the combined, follow-up treatment sample (N = 47). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 20). Step 2 is only provided once to avoid

redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

Table A1.11b. *Associations Between Changes in Emotional Intelligence and Social Disability After Adjusting for Changes in Neurocognitive Function and Psychopathology (Case Deletion)*

	Δ SAS-II Total[c]			Δ Interpersonal Anguish			Δ Sexual Relations			Δ Household Relations			Δ Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
ΔNeurocognitive Composite	-.04	.08	-.07	.05	.05	.14	-.85	1.41	-.10	.00	.07	.00	-.10	.06	-.22
ΔBPRS Total	1.22	.27	.62*	.58	.17	.49*	11.2	4.56	.37*	.65	.25	.47*	.88	.20	.58*
General Emotional Intelligence															
Step 3															
ΔGeneral Emotional Intelligence[b]	-.01	.01	-.17	-.00	.00	-.08	-.03	.10	-.05	-.01	.01	-.39*	-.00	.00	-.07
Step 4															
ΔGeneral Emotional Intelligence X Gender	.00	.02	.00	.01	.01	.16	-.20	.25	-.13	.01	.01	.15	-.01	.01	-.08
Emotional Regulation															
Step 3															
ΔEmotional[b] Regulation	-.02	.01	-.26†	-.01	.01	-.16	.06	.14	.07	-.02	.01	-.50*	-.00	.01	-.09
Step 4															
ΔEmotional Regulation X Gender	.01	.03	.03	.02	.02	.18	-.09	.44	-.04	.01	.02	.11	-.01	.02	-.06
Emotional Knowledge															
Step 3															
ΔEmotional[b] Knowledge	-.00	.01	-.06	.00	.00	.10	-.08	.11	-.11	-.00	.01	-.13	-.00	.01	-.12
Step 4															
ΔEmotional Knowledge X Gender	-.01	.02	-.09	.01	.01	.17	-.27	.26	-.18	.02	.02	.21	-.01	.01	-.17

Note. Analyses were conducted on the combined, follow-up treatment sample (N = 47). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

Change Note. Relations between changes in emotion regulation and SAS-II total scores are now only marginally significant (p = .08).

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 20). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

†$p < .15$, *$p < .05$, two-tailed.

Table A1.11c. *Associations Between Changes in Emotional Intelligence and Social Disability After Adjusting for Changes in Neurocognitive Function and Psychopathology (Case Inclusion/No Treatment)*

	Δ SAS-II Total[c]			Δ Interpersonal Anguish			Δ Sexual Relations			Δ Household Relations			Δ Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
ΔNeurocognitive Composite	-.03	.08	-.05	.05	.05	.16	-.88	1.33	-.10	.02	.07	.06	-.09	.06	-.22
ΔBPRS Total	1.08	.25	.59*	.58	.16	.51*	9.56	4.30	.33*	.61	.24	.44*	.71	.20	.50*
General Emotional Intelligence															
Step 3															
ΔGeneral Emotional Intelligence[b]	-.01	.01	-.19	-.00	.00	-.10	-.02	.09	-.04	-.01	.01	-.38*	-.00	.00	-.08
Step 4															
ΔGeneral Emotional Intelligence X Gender	-.00	.02	-.01	.01	.01	.15	-.19	.25	-.12	.01	.01	.11	-.01	.01	-.11
Emotional Regulation															
Step 3															
ΔEmotional[b] Regulation	-.02	.01	-.32*	-.01	.01	-.18	.00	.13	.00	-.02	.01	-.48*	-.01	.01	-.13
Step 4															
ΔEmotional Regulation X Gender	.00	.03	.02	.02	.02	.14	-.20	.44	-.08	.02	.02	.14	-.01	.02	-.05
Emotional Knowledge															
Step 3															
ΔEmotional[b] Knowledge	-.00	.01	-.05	.00	.00	.07	-.04	.11	-.06	-.01	.01	-.15	-.00	.01	-.10
Step 4															
ΔEmotional Knowledge X Gender	-.01	.02	-.10	.01	.01	.17	-.22	.26	-.14	.01	.02	.10	-.02	.01	-.22

Note. Analyses were conducted on the combined, follow-up treatment sample (*N* = 47). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

Change Note. No significant changes.

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 20). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

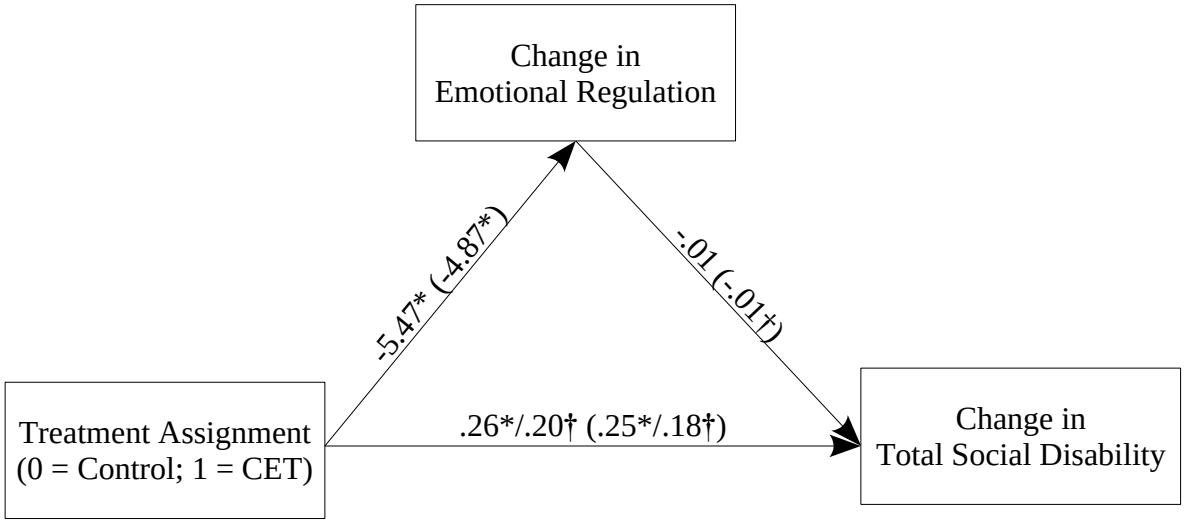
3. Mediating Effect of Improved Emotional Intelligence on The Relationship Between Treatment and Social Disability

Mediator analyses of the indirect effect of treatment assignment (CET) on improvements in social disability through improved emotion regulation under different outlier conditions are presented in Figures A1.2a and A1.2b As can be seen in these figures, the direct effect of treatment assignment was reduced in all outlier conditions once the mediating effect of changes in emotion regulation was introduced to into the models. However in the case deletion condition, there was no longer a marginally significant relation between changes in emotion regulation and changes in overall social disability. Nonetheless, the indirect effect of CET on improved overall social disability through improved emotion regulation continued to be marginally significant in both outlier conditions as it was in the primary analyses ($z' = 1.23$, $p = .145$ and $z' = 1.29$, $p = .118$, respectively). As with the primary analyses, these mediating effects were somewhat stronger when restricted to individuals living with significant others ($z' = 1.52$, $p = .045$ and $z' = 1.47$, $p = .053$, respectively; see Figures A1.3a and A1.3b). Under case deletion and case inclusion outlier conditions, reverse mediation analyses continued to point to a marginal bidirectional effect of changes in emotion regulation on overall social disability for the whole sample ($z' = -1.17$, $p = .146$ and $z' = -1.23$, $p = .125$, respectively) and those living with significant others ($z' = -1.51$, $p = .046$ and $z' = -1.52$, $p = .045$, respectively).

Mediator analyses adjusting for changes in neurocognitive function and psychopathology under case deletion and case inclusion outlier conditions also remained consistent with primary. Under case inclusion changes in emotion regulation continued to have an mediational effect on the relationship between treatment assignment and changes in overall social disability in the whole sample and those living with significant others ($z' = 1.48$, $p = .053$ and $z' = 1.28$, $p = .097$,

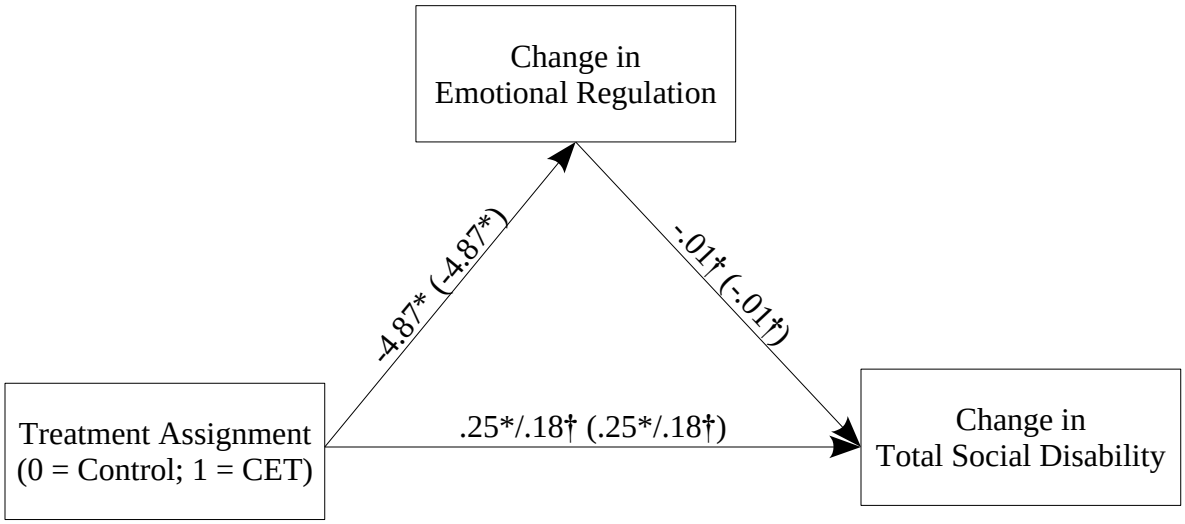
respectively), however these effects were somewhat reduced under the case deletion outlier condition ($z' = 1.20$, $p = .140$ and $z' = 1.22$, $p = .111$). As with the primary analyses, weak trends was observed for reverse mediation in the whole sample ($z' = -.95$, $p = .220$ and $z' = -1.09$, $p = .171$, respectively) and individuals living with significant others ($z' = -1.08$, $p = .166$ and $z' = -1.13$, $p = .154$, respectively) after adjusting for changes in neurocognitive function and psychopathology in both outlier conditions. Taken together, these analyses are largely consistent with the findings from the primary analyses using winsorization, and continue to tentatively support the possibility of changes in emotion regulation mediating, perhaps bidirectionally, the effect of CET on improvements in overall social disability.

Figure A1.2a. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation (Case Deletion)



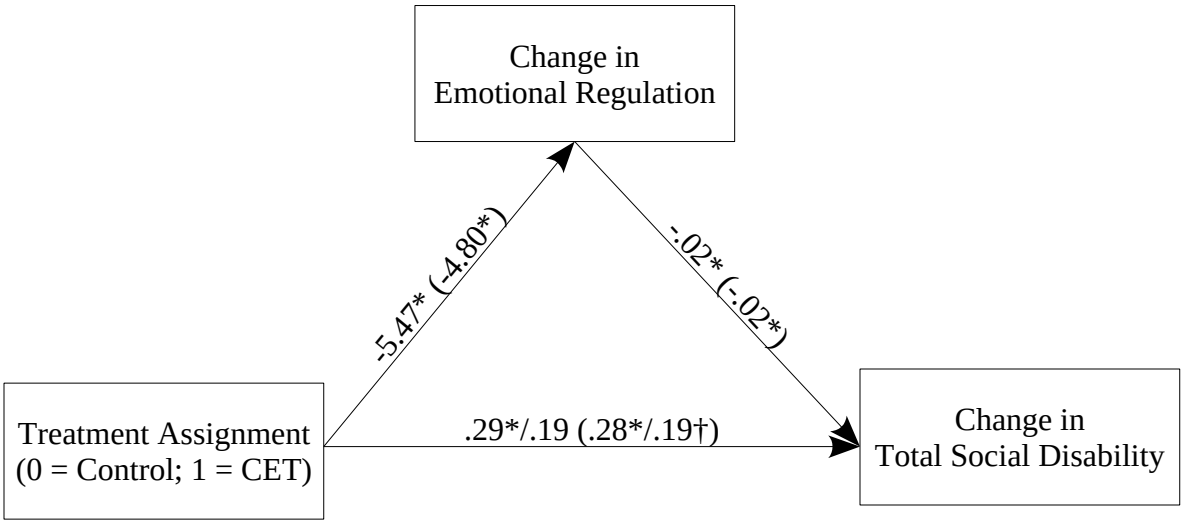
Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator. Coefficients from primary analyses using winsorization are presented in parentheses.
CET = Cognitive Enhancement Therapy
Change Note. Although the path between changes in emotion regulation and changes in social disability is no longer marginally significant ($p = .17$), a trend continues to remain indicating a marginally significant indirect effect of treatment assignment on changes in total social disability through changes in emotion regulation (indirect effect = .07, $p = .145$).
†$p < .15$, *$p < .05$, two-tailed.

Figure A1.2b. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation (Case Deletion)



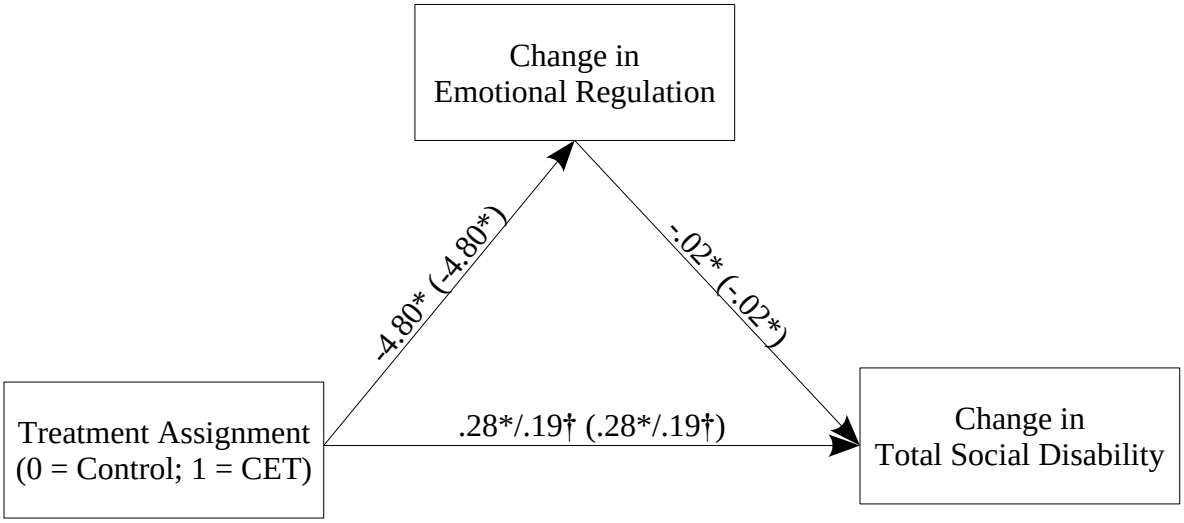
Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator. Coefficients from primary analyses using winsorization are presented in parentheses.
CET = Cognitive Enhancement Therapy
Change Note. No significant changes.
†$p < .15$, *$p < .05$, two-tailed.

Figure A1.3a. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation Among Participants Living with Significant Others (Case Deletion)



Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator. Coefficients from primary analyses using winsorization are presented in parentheses.
CET = Cognitive Enhancement Therapy
Change Note. No significant changes.
†$p < .15$, *$p < .05$, two-tailed.

Figure A1.3b. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotional Regulation Among Participants Living with Significant Others (Case Inclusion/No Treatment)



Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator. Coefficients from primary analyses using winsorization are presented in parentheses.
CET = Cognitive Enhancement Therapy
Change Note. No significant changes.
†$p < .15$, *$p < .05$, two-tailed.

APPENDIX B - RESULTS FOR ORIGINAL MSCEIT BRANCHES

Primary analyses examining the relationship between emotional intelligence and social disability in this research made use of the 1- and 2-factor solutions for the MSCEIT identified as part of the psychometric aims of this investigation. While the use of these factor solutions for primary analyses was empirically driven and most appropriate for this sample, and perhaps the schizophrenia population in general, the majority of research on the MSCEIT has made use of the 4-factor or branch model of emotional intelligence outlined by Salovey and Mayer (1999). To both explore the associations between MSCEIT performance and social disability at the branch level, and ensure some comparability between this investigation and other studies of the MSCEIT in healthy samples, analyses were conducted to examine the relations between individual MSCEIT branch scores and SAS-II scores, which are presented in this appendix.

Overall, the results of these analyses largely support the decision to use an empirically derived factor structure that collapsed MSCEIT branches 1 and 3 and branches 2 and 4, as social disability relations with branches 2 and 4 largely occurred in parallel. That is, when social disability was associated with branch 2 (emotion facilitation), it also tended to be associated with branch 4 (emotion management), although these were sometimes at the trend level. Consequently, at times it appeared that the joint contribution of emotion facilitation and management into an emotion regulation factor was more relevant to social disability than either of these 2 components alone, again supporting the empirically derived factor structure identified in this investigation. Further, whether combined or analyzed separately, branches 1 (emotion perception) and 3 (emotion understanding) continued to show little to no relation to social disability in this sample.

A. AIM #2: EXAMINE THE CROSS-SECTIONAL RELATIONSHIP BETWEEN EMOTIONAL INTELLIGENCE AND SOCIAL DISABILITY

1. Bivariate Relationship Between Emotional Intelligence and Social Disability

Tables A2.1 and A2.2 show cross-sectional relations between MSCEIT branch scores and SAS-II scores at both baseline and follow-up. Results are identical to those found in primary analyses, where no significant relations were observed at baseline, and only small relationships were observed between emotion facilitation and management, and household relations.

Table A2.1. *Associations Between Emotional Intelligence and Social Disability at Baseline*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
Branch 1 - Emotion Perception[a]	-.12 (.04)	-.16 (.09)	.11 (.10)	.10 (.16)	.01 (.03)
Branch 2 - Emotion Facilitation	-.14 (-.06)	-.21† (-.13)	.06 (.09)	-.04 (-.02)	-.05 (-.05)
Branch 3 - Emotion Understanding	-.04 (.08)	-.02 (.12)	-.01 (.00)	-.02 (.03)	.05 (.08)
Branch 4 - Emotion Management	-.08 (.01)	-.05 (.02)	-.15 (-.12)	-.14 (-.10)	-.02 (.04)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at baseline ($N = 57$). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

†$p < .15$, * $p < .05$, two-tailed.

Table A2.2. *Associations Between Emotional Intelligence and Social Disability at Follow-up*

Variable	SAS-II Total[b]	Interpersonal Anguish	Sexual Relations	Household Relations	Social Leisure
Branch 1 - Emotion Perception[a]	.01 (.09)	.09 (.20)	.04 (-.01)	-.08 (.04)	-.03 (.02)
Branch 2 - Emotion Facilitation	-.03 (.02)	.06 (.11)	.05 (-.02)	-.27† (-.28†)	-.05 (.01)
Branch 3 - Emotion Understanding	-.01 (.03)	.04 (.18)	-.06 (-.20)	-.15 (-.07)	-.12 (-.14)
Branch 4 - Emotion Management	-.09 (-.08)	.07 (.04)	-.20 (-.18)	-.51* (-.47*)	-.13 (-.16)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined treatment sample at follow-up (N = 47). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

†$p < .15$, * $p < .05$, two-tailed.

2. Relationship Between Emotional Intelligence and Social Disability, Adjusting For Neurocognition and Psychopathology

Table A2.3 shows results from a series of hierarchical linear regression analyses examining the relationship between MSCEIT branch scores and SAS-II scores after adjusting for demographic characteristics, neurocognitive function, and psychopathology. As found in the primary analyses in this investigation, no significant cross-sectional relations persisted between MSCEIT scores and SAS-II scores at baseline, after adjusting for neurocognitive function and psychopathology. In addition, no significant interactions with gender were observed.

Table A2.3. *Associations Between Emotional Intelligence and Social Disability After Adjusting for Neurocognitive Function and Psychopathology*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
Neurocognitive Composite	.05	.07	.09	.05	.04	.15	-.27	1.07	-.04	.03	.08	.07	-.03	.04	-.08
BPRS Total	.96	.32	.46*	.76	.18	.58*	.17	5.09	.01	.08	.30	.05	.16	.20	.14
Branch 1 - Emotion Perception															
Step 3															
Emotion Perception[b]	.00	.00	.05	.00	.00	.08	.05	.07	.13	.00	.00	.18	.00	.00	.07
Step 4															
Emotion Perception X Gender	.01	.01	1.07	.00	.00	.64	.03	.12	.24	.00	.01	.12	.01	.00	1.34
Branch 2 - Emotion Facilitation															
Step 3															
Emotion Facilitation[b]	.00	.00	-.06	-.00	.00	-.15	.05	.06	.12	-.00	.00	-.02	-.00	.00	-.01
Step 4															
Emotion Facilitation X Gender	.01	.01	.51	.01	.00	.89	-.04	.12	-.30	-.01	.01	-1.40	.00	.00	.35
Branch 3 - Emotion Understanding															
Step 3															
Emotion Understanding[b]	.00	.01	.11	.00	.00	.14	.01	.09	.02	.00	.01	.03	.00	.00	.14
Step 4															
Emotion Understanding X Gender	.01	.01	.65	.00	.01	.59	.24	.18	1.53	-.02	.01	-1.83	.00	.01	.14
Branch 4 - Emotion Management															
Step 3															
Emotion Management[b]	.00	.01	-.02	-.00	.00	-.04	-.06	.08	-.12	-.00	.01	-.12	.00	.00	.06
Step 4															
Emotion	.01	.01	.99	.01	.01	.79	.00	.19	.03	-.01	.01	-.82	.00	.01	.65

Management X Gender	

Note. Analyses were conducted on the combined treatment sample at baseline (N = 57). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 16). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

* $p < .05$, two-tailed.

B. AIM #3: EXAMINE THE LONGITUDINAL CONTRIBUTION OF CHANGES IN EMOTIONAL INTELLIGENCE TO SOCIAL DISABILITY

1. Relationship Between Treatment and Changes in Emotional Intelligence and Social Disability

Analyses of the relationship between treatment assignment and improvements in MSCEIT branch scores are presented in Table A2.4. As can be seen in this Table, the CET demonstrated significant effects on emotion facilitation and management, as well as emotion understanding. Marginally significant effects were also observed on emotion perception. These results are similar to those found in primary analyses, where CET effects on emotion regulation were slightly stronger.

Table A2.4. *Effect of Treatment on Changes in Emotional Intelligence and Social Disability*

	EST (*N* = 24)		CET (*N* = 23)				
	Baseline	Year 1	Baseline	Year 1	Relationship Between Changes and Treatment[c]		
Variable	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*F*	p^d	*R*
MSCEIT[a]							
Branch 1 - Emotion Perception	94.30 (16.79)	91.60 (17.22)	87.13 (15.98)	92.44 (16.54)	2.91	.095	.25
Branch 2 - Emotion Facilitation	94.55 (15.73)	89.36 (17.77)	93.42 (17.28)	96.73 (15.86)	4.64	.037	.31
Branch 3 - Emotion Understanding	86.49 (13.01)	88.39 (12.36)	87.96 (11.14)	94.73 (9.14)	5.46	.024	.33
Branch 4 - Emotion Management	84.36 (10.56)	85.25 (10.59)	88.50 (12.30)	92.92 (9.65)	5.14	.028	.32
SAS-II[b]							
Total	1.42 (.41)	1.33 (.46)	1.46 (.59)	1.11 (.50)	4.47	.040	-.30
Interpersonal Anguish	.96 (.34)	.85 (.33)	.97 (.29)	.81 (.29)	.37	.544	.09
Sexual Relations	15.37 (6.52)	15.17 (5.67)	12.83 (7.21)	10.99 (7.48)	1.59	.214	-.18
Household Relations	.68 (.38)	.64 (.34)	.59 (.34)	.47 (.34)	1.32	.259	.19
Social Leisure	.79 (.21)	.74 (.32)	.89 (.31)	.65 (.40)	4.52	.039	-.30

Note. Analyses were conducted on the combined, follow-up treatment sample (*N* = 47). CET = Cognitive Enhancement Therapy; EST = Enriched Supportive Therapy; MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate better performance

[b]Lower scores indicate less social disability

[c]Results from regression models predicting residualized change scores from treatment assignment

[d]Two-tailed test.

2. Relationship Between Changes in Emotional Intelligence and Social Disability

Tables A2.5 and A2.6 show longitudinal relations between MSCEIT branch and SAS-II total scores among the entire follow-up sample and those living with significant others. Consistent with the primary analyses of this research, emotion facilitation and management branches showed the most pervasive pattern of relations with changes in SAS-II scores, particularly with regard to overall social adjustment and household relations. These relations were also somewhat stronger when examined within the subsample of individuals living with significant others. In addition, several other relations with social adjustment were observed that were not present in the primary analyses. In the total sample, these included marginal relations between improvements in emotion facilitation and understanding, and improvement in social leisure. In addition, a significant relationship between improved emotion understanding and sexual relations existed in the total sample after adjusting for demographic characteristics. Again, some of the relations were stronger within the sample of individuals living with significant others. Further, an additional marginal relation between improved emotion management and reductions in interpersonal anguish were observed after adjusting for demographic characteristics in the sample of individuals living with significant others.

Analysis of the persistence of these relations after adjusting for neurocognitive functioning and psychopathology indicated that only improvements in emotion facilitation remained significantly associated with improvements in overall social disability, which is partially consistent with primary analyses. In addition, consistent with primary analyses, improvements in emotion facilitation and management were also associated with improved household relations after adjusting for neurocognitive function and psychopathology. A single relationship was found between improved emotion understanding and improved sexual relations that was not present in primary analyses. Further, while no gender by MSCEIT score

interactions were found in primary analyses in this investigation, a significant gender by changes in emotion facilitation interaction effect on changes in household relations. Closer inspection of this interaction indicated that while a strong significant relation existed between improvements in emotion facilitation and improved household relations among males (β = -.80, p < .001), a marginally significant relation existed among females indicating the opposite pattern of results, with improved emotion facilitation being associated with worse household relations (β = .78, p = .104). This latter relationship needs to be interpreted with caution given the modest number of females for whom household relations data were available at follow-up (n = 12).

Table A2.5. *Associations Between Changes in Emotional Intelligence and Social Disability*

Variable	ΔSAS-II Total[b]	ΔInterpersonal Anguish	ΔSexual Relations	ΔHousehold Relations	ΔSocial Leisure
ΔBranch 1 - Emotion Perception[a]	.09 (.10)	.17 (.14)	.10 (.11)	-.05 (-.09)	.04 (.05)
ΔBranch 2 - Emotion Facilitation	-.29* (-.30*)	-.13 (-.11)	.12 (.00)	-.20 (-.32†)	-.24† (-.21)
ΔBranch 3 - Emotion Understanding	-.10 (-.11)	-.01 (.08)	-.18 (-.33*)	.10 (.07)	-.23† (-.24†)
ΔBranch 4 - Emotion Management	-.23† (-.22†)	-.15 (-.17)	-.15 (-.14)	-.50* (-.53*)	-.06 (-.09)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample (N = 47). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†p < .15, * p < .05, two-tailed.

Table A2.6. *Associations Between Changes in Emotional Intelligence and Social Disability Among Individuals Living With Significant Others*

Variable	ΔSAS-II Total[b]	ΔInterpersonal Anguish	ΔSexual Relations	ΔHousehold Relations	ΔSocial Leisure
ΔBranch 1 - Emotion Perception[a]	.00 (-.04)	.23 (.17)	.08 (.06)	-.05 (-.09)	-.05 (-.03)
ΔBranch 2 - Emotion Facilitation	-.35* (-.34*)	-.18 (-.13)	.14 (.07)	-.20 (-.32†)	-.24 (-.23)
ΔBranch 3 - Emotion Understanding	-.19 (-.20)	-.07 (-.02)	-.16 (-.31†)	.10 (.08)	-.38* (-.35*)
ΔBranch 4 - Emotion Management	-.46* (-.46*)	-.25 (-.31†)	-.19 (-.17)	-.50* (-.54*)	-.18 (-.20)

Note. Partial correlations adjusting for gender, education, illness length and diagnosis appear in parentheses. Analyses were conducted on the combined, follow-up treatment sample of individuals who were living with a significant other (N = 35). MSCEIT = Mayer-Salovey-Caruso Emotional Intelligence Test, SAS-II = Social Adjustment Scale-II

[a]Higher scores indicate improvements in performance

[b]Lower scores indicate reductions in social disability

†$p < .15$, *$p < .05$, two-tailed.

Table A2.7. *Associations Between Changes in Emotional Intelligence and Social Disability After Adjusting for Changes in Neurocognitive Function and Psychopathology*

	SAS-II Total[c]			Interpersonal Anguish			Sexual Relations			Household Relations			Social Leisure		
Variable/Step[a]	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Neurocognitive Function and Psychopathology															
Step 2															
ΔNeurocognitive Composite	-.04	.08	-.07	.04	.05	.13	-1.05	1.28	-.13	.02	.06	.06	-.09	.06	-.23
BPRS Total	1.08	.25	.59*	.58	.16	.51*	9.62	4.29	.33*	.61	.24	.44*	.71	.20	.50*
Branch 1 - Emotion Perception															
Step 3															
ΔEmotion Perception[b]	-.00	.00	-.03	.00	.00	.02	.03	.07	.06	-.01	.00	-.23	-.00	.00	-.04
Step 4															
ΔEmotion Perception X Gender	-.01	.01	-.09	.00	.01	.06	-.18	.17	-.17	.01	.01	.24	-.01	.01	-.10
Branch 2 - Emotion Facilitation															
Step 3															
ΔEmotion Facilitation[b]	-.01	.00	-.34*	-.00	.00	-.17	.02	.07	.05	-.01	.00	-.36*	-.00	.00	-.19
Step 4															
ΔEmotion Facilitation X Gender	.01	.01	.15	.01	.01	.13	-.07	.16	-.07	.02	.01	.41*	.00	.01	.08
Branch 3 - Emotion Understanding															
Step 3															
ΔEmotion Understanding[b]	-.01	.01	-.12	.00	.00	.03	-.22	.11	-.32*	.00	.01	.10	-.01	.01	-.22
Step 4															
ΔEmotion Understanding X Gender	-.00	.02	-.04	.01	.01	.26	.02	.25	.01	-.02	.02	-.22	-.02	.01	-.22
Branch 4 - Emotion Management															
Step 3															
ΔEmotion Management[b]	-.01	.01	-.18	-.00	.00	-.16	-.06	.11	-.08	-.02	.01	-.49*	-.00	.01	-.00
Step 4															

ΔEmotion Management X Gender	-.00	.02	-.02	.01	.01	.15	-.18	.27	-.10	-.00	.01	-.06	-.02	.01	-.21

Note. Analyses were conducted on the combined, follow-up treatment sample (N = 47). SAS-II = Social Adjustment Scale-II, BPRS = Brief Psychiatric Rating Scale

[a]Step 1 adjusts for shared variance with demographic and clinical characteristics and is not presented to reduce visual clutter and avoid redundancy (see Table 20). Step 2 is only provided once to avoid redundancy.

[b]Higher scores indicate better performance

[c]Lower scores indicate less social disability

†$p < .15$, *$p < .05$, two-tailed.

3. Mediating Effect of Improved Emotional Intelligence on The Relationship Between Treatment and Social Disability

Despite the slightly increased pattern of pervasive relations between MSCEIT branch and SAS-II scores than was present in the primary analyses, only a single model continued to satisfy all of Baron and Kenny's (1986) criteria for proceeding with statistical tests of mediation (see Table A2.8). Consistent with primary analyses, this model consisted of the mediating relationship of emotion facilitation on CET treatment effects on overall social disability. In primary analyses it was the combination of emotion facilitation and management in an emotion regulation factor that was the candidate mediator between CET and changes in overall social disability.

Table A2.8. *Summary of Possible Emotional Intelligence Mediational Models of the Relationship Between Treatment and Changes in Social Disability*

Mediation Model	Criteria 1: Relationship between treatment and Δsocial disability?	Criteria 2: Relationship between treatment and Δemotional intelligence?	Intermediate Criteria 3: Relationship between Δsocial disability and Δemotional intelligence?
1. Treatment → ΔEmotion Perception → ΔSAS-II Total	X		
2. Treatment → ΔEmotion Perception → ΔInterpersonal Anguish			
3. Treatment → ΔEmotion Perception → ΔSexual Relations			
4. Treatment → ΔEmotion Perception → ΔHousehold Relations			
5. Treatment → ΔEmotion Perception → ΔSocial Leisure	X		
6. Treatment → ΔEmotion Facilitation → ΔSAS-II Total	X	X	X
7. Treatment → ΔEmotion Facilitation → ΔInterpersonal Anguish		X	
8. Treatment → ΔEmotion Facilitation → ΔSexual Relations		X	
9. Treatment → ΔEmotion Facilitation → ΔHousehold Relations		X	X
10. Treatment → ΔEmotion Facilitation → ΔSocial Leisure	X	X	
11. Treatment → ΔEmotion Understanding → ΔSAS-II Total	X	X	
12. Treatment → ΔEmotion Understanding → ΔInterpersonal Anguish		X	
13. Treatment → ΔEmotion Understanding → ΔSexual Relations		X	X
14. Treatment → ΔEmotion Understanding → ΔHousehold Relations		X	
15. Treatment → ΔEmotion Understanding → ΔSocial Leisure	X	X	

16. Treatment → ΔEmotion Management → ΔSAS-II Total	X	X	
17. Treatment → ΔEmotion Management → ΔInterpersonal Anguish		X	
18. Treatment → ΔEmotion Management → ΔSexual Relations		X	
19. Treatment → ΔEmotion Management → ΔHousehold Relations		X	X
20. Treatment → ΔEmotion Management → ΔSocial Leisure	X	X	

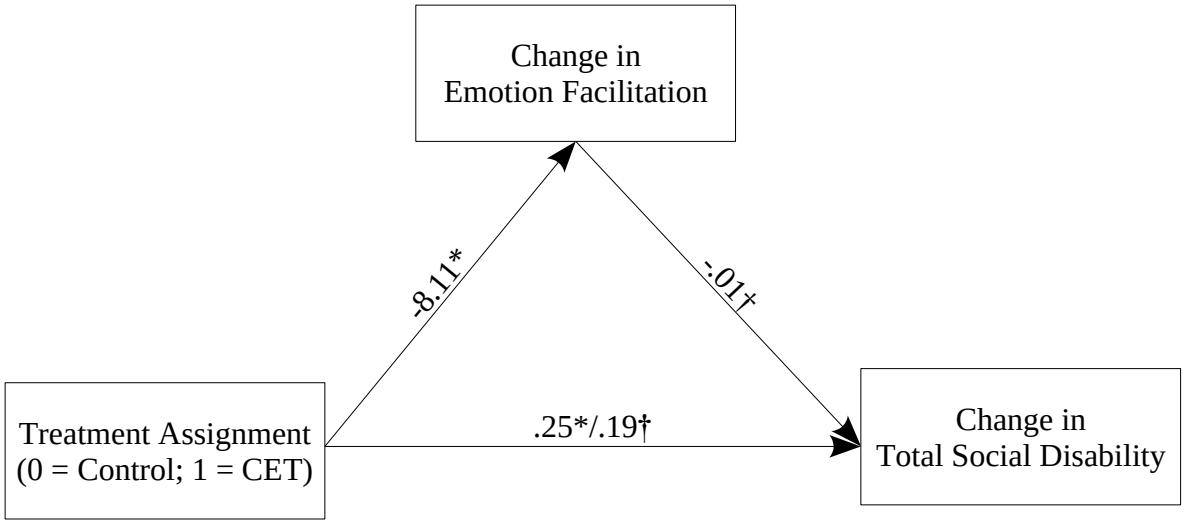
Results from mediator analyses examining the mediational effect of changes in emotion facilitation on CET effects on overall social disability (see Figure A2.1) indicated that consistent with primary analyses, emotion facilitation marginally mediated CET effects on overall social disability ($z' = 1.23$, $p = .121$), although reverse mediation was also suggested ($z' = -1.23$, $p = .120$). After adjusting for demographic characteristics, neurocognitive function, and psychopathology, trends were still present for emotion facilitation mediating CET effects on overall social disability ($z' = 1.34$, $p = .082$), as well as for reverse mediation ($z' = -1.11$, $p = .167$).

Investigation of those living with significant others revealed that the mediational effects of changes in emotion facilitation on CET effects on overall social disability were not stronger in the subsample of individuals living with significant others ($z' = 1.14$, $p = .136$) (see Figure A2.2), which is contrary to results from primary analyses finding somewhat stronger mediational effects among individuals living with significant others. However, as suggested by the stronger relationship between emotion management and overall social disability in this subsample (see Table A2.6), change in emotion management ability was a significant mediator of CET effects on overall social disability among individuals living with family members ($z' = 1.50$, $p = .034$) (see Figure A2.3). This effect remained marginally significant after adjusting for demographic characteristics, and neurocognitive functioning and psychopathology ($z' = 1.18$, $p = .125$), and the presence of reverse mediation was suggested both before ($z' = -1.61$, $p = .032$) and after adjusting for these confounders ($z' = -1.13$, $p = .144$).

Taken together, these branch-level mediational analyses support primary analyses of the mediational effects of an emotion regulation factor on CET effects on changes in overall social disability. These results also potentially add some specificity to the findings in primary analyses,

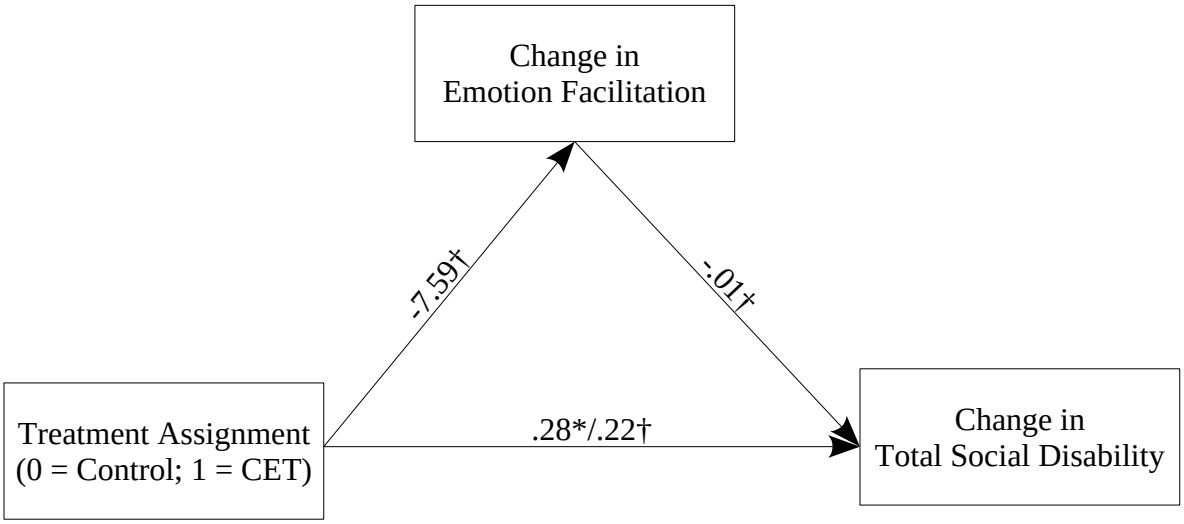
by indicating that changes in emotion facilitation may have contributed to the most of the mediation effects among the whole sample, whereas the increase in mediational effects among individuals living with family members may have been due primarily to relations with changes in emotion management. All effects may nonetheless be bidirectional, as suggested in primary analyses. Further, many of these effects are only marginally significant and these mediational models represent a small minority of possible models that were not appropriate for testing. As such, these results continue to need to be interpreted with caution until studies employing larger sample sizes can provide confirmation of these findings.

Figure A2.1. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotion Facilitation



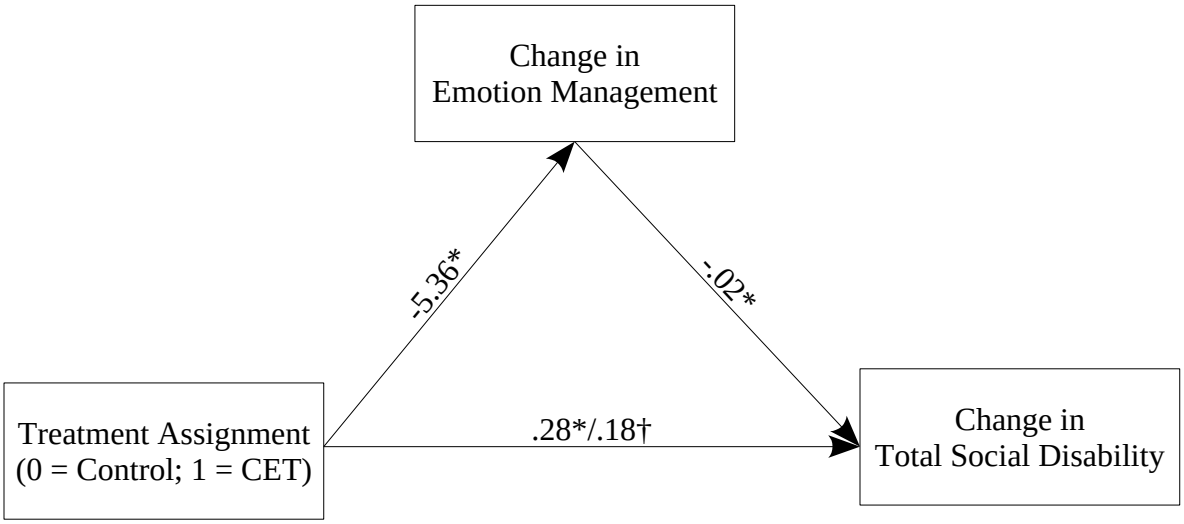
Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator.
CET = Cognitive Enhancement Therapy
†$p < .15$, *$p < .05$, two-tailed.

Figure A2.2. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotion Facilitation Among Participants Living with Significant Others



Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator.
CET = Cognitive Enhancement Therapy
†$p < .15$, *$p < .05$, two-tailed.

Figure A2.3. Mediating Effect of Treatment on Changes in Total Social Disability Through Changes in Emotion Management Among Participants Living with Significant Others



Note. Path parameters are unstandardized coefficients. Coefficients to the right of the forward slash (/) indicate effects after adjusting for the mediator.
CET = Cognitive Enhancement Therapy
†$p < .15$, *$p < .05$, two-tailed.

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