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Pathways to Social Functioning via Emotion Regulation in People with Serious Mental Illness

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PATHWAYS TO SOCIAL FUNCTIONING VIA EMOTION REGULATION IN PEOPLE WITH SERIOUS MENTAL ILLNESS

by

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Recent research on social cognitive deficits associated with serious mental illness (SMI) has demonstrated a range of emotion processing difficulties, from emotion perception to emotion regulation. Whereas emotion perception deficits are well documented in this population, little is understood about emotion regulation and the relationship of emotion regulation to other abilities and impairments.

Method. Participants included 41 individuals with SMI recruited from a day rehabilitation program. Assessments included a range of functional domains, including symptom severity, neurocognition, social cognition, emotion regulation, and social functioning.

Results. Emotion dysregulation was hypothesized to be associated with more severe positive symptoms, poorer neurocognitive functioning, and poorer social and community functioning. Results were mixed across the various assessments. There was some evidence of a relationship between psychiatric symptom severity and emotion dysregulation. However, global neurocognition explained very little of the variance in emotion regulation. Individuals with poorer emotion regulation tended to have poorer self-reported social functioning, and positive symptom severity accounted for some of the variance in this relationship. Path analysis modeling summarized these relationships.

Conclusions. This study demonstrates that emotion regulation explains variance in social functioning, but much remains to be understood about how emotion regulation relates to other biosystemic domains in this population. Limitations in valid assessment in the SMI population hinder ongoing progress, and this should be a key focus of future research, as orthogonal functional domains require individual attention in clinical assessment and research. Assessing segregated processes and investigating interactions among those processes reveal important relationships among subgroups of this population that would otherwise be missed. Though symptom severity and neurocognitive impairments have historically been the focus of treatment development for SMI, this work clearly demonstrates that individuals with SMI also have impaired emotion regulation abilities. Furthermore, that these abilities share an interactive relationship with social functioning and symptom severity reinforces their importance as potential treatment targets. Integrating emotion regulation skills training into the psychiatric rehabilitation toolkit is therefore a worthwhile future endeavor.

DEDICATION

To my family,

whose unconditional support

was the foundation of this entire endeavor.

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CHAPTER 1

INTRODUCTION

The biosystemic paradigm of psychopathology conceptualizes human functioning as a complex network of interrelated processes and mechanisms (Spaulding, Sullivan, & Poland, 2003). Those processes and mechanisms can usefully and heuristically be organized into five categorical levels of analysis (in order of most molecular to most molar): neurophysiological, neurocognitive, social cognitive, sociobehavioral, and socioenvironmental. Psychopathology is conceptualized as the result of abnormal or deficient operation of those processes and mechanisms across all levels as they interact to maintain stable functioning. Although the processes are causally linked throughout the biosystem, for the purposes of treatment and rehabilitation, they are independent enough to require separate, specific targeting. Biosystemic approaches to psychiatric rehabilitation therefore encourage research that addresses functioning at all the respective levels.

The last decade of research on schizophrenia has seen an expansion of focus from the neuropsychological level of analysis to the social cognitive level, in part because it complements biosystemic psychopathology and also because measures of social cognitive impairments appear to be more proximal than measures of neurocognitive impairments to real world behavior and to psychosocial treatment effects. A broad range of deficits in social cognition are well-documented in the serious mental illness (SMI) literature (Corrigan & Penn, 2001b; Penn, Sanna, & Roberts, 2008). Furthermore,

difficulty processing social information has wide-ranging functional implications, as normal processing is fundamental for social and occupational relationships, as well as general community functioning. The development of social cognitive paradigms for schizophrenia has primarily been guided by the analysis of cognitive processes thought to be essential to basic social functioning and found to be impaired in SMI. These include paradigms that range from molecular perceptual processes to more molar processes that neuropsychologists would consider to be in the executive domain.

On the more molecular end of the biosystemic spectrum, most research on the social cognitive deficits associated with SMI has focused on emotion processing. This research has shown that individuals with schizophrenia have a range of difficulties in processing emotions, from difficulties in perception of others' emotions to difficulties in expression and regulation of one's own emotions. The clinical significance of these deficits in emotion processing is demonstrated by their association with dimensions of adaptive social functioning (Couture, Penn, & Roberts, 2006).

The difficulties in emotion processing observed in SMI, particularly emotion regulation, and their relationship to other biosystemic and functional domains, are the focus of this dissertation. The following sections address, in turn, (1) the basic concepts of emotion perception and emotion regulation as they are used in contemporary research on schizophrenia; (2) the nature of impairment in emotional functioning in schizophrenia and other serious mental illness; and (3) treatment approaches that target emotional impairments. Analysis of previous findings and other background information indicates that further progress in assessment and treatment will require a better understanding of the relationships between emotion impairments and other aspects of serious mental

illness. The subsequent sections describe an empirical, quantitative analysis of those relationships.

CHAPTER 2

CONCEPTUAL FOUNDATIONS

Definitions

Social cognition. Social cognition is defined as "the processes and functions that allow a person to understand, act on, and benefit from the interpersonal world" (Corrigan & Penn, 2001a, p. 3). There are generally considered to be four domains of social cognition: (1) attributional style, or judging the causes of events; (2) theory of mind, or understanding the contents of others' minds; (3) social perception, or perceiving social cues in social situations; and (4) emotion perception, or recognizing emotional expressions from facial, vocal, and postural cues. Individuals with SMI have been shown to have deficits in each of these areas (Corrigan & Penn, 2001b; Feinberg, Rifkin, Schaffer, & Walker, 1986), pointing to a pervasive deficit in the social cognitive domain.

These deficits have functional implications, as each of these domains of social cognition is associated with functional dimensions such as social skills, social problem solving, and broad community functioning (Addington, Saeedi, & Addington, 2006; Brekke, Kay, Lee, & Green, 2005; Corrigan & Toomey, 1995; Ihnen, Penn, Corrigan, & Martin, 1998; Kee, Green, Mintz, & Brekke, 2003; Kim, Doop, Blake, & Park, 2005; Mathews & Barch, 2010; Mueser et al., 1996; Pinkham & Penn, 2006; Poole, Tobias, & Vinogradov, 2000). Thus, deficits in social cognition have both proximal and far-reaching consequences for daily functioning.

Emotion. Much of the research on social cognitive deficits in schizophrenia has focused on emotion processing. Indeed, emotional abnormalities have long been observed as one of the hallmark features of schizophrenia (American Psychiatric Association

[APA], 2000; Bleuler, 1950; Kraepelin, 1919). A discussion of these abnormalities must begin with a conceptualization of what emotions are and how they are normally processed.

Many researchers have theorized about what emotions are and how they are generated. The result has been many similar theories and considerable historical debate regarding a consensual definition of emotion (Bloch, Moran, & Kring, 2010; Kleinginna & Kleinginna, 1981; Solomon, 2010). Among these are such classic theories as the James-Lange, Cannon-Bard, and Schachter-Singer theories. William James (1884, 1890), one of the first psychologists, theorized about emotion. The James-Lange theory posits that an emotional event is perceived and causes physiological arousal; that physiological arousal is interpreted, and emotions occur as a result of that interpretation. This theory was later supplanted by the Cannon-Bard theory, which posits the opposite: an emotional event is perceived and simultaneously causes physiological arousal and emotion (Cannon, 1932). Still later, Schachter and Singer's (1962) two-factor theory was similar to the James-Lange theory but emphasized the role of cognition in the generation of emotion. This theory posits that an emotional event is perceived and causes physiological arousal; that arousal is given a cognitive label which accounts for the situational context.

Each of these classic theories emphasizes (1) the importance of the occurrence of an event, (2) perception of that event, (3) physiological arousal, and (4) emotional responses. Gross and Thompson (2009) have combined the salient factors of these and the many other theories of emotions into the modal model of emotion. The modal model reflects the core features of emotion emphasized across theories and represents a consensual understanding of how emotions are generated. Gross and Thompson (2009)

establish three core features of emotions. The first feature regards appraisal: Emotions arise when an individual attends to an event, perceives it, and appraises it for its meaning (including its personal relevance, familiarity, and valence). Thus, an emotion requires both attention to a stimulus and an appraisal of that stimulus as pertinent to one's goals. The second feature regards the changes that occur as a result of the first feature: Emotions are multi-faceted phenomena that involve systemic changes – in thoughts (subjective experience), behavior, and physiology. The final feature regards the flexibility of emotional responses: The systemic changes associated with emotions are rarely obligatory. It is this final feature which makes emotion regulation possible, as it is the flexibility of emotional responses that is regulated.

For the purposes of the present analysis, the modal model's emphasis on the typical features of emotions seems most pertinent to understanding what emotions are and how they can disrupt biosystemic processing when their regulation is dysfunctional, as in SMI. Such an understanding of how emotions are generated is foundational for the following discussion of how emotions can be regulated.

Emotion regulation. Gross and Thompson's (2009) definition of emotion thus stimulates a definition of emotion regulation: Emotion regulation is a heterogeneous set of processes that are automatic or controlled and influence emotions in oneself, others, or both. Thus, emotion regulation strategies influence how emotion is experienced – the quality, intensity, timing, and dynamic features of thoughts, behaviors, and physiology. This definition conceptualizes emotion regulation as the regulation *of* emotions, versus an alternative conceptualization of regulation *by* emotions. This second conceptualization refers to how emotions regulate thoughts, behaviors, physiology, or even other people.

The focus of the present analysis is to evaluate how systemic processes are themselves regulated, thus motivating the choice of a definition consistent with the former conceptualization.

This definition prompts discussion of the core features of emotion regulation. First, both positive and negative emotions can be regulated, and they can either be increased or decreased (Gross & Thompson, 2009). The focus of the majority of the research on emotion regulation has been on how individuals decrease negative emotions or increase positive emotions. However, such a focus ignores strategies that increase negative emotions or decrease positive emotions – strategies that might be associated with psychopathology. The question arises: Does a pathological excess of negative emotion arise from underutilization of strategies associated with positive emotion changes (i.e., decreasing negative emotions, increasing positive emotions), for example, or alternatively, from overuse of strategies associated with negative emotion changes (i.e., increasing negative emotions, decreasing positive emotions)? This question is as yet unanswered.

A second feature of emotion regulation is that regulation can occur either consciously or unconsciously (Gross & Thompson, 2009). Thus, emotions can be deliberately changed, or they can be regulated automatically, via habitual processes. For example, a reaction of disappointment to the receipt of an unsatisfactory gift might be deliberately hidden (or suppressed) initially; but this reaction may become automatic if repeated over time. Importantly, it is unclear whether the distinction between conscious and unconscious regulation is dimensional (i.e., continuous) or categorical (Gross & Thompson, 2009). That is, it is possible that emotion regulation may have some

characteristics of both. Moreover, it is possible that emotion regulation may be a *multi*dimensional phenomenon with categorical characteristics.

Third, no strategy of regulating emotions is inherently adaptive or maladaptive (Gross & Thompson, 2009). Consideration of the context in which the emotion is to be regulated is always key in an evaluation of the adaptive nature of a strategy (Gross, 1998; Thompson & Calkins, 1996). For example, whereas habitual suppression of emotional reactions is generally associated with poor functional outcomes (reviewed below), adopting this strategy in a situation such as receiving an unsatisfactory gift may be the most adaptive response for social functioning.

Finally, emotion regulation involves changes in how emotional responses are coordinated (Gross & John, 2003; John & Gross, 2004). As discussed above, emotional responses entail systemic changes – coordinated and interrelated changes in thoughts (subjective experience), behavior, and physiology. Various strategies of regulating emotions are associated with differences in how and when these components of emotional responses are changed.

Emotion Regulation Strategies

Once an emotional stimulus is perceived and appraised, many strategies exist for changing the type, timing, experience, or expression of emotions (Gross, 1998). These strategies can be organized along several dimensions. One dimension separates strategies into those that are internal (e.g., suppressing disappointment) versus external (e.g., taking a walk to defuse anger; Thompson, 1994). Another dimension focuses on the resources used to regulate emotions by separating strategies into those that focus on modifying cognitions (e.g., reinterpreting a loved one's death as the end of suffering) versus

behaviors (e.g., seeking social support) versus situations (e.g., hiding controversial artwork from conservative visitors; Eisenberg et al., 1995). A final dimension focuses on when various strategies impact the emotion generative process by separating strategies into those that attempt to change the cause of the emotion (antecedent-focused strategies; e.g., avoiding offensive acquaintances) versus the response to the emotion (response-focused strategies; e.g., engaging in aerobic exercise to decrease physiological and experiential components of emotion; Gross & Muñoz, 1995). This dissertation will focus on the antecedent- versus response-focused dimension, as it appears to be the most influential in the literature and the most useful for understanding the abnormalities in the use of these particular strategies in SMI.[1]

The modal model of emotion implicates areas in the emotion generative process where regulation might occur: changing the situation, attention, appraisal, or response. Antecedent- and response-focused strategies vary in the time at which they occur in the emotion generative process (Gross, 2001; Gross & Muñoz, 1995). In the modal model of emotion, antecedent-focused strategies occur before appraisals and can affect the situation, attention, or appraisal. These changes thereby alter the emotional response (Gross, 2002). In contrast, response-focused strategies occur after appraisals and attempt to change an emotional response that has already begun to occur (Gross, 2002).

Antecedent-focused strategies. Antecedent-focused strategies are grouped into four families: situation selection, situation modification, attentional deployment, and cognitive change (Gross, 2002).

Situation selection and situation modification make it more or less likely that an emotional situation will occur that leads to desirable or undesirable emotions. They serve

to alter an emotional situation that has already occurred to modify the emotional impact. The "situations" targeted by situation selection and situation modification strategies can be either internal (e.g., thoughts) or external to the individual.

Attentional deployment strategies are used to select which aspect of a situation is attended to, and they are typically used when it is impossible to use situation selection or situation modification strategies. Common examples of attentional deployment are distraction and concentration. With distraction, attention is deployed to a less emotionally-arousing aspect of the situation or away from the situation; with concentration, attention is deployed to the situation. Rumination, common to many forms of psychopathology, is an example of concentration (Gross & Thompson, 2009).

Finally, cognitive change strategies alter the meaning attached to an emotional event. An extensively-researched example of cognitive change is reappraisal (Gross, 2002; John & Gross, 2009; Ochsner & Gross, 2009), a strategy in which the meaning of a situation is interpreted in such a way as to alter the emotional response.

Response-focused strategies. There is only one family of response-focused strategies, called response modulation (Gross, 2002).

Response modulation strategies attempt to change emotional responses once they have already been elicited. Common examples include the use of drugs and alcohol (Morris & Reilly, 1987), exercise (Thayer, Newman, & McClain, 1994), relaxation (Borkovec & Costello, 1993), and suppression. Suppression refers to the hiding or avoidance of an emotional expression and has been widely researched as an emotion regulation strategy (Beevers, Wenzlaff, Hayes, & Scott, 1999; Purdon, 1999; Richards & Gross, 2006; Salters-Pedneault, Steenkamp, & Litz, 2010).

Reappraisal and suppression. Reappraisal, an antecedent-focused strategy that can alter an emotional response before it has been initiated, and suppression, a response-focused strategy that alters an emotional response that has already begun, have different affective, cognitive, and social consequences.

Affective consequences. Individuals who tend to use reappraisal as an emotion regulation strategy report fewer symptoms of depression, more satisfaction with their lives, higher self-esteem, higher levels of optimism, and higher levels of general well-being, versus individuals who tend to use suppression (Gross & John, 2002, 2003). Moreover, individuals who use reappraisal are better able to regulate negative moods than are individuals who use suppression.

This may be, in part, because reappraisal tends to be effective at decreasing the *experience* of negative emotions, whereas suppression tends to have no effect (Gross, 1998, 2002; Gross & John, 2002, 2003). Both strategies are effective at decreasing behavioral expressions of disgust (Gross, 1998, 2002); however, suppression increases sympathetic nervous system activation, whereas reappraisal does not (Gross, 2002). That is, individuals who habitually suppress negative emotions continue to experience the negative emotions at at least a physiological level; in contrast, individuals who use reappraisal may actually experience less negative emotion.

Cognitive consequences. Suppression appears to require more cognitive resources, thus using resources typically allocated towards other processing. The result is impaired verbal memory, including memory for social information such as peoples' names (Gross & John, 2002; Richards & Gross, 2000). No memory impairment is observed when individuals use reappraisal to manage emotions (Gross & John, 2002;

Richards & Gross, 2000). Given the pervasive cognitive deficits associated with SMI, the effect of emotion regulation strategies on cognitive resources is especially relevant.

Social consequences. Consistent with these differences in affective and cognitive consequences are differences between reappraisal and suppression in social consequences. Individuals who reappraise have more social support, both emotionally and instrumentally; and they are more likely to seek out social support than individuals who use suppression (Gross & John, 2002). Individuals who reappraise are more likely to share their emotions with others (Gross & John, 2002, 2003). Consequently, they are better liked by their peers (Gross & John, 2002, 2003). Individuals who suppress tend to avoid the attachment and intimacy associated with close relationships (Gross & John, 2003). Suppression even appears to have negative consequences on a conversational partner – interacting with a person who suppresses is associated with increases in blood pressure, whereas there appears to be no impact on blood pressure when interacting with a person who reappraises (Butler et al., 2003). The result of these social consequences is that individuals who habitually reappraise tend to have improved interpersonal functioning relative to individuals who suppress (John & Gross, 2004).

Neural Correlates of Emotion Processing

The previous discussion has focused on the social cognitive domain and its relationship to the neuropsychological and sociobehavioral domains. But in a biosystemic paradigm, abnormalities potentially extend to more molecular levels of organismic functioning, including neurophysiology. The human brain is designed for processing social and emotional information and contains neural circuits specifically designed for those purposes.

Emotional appraisal system. Across electrophysiological, neuropsychological, and functional neuroimaging studies, the amygdala, insula, striatum, and medial orbitofrontal cortex are consistently implicated in emotion processing (Bush, Luu, & Posner, 2000; Davidson & Irwin, 1999; Davidson, Jackson, & Kalin, 2000; Denny, Silvers, & Ochsner, 2010), with activations during rises in emotion and attenuations during reductions in emotion. Although individual experiments have implicated additional areas, the contribution of the above regions has been repeatedly documented (Brunet-Gouet & Decety, 2006).

The amygdala is broadly implicated in detecting the emotional salience of a stimulus and generating physiological reactions to emotional events (Adolphs, 2002, 2003; Aleman & Kahn, 2005; Denny et al., 2010; LeDoux, 2000; McDonald, 1998; van Rijn, Aleman, & Kahn, 2005). Moreover, it appears to have particular importance in the perception of others' emotional states, especially perceptions of basic fear and sadness (Adolphs, 2002, 2003; Calder, Lawrence, & Young, 2001). Although each of the areas in this emotion processing network is activated for all types of emotions, the insula and striatum (and, in general, the basal ganglia structures) are specifically implicated in the evaluation of potentially distressing and aversive stimuli (Scott, Heltzeg, Koeppe, Stohler, & Zubetla, 2006; Straube & Miltner, 2011). Finally, the medial orbitofrontal cortex is implicated in evaluating the affective valence of a stimulus and its contextual meaning (Davidson & Irwin, 1999).

Emotion regulation system. Attempts to regulate emotions require modulation of the emotional appraisal system. Specifically, across studies, reappraisal consistently invokes a network including the lateral prefrontal cortex, medial prefrontal cortex, dorsal

anterior cingulate cortex, and lateral orbitofrontal cortex (Denny et al., 2010). The prefrontal regions are the areas most tied to reappraisal and suppression, as they are associated with developing alternative ways of conceptualizing an emotional situation (Ochsner & Gross, 2005, 2009; Ochsner et al., 2004). Moreover, these areas are implicated in general cognitive control of emotion (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001; Banich et al., 2009; Green & Malhi, 2006; Ochsner & Gross, 2005) and development of adaptive responses to shifting social situations. The prefrontal cortex and anterior cingulate cortex appear to serve an inhibitory role in modulating activity in the emotional appraisal systems reviewed above. Increased activity in these areas is correlated with decreased activity in subcortical structures such as the amygdala. As no direct connections exist between the lateral and dorsal regions of the prefrontal cortex and the amygdala, the medial prefrontal cortex may serve as the link between cognitive control of emotion and emotional processing (Green & Malhi, 2006). In general, these structures serve a top-down interpretive function for emotional stimuli being processed in subcortical structures in a reciprocal, interactive manner (Denny et al., 2010; Ochsner & Gross, 2005).

CHAPTER 3

EMOTIONAL ABNORMALITIES IN SMI

The foregoing discussion summarized the basic concepts pertinent to researching emotion processing in SMI and serves as a foundation for discussing the nature of the impairment in emotion functioning in SMI. Individuals with SMI demonstrate abnormalities in all aspects of emotion processing – from perception of others' emotions to experience and regulation of their own emotions.

Emotion Perception

Individuals with schizophrenia have a general deficit identifying the correct emotions associated with facial expressions (Feinberg et al., 1986; Novic, Daniel, & Perline, 1984; Salem, Kring, & Kerr, 1996; Schneider, Gur, Gur, & Shtasel, 1995; Walker, McGuire, & Bettes, 1984). Whereas psychiatrically healthy individuals identify an average of 71% of emotional facial expressions correctly, individuals with schizophrenia accurately identify just 64% (Kohler et al., 2003).

Several studies have attempted to determine the specific features associated with this emotion perception deficit. Individuals with schizophrenia commit more errors when identifying negative facial expressions than when identifying positive facial expressions (Borod, Martin, Alpert, Brozgold, & Welkowitz, 1993; Muzekari & Bates, 1977; Van't Wout et al., 2007; Zuroff & Colussy, 1986). Specifically, individuals with schizophrenia appear to be impaired in the recognition of negative emotions such as fear, anger, shame, and disgust (Dougherty, Bartlett, & Izard, 1974; Kohler et al., 2003; Mandal & Palchoudhury, 1985) but not positive emotions such as joy and happiness (Dougherty et al., 1974; Kohler et al., 2003).

Accompanying this better recognition of positive versus negative emotions is increased efficiency in processing positive emotions in schizophrenia (Silver, Bilker, & Goodman, 2009). Happy facial expressions are not only processed more accurately in schizophrenia, but they are also processed more rapidly and efficiently than sad facial expressions. Nevertheless, individuals with schizophrenia process emotional expressions more slowly and less efficiently than healthy controls. Therefore, it appears that recognizing emotional facial expressions takes longer and is less productive in schizophrenia than in comparison participants.

Of note, individuals with schizophrenia also appear to be impaired at recognizing neutral facial expressions. One study found that they accurately identified 70% of neutral facial expressions, compared to 86% correctly identified by comparison participants (Kohler et al., 2003). Both groups most often misidentified neutral expressions as happy or sad. However, of the neutral expressions misidentified by individuals with schizophrenia, 23% were mislabeled as disgust, versus 5% in the comparison group. Thus, individuals with schizophrenia appear to have a negative bias in their emotion perception. Not only are individuals with schizophrenia impaired when asked to identify neutral facial *expressions*, they are also impaired when asked to identify neutral *faces* (i.e., indicate whether they have seen the face before; Silver et al., 2009). Thus, individuals with schizophrenia are impaired in processing neutral faces, regardless of whether they are evaluating its emotional content or its identity; but they are more impaired when their task involves emotional processing.

Furthermore, whereas increasing the intensity of a facial expression aids control participants' identification of the emotion, individuals with schizophrenia benefit

significantly less from this compensation (Kohler et al., 2003). That is, they are impaired at recognizing mild emotional expressions (correctly identifying 5% fewer expressions than controls) but even more impaired at recognizing extreme emotional expressions (correctly identifying 7% fewer expressions than controls). Interestingly, this suggests that increasing the intensity of emotions is unlikely to aid emotion perception in schizophrenia.

Consistent with these reported difficulties in facial emotion perception, individuals with schizophrenia also experience difficulty identifying emotions expressed in voices (Hooker & Park, 2002; Vaskinn et al., 2007). In affective prosody tasks, they have more difficulty identifying negative emotions, with a particular impairment for identifying sadness (Bozikas et al., 2006) and fear (Edwards, Pattison, Jackson, & Wales, 2001). This is the same pattern observed in visual emotion tasks. However, this is in contrast to what is observed in healthy controls, wherein negative emotions are easier to perceive in voices than positive emotions (see Edwards, Jackson, & Pattison, 2002, for review). Interestingly, this impairment is also present in individuals with schizotypic personality traits (Phillips & Seidman, 2008). Schizotypal Personality Disorder is considered an attenuated form of schizophrenia, representing a premorbid stage of the disorder (Raine, 2006). Therefore, that impairment recognizing emotions in vocal prosody is identifiable in schizotypy suggests that this difficulty may be fundamental to schizophrenia.

Integrated emotion perception tasks allow participants to benefit from both visual and vocal affective cues. These more ecologically valid tasks more closely approximate actual stimuli encountered in daily life. In a videotaped emotion

perception task with just video (i.e., no audio), individuals with schizophrenia have more difficulty identifying happy emotions than controls; however, they perform as well as controls when identifying sad or angry emotions (Bellack, Blanchard, & Mueser, 1996). This more accurate perception of positive versus negative emotions parallels the results reported above with static visual stimuli. When audio is added to the video, individuals with schizophrenia have more difficulty identifying sad emotions than controls; however, they perform as well as controls when identifying happy or angry emotions. This suggests that the vocal cues help individuals with schizophrenia identify happy and angry emotions. However, integrated cues do not appear to help their perception of sad emotions.

Emotion Paradox

As discussed previously, similar neural systems are implicated in emotional expression and emotion perception (Brunet-Gouet & Decety, 2006). As emotion perception is abnormal in this population, it follows, then, that emotional expression, and possibly emotional experience, may also show abnormalities.

Schizophrenia has been characterized by flat affect since its very conceptualization (Bleuler, 1950; Kraepelin, 1919), and indeed flat and inappropriate affect continue to be considered hallmark characteristics of the disorder (APA, 2000). However, individuals with schizophrenia report emotional experiences consistent with the levels reported by psychiatrically healthy individuals. This discrepancy between the reduced expression but self-reported normal experience of emotion in schizophrenia has been termed the “emotion paradox” (Aleman & Khan, 2005).

Emotional experience. Some research reports that individuals with schizophrenia demonstrate reduced experience of positive affect (Livingstone, Harper, & Gillanders, 2009; Reske et al., 2007; Schneider et al., 1995; Suslow, Roestel, Ohrmann, & Arolt, 2003) and increased experience of negative affect (Livingstone et al., 2009; Suslow et al., 2003). However, other studies have found that these individuals self-report similar levels of emotional experience as psychiatrically healthy controls in the laboratory (Gur, et al., 2006; Kring, Kerr, & Earnst, 1999; Kring & Neale, 1996). This seems at odds with the increased reporting of anhedonia, defined as the diminished capacity to experience pleasure, in this population (Horan, Kring, & Blanchard, 2006; Kirkpatrick, Fenton, Carpenter, & Marder, 2006; Phillips & Seidman, 2008).

The resolution of the discrepancy between normal self-reports of emotional experience and increased self-reports of anhedonia may lie in the affective valence being assessed. Individuals with schizophrenia report experiencing significantly more negative affect and significantly less positive affect in their daily lives than healthy controls (Myin-Germeys, Delespaul, & Van Os, 2005). This diminished daily experience of positive affect supports their increased reporting of anhedonia but may in part be related to biased expectations, whereby they avoid engaging in rewarding activities because they do not expect to derive pleasure from them (Watson & Naragon-Gainey, 2010). Nevertheless, it appears that, compared to controls, individuals with schizophrenia spend a significantly greater proportion of their days experiencing negative emotions and a significantly lesser proportion of their days experiencing positive emotions. Overall, the research indicates that, on average, individuals with schizophrenia are able to experience emotions to a similar degree as normal controls,

but their individual experience clearly differs from that of psychiatrically healthy controls.

These experience sampling findings are supported by findings from a mood induction task, in which individuals with schizophrenia became less happy in response to happy induction and less sad in response to sad induction than controls (Schneider et al., 1995). This suggests that they have an attenuated emotional experience. Interestingly, they are also sadder during happy induction and happier during sad induction than controls, indicating some degree of inappropriate emotion induction in schizophrenia.

In addition to these behavioral findings are physiological findings of abnormal emotional experience. Individuals with schizophrenia demonstrate greater skin conductance than normal controls in response to affective stimuli (Kring & Neale, 1996). This suggests that these individuals are experiencing heightened physiological arousal and is in contrast with the idea that they have a diminished capacity to experience emotions. Neurologically, individuals with schizophrenia exhibit reduced activation in emotion processing circuitry (right amygdala, left orbitofrontal cortex, fusiform gyrus, cuneus) in response to non-aversive (though still affectively arousing) stimuli and near normal to above normal activation in emotion processing circuitry (medial prefrontal cortex) in response to aversive stimuli (Taylor, Liberzon, Decker, & Koeppe, 2002). These physiological and neurological data indicate that emotion processing may be somewhat disrupted in schizophrenia, which may affect the experience of emotion.

Emotional expression. The crux of the emotion paradox is that although emotional *experience* appears to be within normal limits, emotional *expression* appears to be abnormal in schizophrenia. Affective flattening, the restriction in the range of emotional expression in response to emotional stimuli, is a characteristic symptom of schizophrenia (APA, 2000; Kring & Neale, 1996). Thus, individuals with schizophrenia appear to have a disjunction between their near-normal emotional experience and their flattened expression of this experience.

In daily life, individuals with schizophrenia are overall less facially expressive than psychiatrically healthy controls (Berenbaum & Oltmanns, 1992; Kring & Neale, 1996; Mandal, Pandey, & Prasad, 1998; Martin, Borod, Alpert, Brozgold, & Welkowitz, 1990; Schneider et al., 1995; Tremeau et al., 2005). Specifically, they spontaneously smile less often and for a shorter period of time. They also use fewer facial coverbal gestures (facial or head movements intended to illustrate or replace speech) and use fewer words in emotional conversation (Gottheil, Paredes, Exline, & Winkelmayer, 1970; Tremeau et al., 2005). Further, of universally recognized emotions, they appear to have the greatest impairment expressing happiness verbally (Gottheil et al., 1970). Interestingly, overall facial expressiveness and the number of facial coverbal gestures have been found to depend on the number of words used in this population (Tremeau et al., 2005). This again highlights the interaction between neurocognition [here, specifically alogia, another common symptom of schizophrenia (APA, 2000)] and social cognition (here, specifically emotional expression).

Individuals with schizophrenia demonstrate reduced spontaneous, involuntary facial activity in response to a variety of affective stimuli in the laboratory (Gaebel &

Wölwer, 1992; Martin et al., 1990). Research indicates that, like healthy controls, individuals with schizophrenia make more positive facial expressions in response to positive film clips than negative facial expressions in response to negative film clips (Kring & Neale, 1996). However, individuals with schizophrenia make fewer positive facial expressions in response to positive film clips than normal controls; moreover, they make fewer negative facial expressions in response to negative film clips than controls (Kring & Neale, 1996). Further, they appear to have the greatest impairment expressing anger (Gottheil et al., 1970). Thus, individuals with schizophrenia appear to make expressions in response to affective stimuli but to an attenuated degree.

Although they make fewer facial expressions, individuals with schizophrenia do appear to exhibit activity in the facial muscles associated with appropriate facial reactions to affective stimuli (Earnst et al., 1996; Kring et al., 1999). That is, like control participants, individuals with schizophrenia show greater activity in the muscles associated with frowning in response to negative pictures than in response to positive pictures (Kring et al., 1999). Moreover, individuals with schizophrenia tend to show the same pattern of facial responsivity in the muscles associated with frowning and smiling in response to happy, sad, fearful, and angry stimuli (Kring et al., 1999). Thus, it appears that individuals with schizophrenia are expressing appropriate facial expressions but to such an attenuated degree that they are not perceived.

As mentioned, individuals with schizophrenia tend to have reduced involuntary emotional expressiveness, especially for positive emotions (Martin et al., 1990). Additionally, individuals with schizophrenia demonstrate reduced facial activity when voluntarily attempting to mimic a facial expression (Gaebel & Wölwer, 1992; Tremeau

et al., 2005). Moreover, they are less accurate at generating facial expressions (Gottheil, Thornton, & Exline, 1976), both by verbal instruction and imitation (Borod et al., 1990; Schwartz, Mastropaolo, Rosse, Mathis, & Deutsch, 2006). When asked to imitate an emotional facial expression, individuals with schizophrenia show significantly worse performance than controls (Gaebel & Wölwer, 1992). They have the most pronounced deficit when attempting to imitate fearful, sad, and angry expressions (Gaebel & Wölwer, 1992; Tremeau et al., 2005); they have the least pronounced deficit when attempting to imitate happy and surprised expressions (Tremeau et al., 2005). Not only are individuals with schizophrenia less accurate at imitating facial expressions, they also give fewer emotional expressions on command and spend less time expressing those emotions (Tremeau et al., 2005). Similarly, they also demonstrate reduced facial activity when attempting to simulate emotions when the stimulus is an emotional word (e.g., fear) rather than an emotional facial expression (Gaebel & Wölwer, 1992; Tremeau et al., 2005). Therefore, it appears that they not only experience difficulty expressing emotions spontaneously, but they also experience difficulty expressing emotions voluntarily.

These abnormalities of emotion expression in schizophrenia may be influenced by emotion perception deficits (Sachs, Steger-Wuchse, Kryspin-Exner, Gur, & Katschnig, 2004). Individuals with poorer performance on emotion perception measures tend to have greater affective flattening (Kohler et al., 2003). However, other research indicates that these processes are independent (Kring & Neale, 1996; Silver & Shlomo, 2001). For example, emotion perception performance was significantly positively correlated with negative symptoms such as affective flattening in one study, but this

correlation was no longer significant when age, length of illness, accumulated time in the hospital, or education were added as a covariate (Silver, Shlomo, Turner, & Gur, 2002). This suggests that the relationship between emotion perception and emotional expression may be complicated and deserving of further empirical attention.

Emotion Regulation

This emotion paradox, the disjunction between the experience and the expression of emotion in schizophrenia, may reflect difficulties in emotion regulation. That is, the impaired emotion perception, irregular emotional experience, and abnormal emotional expression observed in this population may be a result of poor skills with emotion regulation strategies such as reappraisal or suppression.

Indeed, individuals with schizophrenia demonstrate maladaptive coping with stress (Livingstone et al., 2009), and their chosen methods to regulate distress tend to be avoidant (e.g., suppression; van den Bosch, van Asma, Rombouts, & Louwerens, 1992). This preference for avoidant strategies like suppression is in contrast to the preference for reappraisal made by psychiatrically healthy individuals (Livingstone et al., 2009; van der Meer, van't Wout, & Aleman, 2009). Frequent use of suppression may help explain the emotion paradox, wherein suppression leads to an expression of flat affect that is incongruent with the emotional experience. Suppression may also help explain the finding of increased skin conductance in this population (van der Meer et al., 2009), as suppression leads to greater physiological reactivity than reappraisal. Thus, the choice of regulatory strategy may mediate the relationship between emotional experience and emotional expression.

Some research indicates that individuals with schizophrenia are able to suppress their feelings in response to emotional film clips but have difficulty amplifying their feelings (Henry et al., 2007). This suggests that it is easier for them to try to down-regulate than to up-regulate their emotional experience. Moreover, it suggests that individuals with schizophrenia not only have a skill deficit (i.e., they do not have the skills to increase positive experiences) but also maladaptive use of intact skills (i.e., overreliance on suppression to decrease negative experiences; Kring & Werner, 2004). Interestingly, the film clips in this cited study were intended to elicit amusement. The relative ease with which individuals with schizophrenia were able to adopt the emotion regulation strategy of suppression to down-regulate positive feelings may relate to their increased reporting of anhedonia whereby they report diminished levels of pleasure in their emotional experience.

Further, research indicates that affective flattening, an abnormality in emotional expression, may be a result of poor use of emotional suppression (Ellgring & Smith, 1998). In other words, it has been argued that individuals presenting with flat affect may overuse emotional suppression as a regulatory strategy such that they do not appropriately connect their emotional expression with their emotional experience. The clinical significance of this poor emotion regulation is shown via the variety of negative consequences associated with persistent emotional suppression, including impairing memory, disrupting communication, inhibiting relationship formation, and reducing rapport (Butler et al., 2003; John & Gross, 2004). Thus, the tendency to overuse the regulatory strategy of emotional suppression can not only lead to affective flattening but can also have negative social consequences for individuals with schizophrenia.

The neural circuitry associated with emotion processing also shows abnormalities in schizophrenia (Aleman & Khan, 2005; Brunet-Gouet & Decety, 2006). Generally, reduced activity in the appraisal regions (amygdala, insula, striatum, and medial orbitofrontal cortex) is associated with impairments in perception of and response to emotional stimuli in this population (Brunet-Gouet & Decety, 2006; Denny et al., 2010). However, the evidence supporting hypoactivations in these areas is equivocal (Brunet-Gouet & Decety, 2006), as some studies report hyperactivations in these regions, particularly the amygdala (Holt et al., 2006; Kosaka et al., 2002). The interpretation of these data that is perhaps most consistent with all current reports is that serious mental illness involves impairments in emotion appraisal at multiple levels of biosystemic functioning, including the neurophysiological and neuropsychological levels.

This dysregulation may be due, at least in part, to disrupted connections between the appraisal regions and the cognitive control (i.e., regulation) regions (Das et al., 2007). This is apparent even in individuals who are at risk for developing psychosis (Modinos, Ormel, & Aleman, 2010), indicating that it may be a marker of vulnerability for the disorder.

CHAPTER 4

INTERVENTIONS TARGETING SOCIAL COGNITIVE DEFICITS

The neuropathology associated with schizophrenia and the severity of the associated social cognitive deficits brings into question whether the emotion processing deficits associated with that pathology can be remediated. Several interventions have been developed to target the various systemic deficits associated with SMI. Although these treatments are far from returning individuals with SMI to "normal" functioning, preliminary data do indicate that these deficits are responsive to therapeutic remediation.

Interventions Based on the Generalized Deficit Hypothesis

To the degree that functional neurocognition is a prerequisite for success at any more molar level of processing (e.g., social cognition), strengthening basic neurocognition may lead to improvements in social cognition (Spaulding & Poland, 2001).[2] This is reflected in the *generalized deficit hypothesis*, that social cognitive deficits such as the poor emotion processing observed in schizophrenia are due primarily to cognitive impairment in general, not the failure of some specific cognitive or neurocognitive subsystem (Archer, Hay, & Young, 1992; Huang, Xu, & Chan, 2011; Johnston, Katsikitis, & Carr, 2001; Kerr & Neale, 1993; Mueser et al., 1996; Pomarol-Clotet et al., 2010; Salem et al., 1996). This hypothesis also presumes that remediating cognitive impairment should likewise remediate emotion processing deficits (van der Gaag, Kern, van den Bosch, & Liberman, 2002).

Indeed, cognitive remediation, a treatment program aimed at improving executive functioning deficits, is associated with improvements in social cognition. Cognitive remediation consists of exercises of varying cognitive complexity, from basic perception

to social perception. Although the goal of the program is to target social perception, neurocognitive skills such as attention, memory, and executive functioning are also targeted, as these skills are considered fundamental for improved social perception (van der Gaag et al., 2002). These areas are targeted with training in such strategies as self-instruction, memory enhancement, inductive reasoning, and compensatory training procedures, strategies which have proven successful in remediating other types of deficits in schizophrenia (Kern, Wallace, Hellman, Womack, & Green, 1996).

Consistent with its goal of remediating neurocognitive deficits, cognitive remediation is associated with improvements in verbal and visual memory, sustained attention, and executive functioning in schizophrenia (Hodge et al., 2010; Reeder, Newton, Frangou, & Wykes, 2004). Furthermore, it is associated with improvements in emotion perception in this population. Although individuals with schizophrenia commit 35-55% more errors in emotion tasks than healthy controls before treatment, their performance approaches that of untreated healthy controls following treatment (van der Gaag et al., 2002).

Finally, cognitive remediation is associated with improvements in social and occupational outcomes, which appear to persist in at least the short-term beyond the conclusion of treatment (Hodge et al., 2010). Importantly, it has been hypothesized that cognitive remediation serves as a mediator between changes in neurocognition and social functioning; without cognitive remediation, changes in neurocognition do not appear to affect social functioning (Reeder et al., 2004).

A more comprehensive cognitive treatment modality is Integrated Psychological Therapy (IPT; Brenner, Hodel, Roder, & Corrigan, 1992; Roder, Mueller, Brenner, &

Spaulding, 2010). Like cognitive remediation, IPT is founded upon the assumption that both neurocognitive and social cognitive deficits need to be addressed therapeutically to effect change in social cognition (Brenner et al., 1992; Wallace et al., 1980). Also like cognitive remediation, IPT consists of exercises of varying cognitive complexity, from basic perception to social perception. These exercises are arranged in five hierarchical subprograms: cognitive differentiation, social perception, verbal communication, social skills, and interpersonal problem solving. With respect to neurocognitive deficits, IPT is associated with improvements in attention, concept formation, and abstract thinking, as well as in spatiotemporal orientation and memory (see Roder, Mueller, Mueser, & Brenner, 2006, for a review). IPT is also associated with improvements in social functioning and social competence (Brenner et al., 1992; Roder et al., 2006; Spaulding, Reed, Sullivan, Richardson, & Weiler, 1999; Zimmer, Duncan, Laitano, Ferreira, & Belmonte-de-Abreu, 2007).

Interventions Based on the Specific Deficit Hypothesis

Although the generalized deficit hypothesis proposes that deficits in emotion processing in schizophrenia may be due to general cognitive impairment, its competitor, the *specific deficit hypothesis*, proposes that these deficits may be due to a specific deficit in emotion processing that is independent of general cognitive impairment (Borod et al., 1993; Edwards et al., 2001; Gaebel & Wölwer, 1992; Heimberg, Gur, Erwin, Shtasel, & Gur, 1992; Kosmidis et al., 2007; Poole et al., 2000; Shaw et al., 1999). Thus, the specific deficit hypothesis presumes that remediating emotion processing deficits will require a targeted intervention, as cognitive remediation may only result in slight

improvements in emotion processing (Bryson, Bell, & Lysaker, 1997; Wölwer et al., 2005).

Several treatments have been developed aimed specifically at remediating emotion processing deficits. Penn and Combs (2000) randomly assigned inpatients with schizophrenia to one of four interventions to compare effects on ability to identify facial affect: repeated practice with facial emotion identification, monetary reinforcement for correct identifications, imitation of facial emotions (facial feedback), and a combination of monetary reinforcement and facial feedback. The latter three groups resulted in improvements in facial affect identification to a degree that was comparable to performance of untreated healthy controls (Penn & Combs, 2000). Consistent with these results, two other imitation-based modalities have benefits for improving emotion recognition, the accuracy of facial expressions, and social and behavioral functioning in schizophrenia (Mazza et al., 2010; Schwartz et al., 2006).

The Micro-Expression Training Tool (METT; Russell, Chu, & Phillips, 2006) is a single-session computer-based intervention that attempts to improve emotion recognition. The program retrains where participants visually attend to faces, and pilot data indicate that it improves emotion recognition in outpatients with schizophrenia to a level comparable to untreated healthy controls.

Training of Affect Recognition (TAR; Frommann, Streit, & Wölwer, 2003) is a modality similar to cognitive remediation and IPT in its emphasis on errorless learning strategies such as compensation and positive reinforcement. Results indicate that this program is associated with significant improvements in facial affect recognition and

discrimination, to a degree comparable to that of untreated healthy controls (Frommann et al., 2003; Habel et al., 2010; Wölwer et al., 2005).

Emotion-Focused Therapy (Greenberg, 2004; Greenberg & Bolger, 2001), Dialectical Behavior Therapy (DBT; Linehan, 1993), and Emotional Management Training (Hodel & Brenner, 2002) are perhaps the treatments most aimed at developing emotion regulation skills. These modalities are designed to develop an understanding of adaptive and maladaptive emotions and their sources and then acquire emotion regulation skills. Emotion-Focused Therapy and DBT were not developed for schizophrenia, but the therapeutic goals and skills of both modalities are consistent with deficits observed in schizophrenia. Thus, these modalities may be useful for helping individuals with schizophrenia develop more adaptive emotion regulation skills (van der Meer et al., 2009). In contrast, Emotional Management Training, a subprogram of IPT, was designed specifically for emotion processing deficits observed in schizophrenia (Hodel & Brenner, 1997). This modality leads to improvements in emotional information processing and cognitive processing in this population, beyond what is observed for other common treatments (Hodel & Brenner, 2002).

Finally, Social Cognition and Interaction Training (SCIT; Penn, Roberts, Combs, & Sterne, 2007) is a group-based treatment modality aimed specifically at remediating social cognitive deficits in schizophrenia. It targets the three key social cognitive deficits in this population: emotion perception, attributional style, and theory of mind. It appears to be effective in inpatients (Combs et al., 2007; Penn et al., 2007) and outpatients (Roberts & Penn, 2009; Kleinlein, 2010) at remediating deficits in emotion processing and improving social functioning.

CHAPTER 5

PRESENT STUDY

This review has evaluated the abnormalities in emotion processing observed in schizophrenia. The research on emotion perception clearly demonstrates a deficit in that domain: Individuals with schizophrenia are impaired at recognizing the emotional expressions in faces, voices, and integrated tasks. However, the research for emotional experience and expression are substantially less clear. Although individuals with schizophrenia self-report similar emotional experiences to controls, physiological and experience sampling data indicate their actual experiences may conflict with these reports. This discrepancy points to the possibility that individuals with schizophrenia may be using emotion regulation strategies to alter their perception of their emotional experiences, thereby perceiving their abnormal experience as near normal. Finally, although individuals with schizophrenia frequently present with flattened affect, data indicate subtle movement in facial regions associated with appropriate emotional expressions. This again raises the possibility that emotion regulation strategies, such as suppression, may help explain this observation.

This study further elucidates the relationships between emotion regulation, biosystemic domains, and treatment modalities in SMI. The importance of understanding social cognitive functioning, and particularly emotion regulation, in SMI lies in its relationship to other biosystemic domains, including social and community functioning. Furthermore, contradictory findings and methodological differences across studies have limited conclusions that can be drawn about these relationships.

The first aim of this study is to evaluate the interrelationships between emotion processing and psychiatric symptoms. It is hypothesized that more severe positive symptoms of psychosis will be associated with greater use of suppression as a regulatory strategy (Hypothesis 1). As discussed, suppression is generally associated with the experience of more psychiatric symptoms and reappraisal with fewer (Gross & John, 2003); and this has been replicated in schizophrenia (Henry, Rendell, Green, McDonald, & O'Donnell, 2008). Positive symptoms of schizophrenia, such as auditory hallucinations and paranoid delusions, are associated with increases in the emotional experience of anxiety and depression (Freeman, 2007; Lysaker & Salyers, 2007). Emotional experiences such as these are typically down-regulated (John & Gross, 2009) with regulatory strategies such as suppression or reappraisal. This has been replicated in schizophrenia, whereby the severity of auditory hallucinations has been demonstrated to be associated with greater use of suppression (Badcock, Paulik, & Maybery, 2011).

The second aim of this study is to evaluate the interrelationships between emotion processing and neurocognition. It is hypothesized that individuals with better emotion regulation will have higher scores on neurocognitive assessments of attention, memory, and executive functioning (Hypothesis 2). Just as any other type of cognitive processing, emotion processing requires neural resources. At the most basic level, appraisal of perceived emotional stimuli requires attention to those stimuli. Moreover, online interpretations of emotional stimuli require intact working memory (Green & Malhi, 2006). Finally, reinterpretations of these stimuli and self-regulation require higher order cognitive abilities, such as long-term memory and especially executive functioning (Declerck, Boone, & De Brabander, 2006; Gyurak et al., 2009). Executive

functioning appears to be particularly important for regulating emotions during times of distress. However, there are discrepant results regarding whether this is also the case in schizophrenia (Bak et al., 2008; Penn et al., 1993). Limitations in neurocognitive resources would likely have a greater negative effect on individuals who habitually suppress (such as the schizophrenia population) than those who reappraise because suppression requires more cognitive resources (Badcock et al., 2011).

The third aim of this study is to evaluate the interrelationships between emotion processing and social functioning. It is hypothesized that individuals with better emotion regulation will have higher scores on assessments of social and community functioning (Hypothesis 3a). Habitual use of suppression is associated with greater social functioning difficulties in schizophrenia as compared to habitual use of reappraisal (Badcock et al., 2011; Henry et al., 2008). It is further hypothesized that the relationship between emotion regulation and social and community functioning will interact with severity of positive psychotic symptoms (Hypothesis 3b). Cognitive deficits limit the neural resources available for allocation toward self-regulation, social perception, and attention to external stimuli (Ellgring & Smith, 1998), and this may have negative social consequences. Greater focus on internal stimuli, such as positive psychotic symptoms, may further limit the resources available for attention to external stimuli and thereby compromise social functioning.

The fourth aim of this study is to evaluate the interrelationships between emotion processing and treatment history. It is hypothesized that individuals who have completed group treatment modalities will have better emotion regulation (Hypothesis 4a). More specifically, it is hypothesized that individuals who have completed a group

treatment modality based on the specific deficit hypothesis (e.g., SCIT) will have better emotion regulation than individuals who have completed a group treatment modality based on the generalized deficit hypothesis (e.g., IPT) (Hypothesis 4b). As discussed, treatment modalities have been developed for the purpose of remediating social cognitive and emotion processing deficits in SMI. Of particular interest is the effect of "jumping to conclusions" on emotion processing. Individuals who have experienced psychosis are more likely to jump to conclusions, basing conclusions on limited evidence (Dudley, John, Young, & Over, 1997a, 1997b). Moreover, jumping to conclusions is associated with feelings of anxiety, and anxiety with an increase in paranoia (Lincoln, Lange, Burau, Exner, & Moritz, 2010). However, when given more information on which to base conclusions, individuals who have experienced psychosis can change their conclusions (Dudley et al., 1997a, 1997b). The finding that jumping to conclusions may be related to emotion regulation strategies (Livingstone et al., 2009), suggests that modalities aimed at teaching individuals how to evaluate evidence and avoid jumping to conclusions may impact which strategies these individuals use to regulate emotions.

Finally, this study aims to summarize the hypothesized relationships with a path model. It is hypothesized that emotion regulation can be incorporated in pathways to clinical outcome, consistent with pathways previously observed in the SMI population (Hypothesis 5). Previous research has identified pathways to functional outcome in schizophrenia, but none have incorporated emotion regulation. For example, Brekke et al. (2005) established the pathway from neurocognition to global functional outcome via emotion perception. Their results are presented graphically in Figure 5.1.

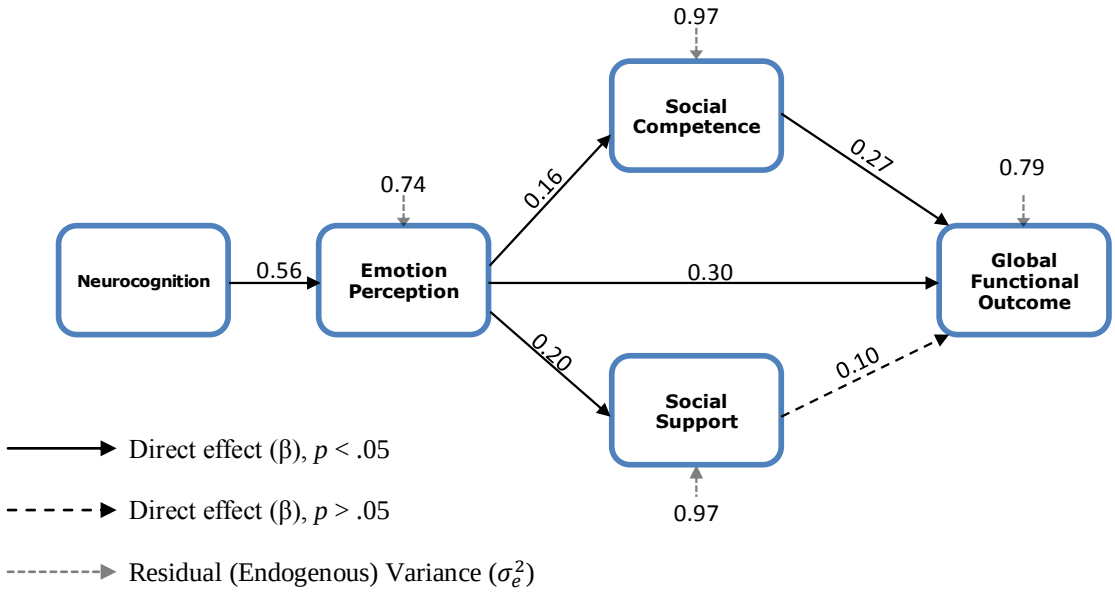

Figure 5.1.

Pathway from Neurocognition to Global Functional Outcome from Brekke et al., 2005

Neurocognition was measured by a composite composed of performance on the Controlled Oral Word Association Test, the Digit Span Distractibility Test, the Degraded-Stimulus Continuous Performance Test, and perseverative errors from the Wisconsin Card Sorting Test.

Emotion perception was measured by a composite composed of performance on the Facial Emotion Identification Test, the Videotape Affect Perception Test, and the Voice Emotion Identification Test.

Social competence was measured by a subscale of the Community Adjustment Form.

Social support was measured by a self-report social support scale adapted from the Medical Outcomes Study Social Support Survey.

Global Functional Outcome was measured from the Role Functioning Scale and included ratings of work, social functioning, and independent living.

In addition, Lipkovich et al. (2009) established the pathway from neurocognition to occupational functioning via symptom severity. Their results are presented graphically in Figure 5.2.

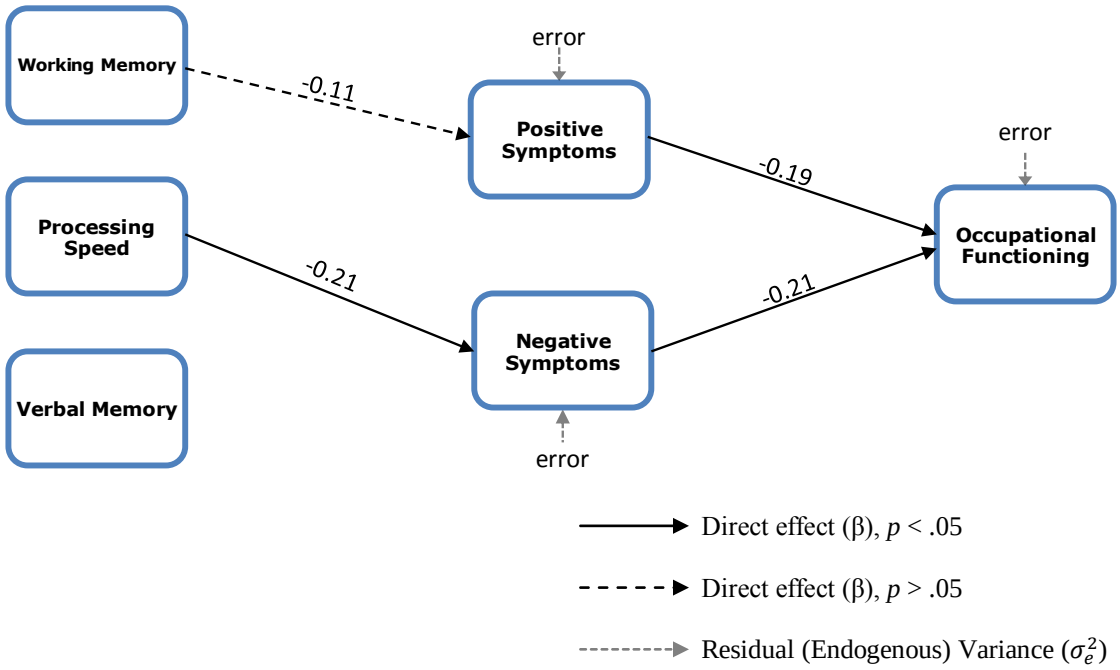

Figure 5.2.

Pathway from Neurocognition to Occupational Functioning from Lipkovich et al., 2009

Working memory was measured by the Letter-Number Sequencing verbal subtest of the Wechsler Adult Intelligence Scale Third Edition.

Processing speed was measured by the average of the WAIS-R Digit-Symbol Coding performance subtest and the Category Instances, and Controlled Oral Word Association Test.

Verbal memory was measured by the Rey Auditory-Verbal Learning Test with Crawford Alternative.

Positive and negative symptoms were measured with the Positive and Negative Syndrome Scale.

Occupational functioning was measured with the Quality of Life Scale Instrumental Role Functioning subdomain.

Inclusion of emotional regulation considerations with these known pathways, and the hypotheses proposed in this study, produces the hypothesized path model described in Figure 5.3. The hypothesized model adapts the results from Brekke et al. (2005) and Lipkovich et al. (2009) to pathways from neurocognition to social functioning via emotion perception and symptom severity. Neurocognition is also expected to predict emotion regulation, following from Hypothesis 2. The path from emotion regulation to social functioning follows from Hypothesis 3, and the path from self-harm to emotion regulation is based on the theory that self-harm is a behavioral

proxy of emotion dysregulation. Finally, the pathways from group skills training participation to emotion perception and social functioning follow from Hypothesis 4 and the hypothesized effects of modalities such as SCIT and IPT on these constructs.

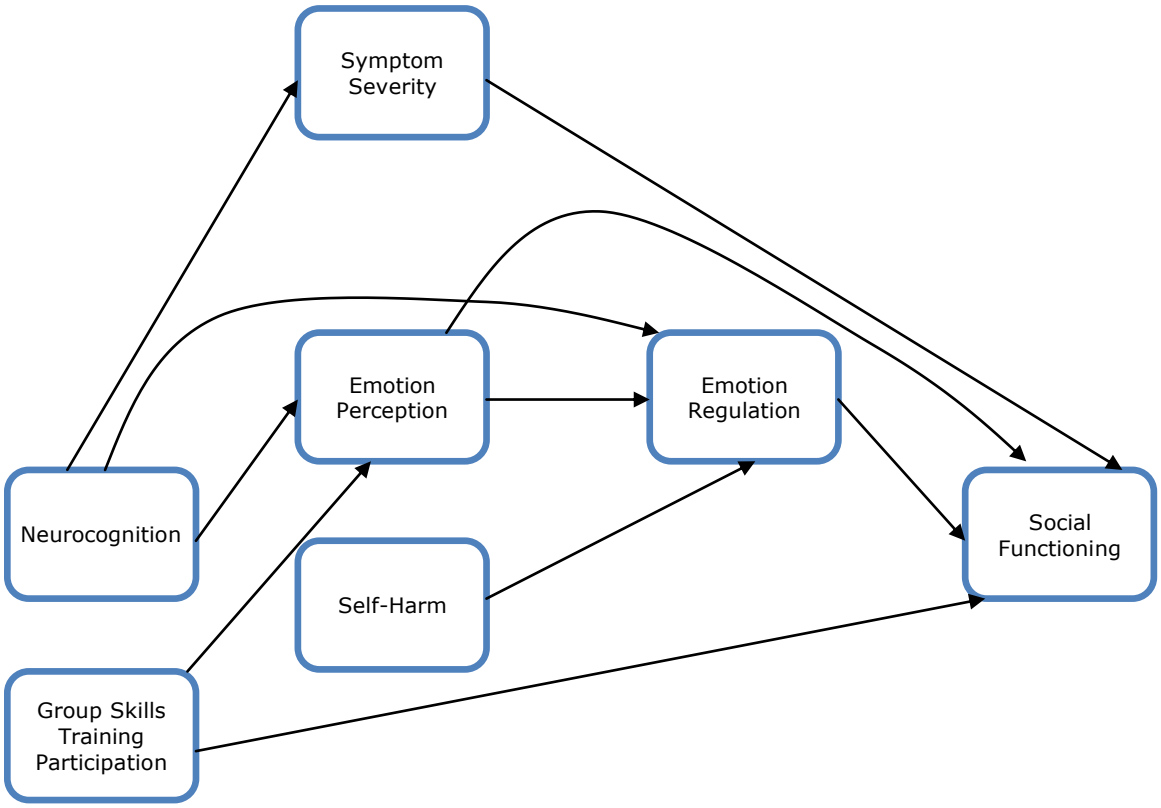

Figure 5.3. Hypothesized Path Model

CHAPTER 6

METHOD

Participants

Participants were recruited from the clients of a local day rehabilitation center serving adults with SMI. Clients of this day rehabilitation center regularly participate in rehabilitative group treatment modalities, including IPT and SCIT. The sample included 42 individuals who collectively are fairly representative of the larger SMI population with respect to psychiatric diagnoses, gender and age distribution, and other demographic and clinical characteristics. One participant was excluded from the analyses, as detailed below. Diagnoses were determined by reviewing participants' clinical records, and all diagnoses were made by a psychiatrist or clinical psychologist. All participants had primary Axis I diagnoses of schizophrenia spectrum disorders (78.1%), Bipolar I Disorder (12.2%), or Major Depressive Disorder (7.3%), and many had secondary Axis I and Axis II disorders. One exclusion criterion was a primary diagnosis of Borderline Personality Disorder. This is primarily a disorder of maladaptive emotion regulation, and the difficulties of individuals with this diagnosis may not be reflective of the difficulties and experiences of the larger SMI population. Participants in the final sample of 41 individuals (32 males, 9 females) included 37 Caucasians, 2 African Americans, and 2 of another race or ethnicity, with an age range of 21 to 70 years (M = 43.6 years; SD = 13.7 years) and education of 10 to 16 years (M = 12.2 years; SD = 1.5 years). Most participants were prescribed antipsychotic medications (85.4%) in addition to other psychotropic medications. Additional demographic and clinical characteristics of the sample are included in Table 6.1.

Table 6.1. *Sample Demographic and Clinical Characteristics*

Variable	*n*	%
Sex		
Male	32	78.0
Female	9	22.0
Ethnicity		
Caucasian	37	90.2
African American	2	4.9
Other	2	4.8
Marital Status		
Single	33	80.5
Divorced	6	14.6
Legal Status at Admission		
Voluntary	18	43.9
Voluntary by Guardian	1	2.4
Mental Health Board Commitment	5	12.2
Court Order	1	2.4
Not Responsible by Reason of Insanity	3	7.3
Axis I Diagnosis		
Schizophrenia, Paranoid Type	12	29.3
Schizophrenia, Disorganized or Undifferentiated Type	5	12.2
Schizoaffective Disorder	15	36.6
Bipolar I Disorder	5	12.2
Major Depressive Disorder	3	7.3
Missing	1	2.4
Second Axis I Diagnosis		
Paraphilia Disorder	2	4.9
Substance Abuse/Dependence (In Remission)	5	12.2
Anxiety Disorder, Not Otherwise Specified	4	9.8
Posttraumatic Stress Disorder	2	4.9
Other (Cognitive Disorder due to Head Injury; Attention Deficit/Hyperactivity Disorder; Asperger's Disorder; Obsessive Compulsive Disorder)	4	9.8
No Diagnosis/Missing	24	58.5
Third Axis I Diagnosis		
Depressive Disorder, Not Otherwise Specified	2	4.9
Other (Substance Abuse/Dependence; Cognitive Disorder, Not Otherwise Specified; Post-Traumatic Stress Disorder; Intermittent Explosive Disorder; Obsessive Compulsive Disorder)	5	12.2
No Diagnosis/Missing	34	82.9
Axis II Diagnosis		
Personality Disorder, NOS	3	7.3
Borderline Intellectual Functioning	2	4.9
No Diagnosis/Deferred/Missing	36	87.8

Variable	*n*	%
Medications		
Antipsychotic	35	85.4
Anticonvulsant/Mood Stabilizer/Lithium	21	50.4
Antidepressant	18	43.9
Anxiolytic	12	29.3
Addiction	2	4.9
Levothyroxine	2	4.9
Stimulant	1	2.4

Measures

The following measures comprise the battery of instruments included in the study.

Clinical instruments.

Brief Psychiatric Rating Scale (BPRS; Ventura et al., 1993). The Brief Psychiatric Rating Scale (BPRS) is a semi-structured interview that rates the presence of 24 psychiatric symptoms over the previous two weeks. The severity of each symptom is rated on a scale from (1) not present to (7) extremely severe. Previous factor analyses have demonstrated that a four factor solution consisting of Thought Disorder (e.g., grandiosity, suspiciousness, hallucinations, unusual thought content), Anergia (motor retardation, uncooperativeness, blunted affect), Affect (somatic concern, anxiety, guilt, depression, hostility), and Disorganization (conceptual disorganization, tension, odd mannerisms and posturing) fits BPRS data well (Mueser, Curran, & McHugo, 1997; Long & Brekke, 1999). Total scores range from 24 to 168, with higher scores reflecting greater symptom severity.

Deliberate Self-Harm Inventory (DSHI; Gratz, 2001). The Deliberate Self-Harm Inventory (DSHI) is a 17-item self-report questionnaire to assess deliberate self-harm. Each item asks participants to indicate whether they have engaged in a particular type of

self-harm and follows affirmative responses with questions about frequency, severity, and duration of harm. This measure will serve as a functional indicator of difficulties with emotion regulation.

In undergraduates, the overall internal consistency (α) is .82. Item-total correlations (*r*) range from .12 to .65. Test-retest reliability is adequate (ϕ = .68, p <.001). Scores correlate with other measures of self-harm, history of suicide attempts, social desirability, and history of psychotherapy.

Researcher and Consumer Emotion Regulation Scale (RACERS; Bottoms, 2011). The Researcher and Consumer Emotion Regulation Scale (RACERS) is a 30-item self-report measure of emotion regulation developed collaboratively with participants at a day rehabilitation center. This new instrument was designed to measure aspects of emotion regulation maximally pertinent to the particular difficulties in this domain associated with SMI. Preliminary psychometric analyses indicate that RACERS has adequate construct validity and internal consistency (α = .77). Scores range from 30 to 180, with higher scores reflecting better emotion regulation.

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The Difficulties in Emotion Regulation Scale (DERS) is a 36-item self-report measure of difficulties with emotion regulation. Participants rate how often the items describe their emotional and behavioral responses to being upset. Each item is rated on a Likert-type scale ranging from (1) almost never to (5) almost always. Items are arranged into 6 subscales: Nonacceptance of Emotional Responses; Difficulties Engaging in Goal-Directed Behavior; Impulse Control Difficulties; Lack of Emotional Awareness; Limited

Access to Emotion Regulation Strategies; and Lack of Emotional Clarity. Scores range from 36 to 180, with higher scores reflecting greater emotion dysregulation.

In undergraduates, the overall internal consistency (α) is .93, ranging from .80 to .89 on subscales. Item-total correlations (*r*) range from .16 to .69.

Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). The Emotion Regulation Questionnaire (ERQ) is a 10-item self-report questionnaire designed to measure the habitual use of suppression and reappraisal. Each item describes a way of controlling particular emotions and is rated on a 7-point Likert scale from (1) strongly agree to (7) strongly agree. Reappraisal scores range from 6 to 42, and suppression scores range from 4 to 28, with higher scores representing greater use of that regulatory strategy. No total score is obtained, as the reappraisal and suppression factors are considered independent.

In undergraduates, the internal consistency (α) for Reappraisal is .79 and for Suppression is .73. Test-retest reliability is .69 for both scales.

Bermond-Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001). The Bermond-Vorst Alexithymia Questionnaire (BVAQ) is a 40-item self-report questionnaire that assesses alexithymia, or a deficiency in the ability to recognize, identify, and distinguish between emotional states. Each item is rated on a 5-point Likert scale from (1) strongly agree to (5) strongly disagree. The items are divided into five subscales in two domains. In the Cognitive domain are the following subscales: Identifying (degree of ability to describe the nature of one's own emotions); Analyzing (degree to which one tries to explain personal emotional states); and Verbalizing (degree to which one is able to verbally communicate about emotional states). In the Affective

domain are the following subscales: Emotionalizing (degree of arousal from emotional stimuli); and Fantasizing (degree of likelihood to fantasize about virtual matters). Scores range from 40 to 200, with higher scores reflecting greater alexithymia. The overall internal consistency (α) is .81 in undergraduates, ranging from .67 to .85 on subscales.

Neurocognition.

The Neuropsychological Assessment Battery – Screening Module (Stern & White, 2003). The Neuropsychological Assessment Battery – Screening Module (NAB-S) is designed to evaluate the neuropsychological functioning of adults aged 18 to 97 who have disorders affecting the central nervous system. As such, it is particularly useful for individuals with SMI, who demonstrate aberrant neurological functioning as reviewed above. The Screening Module briefly screens for impairment in each of five neuropsychological domains: Attention, Language, Memory, Spatial Ability, and Executive Functions. The internal consistency (α) of the NAB-S ranges from .24 to .79. The reliability (G) of the NAB-S ranges from .55 to .91, with an overall reliability of .80.

Trail Making Test - Trails A and B (Reitan & Davidson, 1974). The Trail Making Test (TMT) is a visual search test to assess brain dysfunction and rehabilitative progress in individuals between the ages of 15 and 89 (Strauss, Sherman, & Spreen, 2006). The test is given in two parts, Trails A and Trails B. On Trails A, participants are asked to connect a series of 25 numbered circles in numerical order as quickly as possible; on Trails B, they are asked to connect a series of 25 numbered and lettered circles by alternating the numerical and alphabetical sequences (e.g., connect 1 to A to 2 to B, etc.) as quickly as possible. Generally, the test measures attention, psychomotor

processing speed, and mental flexibility. Scores reflect the time required to complete each part; higher scores reflect slower time to completion.

Reliability of the TMT in individuals with schizophrenia is quite low (α = .36 for Trails A and α = .63 for Trails B). Nevertheless, performance on the TMT is associated with vocational outcome, psychosocial outcome, performance of independent living skills and thus demonstrates substantial utility in this population.

Social cognition.

Face Emotion Identification Task (Kerr and Neale, 1993). The Face Emotion Identification Task (FEIT) is a computer-based task developed to measure emotion recognition in schizophrenia. Participants are shown 19 photographs from Izard (1971) and Ekman (1976), representing happiness, sadness, anger, fear, surprise, and shame. After each photograph, they are asked to identify the present emotion from six choices corresponding to the emotions included in the pictures. The task has an internal consistency (α) of .71 in schizophrenia.

Benton Facial Recognition Test (Benton, Hamsher, Varney, & Spreen, 1983). The Benton Facial Recognition Test (BFRT) measures the ability to recognize unfamiliar faces and is an assessment of perceptual discrimination. Participants are shown a target face and are asked to match that face to other photographs. The first 6 trials consist of straightforward matching of the target to one of six other faces; and the final 16 trials consist of matching the target to three of six faces with altered lighting or orientation. Scores range from 0 to 54, with higher scores reflecting greater perceptual discrimination.

Social functioning.

Social Functioning Scale (Birchwood, Smith, Cochrane, Wetton, & Copestacke, 1990). The Social Functioning Scale (SFS) is a measure of social functioning to assess the efficacy of treatment for schizophrenia. The scale measures skills and behaviors relevant to the impairments of this population. Items are grouped into 7 subscales: Social engagement, Interpersonal behavior, Prosocial activities, Recreation, Independence-Competence (ability to perform independent living skills), Independence-Performance (performance of independent living skills), and Employment/Occupation. Scores range from 0 to 223, with higher scores reflecting better social functioning.

The SFS has good reliability and validity in a sample of individuals with schizophrenia. The overall inter-rater reliability is .94, ranging from .69 to .96 on subscales. The overall scale has an internal consistency (α) of .80, ranging from .69 to .87 on subscales. SFS scores are related to symptom presentation, prosocial behaviors, employment, and performance independent living skills.

Multnomah Community Ability Scale (Barker, Barron, McFarland, & Bigelow, 1994). The Multnomah Community Ability Scale (MCAS) is a functional assessment instrument designed specifically for measuring community functioning in individuals with severe and persistent mental illness living in the community. The original version is intended for clinician completion (most often, case managers) regarding their clients' functioning over the past 30 days; the self-report version (MCAS-SR; Barker, McFarland, & O'Malia, 2004) is intended for consumer completion. The scale includes 17 items covering a range of community abilities in 4 domains: Health, Adaptation, Social Skills, and Behavior. Each item is rated on a 5-point Likert scale; descriptive

anchors vary for each item, but higher ratings reflect greater community functioning. Scores range from 17 to 85, with higher scores reflecting greater community functioning.

The MCAS has good reliability and validity in large community-based SMI populations (Barker et al., 1994; Hendryx, Dyck, McBride, & Whitbeck, 2001; Trauer, 2001; Zani, McFarland, Wachal, Barker, & Barron, 1999). The overall inter-rater reliability is .85, ranging from .32 to .75 on individual items and from .70 to .78 on subscales. The overall test-retest reliability is .83, ranging from .31 to .90 on individual items and from .70 to .82 on subscales. The items appear to be measuring the same construct, as the internal consistency is high ($\alpha = .90$).

Treatment. Participants' completion of IPT and SCIT at the day rehabilitation center will be obtained from their clinical records.

Procedure

Participants were recruited at regular community meetings at a local day rehabilitation center. After a participant expressed interest in the study, the experimental purpose, procedures, risks and benefits were fully explained, and written informed consent was obtained. For participants with legal guardians, guardian consent was obtained before participant assent was obtained.

Participants were asked to complete a battery of measures administered by the author. In total, the battery of self-report and researcher-administered instruments took approximately 3 hours to complete. Most participants completed the battery in one to three sessions over two weeks, depending on individual preferences and fatigue. Two participants did not complete the entire battery, as one was hospitalized before completion of the study and one chose to withdraw from the study. These participants are

included in the analyses for the completed portions of the battery. Participants were compensated $20 for completing the study.

Demographic, clinical, and treatment information was retrieved from treatment records. All data was compiled into a de-identified database for analysis.

Statistical Analyses

Path analyses were conducted in Mplus. Mplus allows for specification of simultaneous regression equations and therefore implies a very specific covariance matrix that better approximates the data than sequential regression equations. Beginning with the hypothesized model, each model specified endogenous (dependent) and exogenous (independent) variables and the covariance between them. Estimated models were evaluated by ensuring that the algorithm converged. Converged models were modified according to theoretical guidance, beginning by examining the normalized residual covariance matrix and individually removing parameters with abnormally large standard errors. Next, parameters with abnormally large modification indices were removed individually, again according to theoretical guidance. Then, model fit statistics were used to evaluate the overall model fit. These included the log-likelihood from the tested model; the log-likelihood of the saturated (unstructured) model in which all variances, covariances, and means are estimated; the Akaike Information Criterion; the Bayesian Information Criterion; the Chi-Square Test of Model Fit; the Root Mean Square Error of Approximation; the Comparative Fit Index; the Tucker Lewis Index; and the Standardized Root Mean Squared Residual. If the estimated model did not have adequate fit, the model parameters were evaluated, and those with non-significant p-values were removed individually. Effect on the model fit was assessed, and those non-significant

parameters that improved model fit when removed were excluded from the model. The model was considered complete when it contained a converged algorithm, stable standard errors, and adequate model fit.

CHAPTER 7

RESULTS

Preliminary Analyses

Mean scores for each of the instruments included in the battery are included in Table 7.1, and the correlation matrices are included in Appendices A – C. Total scores for the Deliberate Self-Harm Inventory were not obtained because many participants with a history of chronic self-harm could not estimate the frequency of this behavior. Therefore, this instrument was used to categorize participants into two groups: Those with a history of self-harm and those with no history of self-harm. Results indicated that 21 participants reported no history of self-harm. Of the 18 participants who indicated a history of self-harm, 8 reported having injured themselves fewer than 6 times, and the remaining 10 individuals had more chronic histories of self-harm.

Table 7.1. *Assessment Descriptive Statistics*

Instrument	*n*	*Scale Range*	*M*	SE	SD
Brief Psychiatric Rating Scale	40	24-168	43.20	1.47	9.31
Thought Disorder Factor	39	3.23-16.32	5.73	0.39	2.41
Anergia Factor	40	2.33-16.32	5.58	0.29	1.80
Affect Factor	40	2.28-15.97	5.87	0.47	2.96
Disorganization Factor	40	1.27-8.88	1.94	0.15	0.92
Researcher & Consumer Emotion Regulation Scale	39	30-180	108.35	2.55	15.95
Difficulties in Emotion Regulation Scale					
Nonacceptance	38	6-30	14.92	1.05	6.47
Goals	38	5-25	13.24	0.72	4.44
Awareness	38	6-30	15.47	0.77	4.75
Strategies	38	8-40	18.08	1.25	7.69
Clarity	38	5-25	11.55	0.66	4.04
Impulse	38	6-30	12.21	0.79	4.88
Total	38	36-180	85.47	3.89	23.95
Emotion Regulation Questionnaire					
Reappraisal	38	6-42	28.97	1.12	6.88
Suppression	38	4-28	16.45	0.74	4.58

Instrument	*n*	*Scale Range*	*M*	SE	SD
Bermond-Vorst Alexithymia Questionnaire					
Cognitive Domain	41	24-120	65.54	2.01	12.85
Verbalizing	41	8-40	25.42	1.12	7.16
Identifying	41	8-40	20.83	1.00	6.40
Analyzing	41	8-40	19.29	0.70	4.46
Affective Domain	41	16-80	45.22	1.46	9.33
Emotionalizing	41	8-40	22.44	0.71	4.52
Fantasizing	41	8-40	22.78	1.09	6.98
Total	41	40-200	110.76	2.33	14.89
Neuropsychological Assessment Battery – Screener (Standard Scores)					
Attention	41		72.73	2.59	16.59
Language	41		100.49	3.10	19.82
Memory	41		80.63	2.58	16.49
Spatial Ability	41		89.10	2.79	17.86
Executive Functioning	41		88.15	2.39	15.32
Total	41		79.88	2.65	16.95
Trail Making Test					
Trails A	41		44.17	3.35	21.46
Trails B	34		98.65	7.56	44.06
Facial Emotion Identification Task					
Proportion Correctly Identified - Overall	41	0-1	0.56	0.03	0.21
Happy	41	0-1	0.85	0.05	0.30
Sad	41	0-1	0.50	0.06	0.38
Angry	41	0-1	0.64	0.05	0.33
Ashamed	41	0-1	0.34	0.05	0.30
Afraid	41	0-1	0.46	0.04	0.26
Surprised	41	0-1	0.76	0.05	0.32
Benton Facial Recognition Test	41	0-54	40.95	0.94	6.00
Social Functioning Scale					
Social Engagement	38	0-15	10.66	0.38	2.35
Interpersonal Behavior	38	0-9	6.97	0.29	1.76
Prosocial Activities	38	0-39	17.18	1.52	9.36
Recreation	38	0-45	19.08	0.80	4.96
Independence – Competence	38	0-66	34.29	1.00	6.17
Independence – Performance	38	0-39	29.26	1.01	6.21
Employment/Occupation	38	0-10	5.45	0.54	3.36
Total	38	0-223	122.89	2.82	17.41
Multnomah Community Ability Scale					
Health	38	5-25	19.34	0.52	3.35
Adaptation	38	3-15	11.37	0.40	2.57
Social Skills	38	5-25	17.98	0.64	4.12
Behavior	38	4-20	18.05	0.37	2.40
Total	38	17-85	66.73	1.47	9.41

Instrument	*n*	*Scale Range*	*M*	SE	SD
Multnomah Community Ability Scale – Self Report					
Health	38	5-25	18.71	0.61	3.74
Adaptation	38	3-15	11.21	0.35	2.17
Social Skills	38	5-25	18.03	0.63	3.87
Behavior	38	4-20	18.34	0.29	1.77
Total	38	17-85	66.29	1.39	8.58
Social Cognition and Interaction Training					
Participated	14				
Not Participated	25				
Progress Rating (Maximum)	14	0-10	8.36	0.37	1.39
Integrated Psychological Therapy					
Participated	24				
Not Participated	15				
Progress Rating (Average)	24	0-10	6.66	0.26	1.29

Total scores were analyzed for each of the 15 instruments administered. Outliers were considered total scores greater than 2.5 standard deviations from the grand mean. The population under investigation is in part defined by departure from the mean. Consistent with this idea, 12 participants (29%) had an outlying score on one instrument, and 1 additional participant had outlying scores on three instruments. Given that 29% of the participants had an outlying score on one instrument in the data set, this pattern of responding was considered normative for the population being studied and the scores were not removed. An exception is a score on Trails B that was more than 4 standard deviations from the mean; this score was removed from the data set. The participant with three outlying scores was removed from the data set entirely for having multiple outlying scores and not representing the larger population.

Performance on the Brief Psychiatric Rating Scale indicates participants' symptoms were generally in the "not present" to "very mild" range (average item score = 1.81, *SD* = 0.40 on scale from 1 to 7). Symptoms related to affective flattening were the

most severe, in the "very mild" to "mild" range on average (average item score = 2.47, *SD* = 1.18), followed by symptoms related to anergia (average item score = 2.38, *SD* = 0.77). Positive symptoms of psychosis and symptoms of disorganization were the least severe on average (Thought disorder average item score = 1.82, *SD* = 0.76; Disorganization average item score = 1.62, *SD* = 0.68).

Participants with a diagnosis of an affective disorder (i.e., Schizoaffective Disorder, Bipolar Disorder, or Major Depressive Disorder) had more severe psychiatric symptoms (*M* = 45.75, *SD* = 9.46) than participants with a diagnosis of schizophrenia (i.e., Schizophrenia Paranoid Type, Disorganized Type, or Undifferentiated Type) (*M* = 39.87, *SD* = 7.90) to a degree approaching clinical significance, $t(37) = 2.01$, $p = .052$. There were no significant differences between the groups on severity of positive symptoms [$t(36) = 1.63$, $p = .112$], affective symptoms [$t(37) = 1.67$, $p = .103$], or disorganization [$t(37) = 1.04$, $p = .307$]. However, participants with a diagnosis of schizophrenia had significantly more severe symptoms of anergia, $t(37) = 2.28$, $p = .029$.

Performance on the Researcher and Consumer Emotion Regulation Scale indicated emotion regulation abilities were in about the average range. The grand mean was 108.35 (*SD* = 15.95), and scores on this instrument range from 30 to 180, with higher scores reflecting greater emotion regulation. There were no significant differences across diagnostic categories on this instrument, $t < 1$.

The grand mean on the Difficulties with Emotion Regulation Scale was 84.47 (*SD* = 23.95), and scores on this instrument range from 36 to 180, with higher scores reflecting more difficulties with emotion regulation. Overall, scores were comparable to those received by patients with schizophrenia in other studies (e.g., Westermann &

Lincoln, 2011). On a 5-point scale (1 = almost never use this strategy; 5 = almost always use this strategy), average item scores ranged from 2.04 (*SD* = 0.81) on the Impulse Control subscale to 2.98 (*SD* = 1.29) on the Nonacceptance of Emotions subscale. This indicated that participants had difficulty with regulating emotions "sometimes" to "about half the time."

Participants with a diagnosis of an affective disorder had more difficulties with emotion regulation (*M* = 92.36, *SD* = 26.31) than participants with a diagnosis of schizophrenia (*M* = 78.13, *SD* = 14.92) to a degree approaching clinical significance, $t(35) = 1.89$, $p = .067$. Participants with a diagnosis of an affective disorder had significantly more difficulties than participants with a diagnosis of schizophrenia with Acceptance of Emotional Responses [$t(35) = 2.17$, $p = .037$] and Engaging in Goal-Directed Behavior [$t(35) = 2.36$, $p = .024$]. There were no significant differences across diagnostic categories on the remaining subscales (Impulse Control Difficulties; Lack of Emotional Awareness; Limited Access to Emotion Regulation Strategies; and Lack of Emotional Clarity).

Performance on the Emotion Regulation Questionnaire indicated that participants tended to slightly agree that they used both suppression and reappraisal as emotion regulation strategies. On a 7-point scale (1 = strongly disagree to using the strategy; 7 = strongly agree to using the strategy), participants rated their use of reappraisal strategies on average a 4.83 (*SD* = 1.15) and their use of suppression strategies on average a 4.11 (*SD* = 1.14). These results are consistent with previous research (e.g., Livingstone et al., 2009). The sample agreed to using reappraisal more than they agreed to using

suppression, $t(37) = 3.52$, $p = .001$. There were no significant differences across diagnostic categories on this instrument, $t < 1$.

The grand mean on the Bermond-Vorst Alexithymia Questionnaire was 110.76 ($SD = 14.89$), and scores on this instrument range from 40 to 200, with higher scores reflecting greater alexithymia. Overall, scores were comparable to those received by patients with schizophrenia in other studies (e.g., van'*t* Wout et al., 2007). On a 5-point scale (1 = This definitely applies; 5 = This in no way applies), participants rated equally the affective and cognitive factors. The affective factor describes the degree to which one tries to explain personal emotional states, degree of ability to describe the nature of one's own emotions, and degree of ability to verbally communicate about emotional states, and participants rated these items on average a 2.83 ($SD = 0.58$). The cognitive factor describes the degree of arousal from emotional stimuli and degree of likelihood to fantasize about virtual matters, and participants rated these items on average a 2.73 ($SD = 0.54$). There were no significant differences across diagnostic categories in degree of alexithymia ($t < 1$). However, participants with a diagnosis of an affective disorder had significantly more difficulty verbally communicating about emotional states ($M = 27.44$, $SD = 7.27$) than participants with a diagnosis of schizophrenia ($M = 23.13$, $SD = 4.85$), $t(38) = 2.03$, $p = .049$.

The standard scores for the Neuropsychological Assessment Battery – Screener indicate that participants scored 1.34 standard deviations below average (age- and education-matched controls) on the total battery. Whereas their performance was average in the Language domain, performance was most impaired in all other domains, with the greatest impairment in the Attention domain (1.82 standard deviations below average).

Participants with a diagnosis of an affective disorder had higher scores on the Memory domain (M = 84.16, SD = 18.07) than participants with a diagnosis of schizophrenia (M = 74.27, SD = 12.17) to a degree approaching significance, $t(38) = 1.88$, $p = .068$. They also performed significantly better tasks of immediate verbal memory [$t(38) = 2.25$, $p = .030$] and verbal fluency and generativity [$t(38) = 2.69$, $p = .010$]. There were no significant differences across diagnostic categories on the remaining domains or tasks of the NAB-Screener.

On average, participants completed Trails A in 44.2 seconds (SD = 21.46 seconds) and Trails B in 98.62 seconds (SD = 44.06 seconds), a statistically significant time difference, $t(33) = 10.66$, $p < .001$. This is consistent with previous findings in schizophrenia of 40.9 seconds to complete Trails A and 97.5 seconds to complete Trails B (Perianez et al., 2007). Of the participants who completed Trails A, 7 were unable to complete Trails B because of frustration with the task or requiring more time than the task allowed. There were no significant differences across diagnostic categories on these instruments.

Overall, participants identified 56% of facial emotions correctly on the Facial Emotion Identification Task. This is consistent with previous findings in schizophrenia of correct identification of 57% (Mueser et al., 1996). Participants were most successful at identifying happy faces (85% identified correctly) and least successful at identifying ashamed faces (34% identified correctly). On average, they correctly identified significantly more positive facial expressions (M = 80.5%, SD = 27.7) than negative facial expressions (M = 48.3%, SD = 22.1%), $t(40) = 8.11$, $p < .001$. There were no significant differences across diagnostic categories on this instrument, $t < 1$.

Performance on the Benton Facial Recognition Test indicated participants correctly identified 40.95 faces on average (*SD* = 2.35). This is comparable to, though slightly higher than, previous findings in schizophrenia of correct identification of approximately 36 faces (Mueser et al., 1996). There were no significant differences across diagnostic categories on this instrument, $t < 1$.

The grand mean on the Social Functioning Scale was 122.89 (*SD* = 17.41), and scores on this instrument range from 0 to 223, with higher scores reflecting better social functioning. This score is comparable to previous reports in schizophrenia of total scores of 122.6 (Addington & Addington, 1999). Greatest performances were in the Interpersonal Behavior (*M* = 6.97, *SD* = 1.76) and Independence – Performance (*M* = 29.26, *SD* = 6.21) domains. Weakest performances were in the Prosocial Activities (*M* = 17.18, *SD* = 9.36) and Recreation (*M* = 19.08, *SD* = 4.96) domains. There were no significant differences across diagnostic categories on overall social functioning, $t < 1$, but participants with a diagnosis of an affective disorder engaged in significantly more prosocial activities than individuals with schizophrenia, $t(35) = 2.18$, $p = .036$.

The grand mean on the Multnomah Community Ability Scale was 66.73 (*SD* = 9.41), and scores on this instrument range from 17 to 85, with higher scores reflecting better community ability. This score is comparable to previous reports in schizophrenia of total scores of 66.5 (Prouteau et al., 2004). Participants' ability was generally in the "slightly impaired" to "moderately impaired" range (average item score = 3.93, *SD* = 1.08 on scale from 1 to 5, with 5 representing "no impairment"). Greatest performance was in the Behavior domain (*M* = 18.05, *SD* = 2.40), and weakest performance was in the Social Skills domain (*M* = 17.98, *SD* = 4.12), but there was no significant difference

between performance in these domains, $t(40) < 1$. There were no significant differences across diagnostic categories on overall community ability, $t(38) = 1.42$, $p = .163$, but participants with a diagnosis of an affective disorder had significantly more impairment in health behaviors (including mood regulation and stress management) than individuals with schizophrenia, $t(38) = 2.52$, $p = .016$.

The grand mean on the Multnomah Community Ability Scale – Self Report version was 66.29 ($SD = 8.58$), and scores on this instrument range from 17 to 85, with higher scores reflecting better community ability. Participants' ability was generally in the "slightly impaired" to "moderately impaired" range (average item score = 3.91, $SD = 1.10$ on scale from 1 to 5, with 5 representing "no impairment"). Greatest performance was in the Behavior domain ($M = 18.34$, $SD = 1.77$), and weakest performance was in the Social Skills domain ($M = 18.03$, $SD = 3.87$) , but there was no significant difference between performance in these domains, $t(37) < 1$. There were no significant differences between clinicans' ratings of participants' community ability on the MCAS and participants' self-reported ratings on the MCAS-SR ($p > .10$ in all cases). There were no significant differences across diagnostic categories on this instrument.

There were 14 participants who had completed Social Cognition and Interaction Training and 24 who had completed Integrated Psychological Therapy. Of these, 12 had completed both SCIT and IPT and 15 participants who had completed neither SCIT nor IPT. Whereas 12 participants had completed IPT but not SCIT, only 2 participants had completed SCIT but not IPT. The overall pattern in the distribution was $X^2(1) = 5.39$, $p = .020$, indicating that fewer people had completed SCIT but not IPT than expected. The sample was evenly divided across diagnostic categories (participants with a diagnosis of

an affective disorder vs. participants with a diagnosis of schizophrenia) in participation in SCIT [$X^2(1) = 0.74$, $p = .391$] and IPT [$X^2(1) = 0.11$, $p = .744$].

The correlation matrix of the variables included in the analyses indicated that there was no systematic variation among variables that would theoretically be more versus less related (see Appendices A-C). This precluded composition of composite latent variables such as an Emotion Regulation variable that includes all of the measures of emotion regulation in the study. Therefore, each hypothesis is tested with a series of models examining the results of various measures to determine whether the same kinds of predictive relationships hold for each of the measures of the relevant constructs.

Variables were centered at the mean score for regression analyses. The categorical variable representing history of self-harm (DSHI) was coded such that 0 represented no history of self-harm and 1 represented a history of self-harm.

Hypothesis 1: Symptom Severity and Emotion Regulation

Hypothesis 1 predicted that more severe positive symptoms of psychosis would be associated with greater use of suppression as a regulatory strategy. Positive symptoms were tested with the Thought Disorder factor of the Brief Psychiatric Rating Scale (BPRS), and suppression was tested with the Suppression subscale of the Emotion Regulation Questionnaire (ERQ). Correlation analyses were conducted to examine the relationship between severity of positive symptoms and use of suppression as a regulatory strategy. As shown in Table 7.2, positive symptom severity and suppression were not significantly correlated ($r = .11$, $p = .521$), indicating that suppression did not contribute to predicting positive symptom severity.

The same analysis was repeated to examine the relationship between severity of positive symptoms and other indicators of emotion regulation. All measures of emotion regulation were non-significantly correlated with positive symptom severity, except greater emotion dysregulation as measured by the Difficulties with Emotion Regulation Scale (DERS) predicted more severe positive symptoms (r = .36, p = .025). Table 7.2 summarizes the analysis results.

The analyses were repeated to examine the relationship between global psychiatric symptom severity and indicators of emotion regulation. Again, suppression did not contribute to predicting global symptom severity (r = .14, p = .388). However, greater overall emotion dysregulation as measured by the DERS predicted more severe psychiatric symptoms (r = .60, p < .001), and this relationship approached significance as measured by RACERS (r = -.29, p = .083). Finally, individuals with a history of self-harm tended to have more severe psychiatric symptoms than individuals with no history of self-harm (r = .44, p = .005). Table 7.2 summarizes the analysis results.

Table 7.2. *Correlation Matrix for Measures of Emotion Regulation and Symptom Severity*

Emotion Regulation Measure	Positive Symptom Severity	Global Symptom Severity
ERQ		
Suppression	.11	.14
Reappraisal	-.13	-.21
RACERS	-.22	-.29*
DERS	**.36****	**.60*****
BVAQ	.02	-.08
DSHI		
0 = no history of self-harm	.11	**.44*****
1 = history of self-harm		

Note.
Bold values are p < .05.
* Correlation is approaching significance at the 0.10 level (2-tailed).
** Correlation is significant at the 0.05 level (2-tailed).
*** Correlation is significant at the 0.01 level (2-tailed).
Positive Symptom Severity = Thought Disorder factor of BPRS.
Global Symptom Severity = Total BPRS score.

ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.

Overall, there was no evidence specifically supporting a relationship between positive symptoms of psychosis and suppression as a regulatory strategy. However, there is some evidence of a broader relationship between more global psychiatric symptoms and more global emotion dysregulation.

Examination of the descriptive statistics indicated differences approaching significance between participants with diagnoses of affective disorders versus participants with a diagnosis of schizophrenia on overall symptom severity [$t(37) = 2.01$, $p = .052$] and global emotion dysregulation as measured by the DERS [$t(35) = 1.89$, $p = .067$]. This indicates that an interaction may exist that accounts for some of the relationship between symptom severity and emotion regulation. A multiple regression analysis was conducted to examine the interaction between emotion regulation (as measured by the DERS), symptom severity (as measured by the BPRS total score), and diagnostic category (as measured by binary grouping of affective diagnoses, coded 0, versus non-affective diagnoses, coded 1). In the regression, variables were centered at the mean prior to the analysis. The first model included symptom severity as a predictor of DERS total score; the second model added diagnostic category as a predictor; and a third model added the interaction between symptom severity and diagnostic category as a predictor, computed as the product of these variables.

The multiple regression model predicting emotion regulation from symptom severity (BPRS) produced $R^2 = .33$, $F(1, 35) = 17.14$, $p < .001$. Adding diagnostic category as a predictor did not significantly improve the model fit ($R^2\Delta = .02$, $p = .276$), nor did including the interaction between symptom severity and diagnostic category ($R^2\Delta = .01$, $p = .398$). The final model produced $R^2 = .37$, $F(3, 33) = 6.35$, $p = .002$. In the final model, symptom severity had a significant positive regression weight ($\beta = .62$, $p = .002$). Diagnostic category had a non-significant regression weight ($\beta = -.17$, $p = .245$), as did the interaction between symptom severity and diagnostic category ($\beta = -.15$, $p = .398$). Overall, it does not appear that a distinction between affective and non-affective diagnoses is responsible for the relationship between symptom severity and emotion regulation.

Hypothesis 2: Neurocognition and Emotion Regulation

Hypothesis 2 predicted that individuals with better emotion regulation would have higher scores on neurocognitive assessments of attention, memory, and executive functioning. A series of analyses were used to test this hypothesis, using the following measures of emotion regulation: Deliberate Self-Harm Inventory (DSHI), Researcher and Consumer Emotion Regulation Scale (RACERS), Difficulties with Emotion Regulation Scale (DERS), Emotion Regulation Questionnaire (ERQ Reappraisal and Suppression subscales), and the Bermond-Vorst Alexithymia Questionnaire (BVAQ). Measures of neurocognition were separated into those measuring attention (NAB-Screener Attention domain and Trail Making Test A), memory (NAB-Screener Memory domain), and executive functioning (NAB-Screener Executive Functioning domain and Trail Making Test B). Correlations between each of these domains and the various assessments of

emotion regulation will be discussed individually. Table 7.3 summarizes the correlation results.

Table 7.3. *Correlation Matrix for Measures of Emotion Regulation and Neurocognition*

Emotion Regulation Measure	Neurocognition Measure				
	NAB-Screener Domain			Trail Making Test	
	Attention	Memory	Executive Functioning	A	B
RACERS	.05	.10	.07	-.13	.08
DERS					
Nonacceptance	.21	-.08	.30*	-.21	**-.39****
Goals	-.002	.03	.09	.03	-.14
Awareness	.03	-.04	.06	-.08	-.14
Strategies	-.03	-.12	.03	-.08	-.32*
Clarity	.02	.03	-.05	.13	-.12
Impulse	**-.34****	-.21	-.21	.22	-.02
Total	-.02	-.10	.07	-.03	-.28
ERQ					
Reappraisal	-.08	.24	-.21	**.32****	.10
Suppression	-.21	-.08	-.28*	.30*	-.01
BVAQ					
Cognitive Domain	.16	.08	.01	-.07	-.25
Verbalizing	.31*	.22	.07	-.13	**-.39****
Identifying	.02	-.02	-.03	.01	-.03
Analyzing	-.07	-.08	-.02	.003	-.07
Affective Domain	.10	-.09	-.20	.16	**.39****
Emotionalizing	.08	.04	.06	.04	**.37****
Fantasizing	.08	-.15	**-.31****	.19	.29*
Total	.19	.01	-.12	.04	.03
DSHI					
0 = no history of self-harm 1 = history of self-harm	.05	.04	-.03	-.25	-.26

Note.
Bold values are $p < .05$.
* Correlation is approaching significance at the 0.10 level (2-tailed).
** Correlation is significant at the 0.05 level (2-tailed).
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.
ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.

Correlations between emotion regulation and attention. The NAB-Screener Attention domain was not significantly correlated with any of the measures of emotion regulation. However, it was significantly correlated with the Impulse Control subscale of the DERS (r = .34, p = .034). This indicates that individuals with higher attention scores tend to have better impulse control, though this relationship does not extend to the more global measure of emotion dysregulation.

Trail Making Test A was only significantly correlated with the Reappraisal subscale of the ERQ (r = .32, p = .05). This indicates that individuals with higher processing speed tend to rely on reappraisal as an emotion regulation strategy. The correlation between Trail Making Test A and the Suppression subscale of the ERQ was approaching significance (r = .30, p = .068), indicating that individuals with higher processing speed may also tend to use suppression as an emotion regulation strategy.

Correlations between emotion regulation and memory. There were no significant correlations between the NAB-Screener Memory domain and any of the measures of emotion regulation, indicating the absence of a relationship between these constructs. Although participants with a diagnosis of an affective disorder had higher scores on the Memory domain than participants with a diagnosis of schizophrenia to a degree approaching significance, $t(38) = 1.88$, p = .068, controlling for diagnostic category did not bring any of the correlations between the NAB-Screener Memory domain and any of the measures of emotion regulation to significance, $p > .05$ in all instances. Therefore, the absence of a relationship between these constructs exists for participants with affective and non-affective diagnoses.

Correlations between emotion regulation and executive functioning. The NAB-Screener Executive Functioning domain was only significantly correlated with the Fantasizing subscale of the BVAQ (r = -.31, p = .047). This indicates that individuals with higher executive functioning scores are more likely to fantasize about virtual matters. The correlation between the NAB-Screener Executive Functioning domain and the Nonacceptance subscale of the DERS was approaching significance (r = .30, p = .071), indicating that individuals with higher executive functioning scores may be less likely to have an accepting attitude toward their emotional responses. The correlation between the NAB-Screener Executive Functioning domain and the Suppression subscale of the ERQ was also approaching significance (r = .30, p = .068), indicating that individuals with higher executive functioning scores may be more likely to use suppression as an emotion regulation strategy.

Trail Making Test B was significantly correlated with the Nonacceptance subscale of the DERS (r = -.39, p = .031) and its correlation with the Strategies subscale of the DERS was approaching significance (r = -.32, p = .084). This indicates that individuals with greater ability to switch cognitive sets tend to be more accepting of their emotional responses but may have poorer access to emotion regulation strategies.

Trail Making Test B was significantly correlated with the Affective domain of the BVAQ (r = .39, p = .023), indicating that individuals with greater ability to switch cognitive sets are more likely to experience arousal from emotional stimuli (r = .37, p = .031). Trail Making Test B was also significantly correlated with the Verbalizing subscale of the BVAQ (r = -.39, p = .023), indicating that individuals with greater ability

to switch cognitive sets are less able to verbally communicate about their emotional states.

Multiple regression analyses predicting emotion regulation from neurocognition. Next, a series of multiple regression analyses were conducted to examine the relationship between emotion regulation and neurocognition. In each analysis, the five domains of the NAB-Screener (attention, language ability, memory, spatial ability, and executive functioning) were centered at the mean and simultaneously entered into the model predicting one of the measures of emotion regulation (DSHI, RACERS, DERS, Reappraisal (ERQ), Suppression (ERQ), or BVAQ). In total, six analyses were conducted. The results are summarized in Table 7.4. Consistent with the limitations found in the correlation matrix, only one model approached significance: the model predicting alexithymia (BVAQ). The model produced $R^2 = .26$, $F(5, 35) = 2.47$, $p = .051$. Significant regression weights included attention ($\beta = .67$, $p = .007$) and executive functioning ($\beta = -.45$, $p = .034$), indicating that these neurocognitive domains contribute to predicting alexithymia, after controlling for performance in other neurocognitive domains.

Table 7.4. *Multiple Regression Models Predicting Emotion Regulation from Neurocognition*

Criterion		Predictors						Model	
		Constant	Attention	Language	Memory	Spatial Ability	Executive Function	R^2	p (F)
DSHI									
	β		.084	.106	-.002	-.027	-.117	.017	.988
	p	**< .001**	.765	.645	.991	.892	.642		
RACERS									
	β		-.195	.297	-.051	.139	.093	.096	.625
	p	.956	.471	.185	.797	.464	.701		
DERS									
	β		.050	-.235	.019	-.248	.177	.129	.463
	p	.927	.853	.282	.920	.186	.465		
ERQ Reappraisal									
	β		.089	-.056	.372	-.135	-.330	.165	.303
	p	.730	.736	.792	.055	.457	.169		
ERQ Suppression									
	β		.002	-.017	.022	-.060	-.263	.080	.730
	p	.937	.995	.939	.913	.754	.294		
BVAQ									
	β		**.669**	-.243	.045	-.233	**-.451**	.261	.051
	p	.999	**.007**	.216	.793	.151	**.034**		

Note.
Bold values are $p < .05$.
Each criterion represents a single regression equation with 5 predictors (the five domains of the NAB-Screener: attention, language ability, memory, spatial ability, and executive functioning). Thus, this table represents the results of 6 independent regression equations.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.
ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.

Summary. Overall, neurocognition appears to explain very little of the variance in emotion regulation. However, as predicted, attention and executive functioning do have the greatest contribution to predicting emotion regulation. Moreover, they provide a unique contribution to predicting emotion regulation after controlling for other aspects of neurocognitive functioning.

Hypothesis 3: Social and Community Functioning and Emotion Regulation

Correlations between social and community functioning and emotion regulation. Hypothesis 3a predicted that individuals with better emotion regulation will have higher scores on assessments of social and community functioning. Again, a series of analyses were used to test this hypothesis, using the five measures of emotion regulation [Deliberate Self-Harm Inventory (DSHI), Researcher and Consumer Emotion Regulation Scale (RACERS), Difficulties with Emotion Regulation Scale (DERS), Emotion Regulation Questionnaire (ERQ Reappraisal and Suppression subscales), and the Bermond-Vorst Alexithymia Questionnaire (BVAQ)] and the three measures of social and community functioning [Social Functioning Scale (SFS), Multnomah Community Ability Scale (MCAS), and Multnomah Community Ability Scale-Self Report (MCAS-SR)]. The results are summarized in Table 7.5.

Social functioning as measured by the Social Functioning Scale was correlated with measures of emotion regulation. Greater use of reappraisal (an indicator of good emotion regulation) as measured by the ERQ was correlated with better social functioning as measured by the SFS (r = .49, p = .002), with a particularly strong relationship with the Prosocial Activities (r = .52, p = .001) subscale. Individuals who had engaged in self-injury in the past (an indicator of poor emotion regulation) tended to have lower social functioning (r = -.56, p < .001), with particularly strong relationships to the interpersonal communication (r = -.60, p < .001) and prosocial activities (r = -.38, p = .019) subscales. Self-reported difficulties with emotion regulation as measured by the DERS were correlated with lower social functioning to a degree approaching significance (r = -.31, p = .056), and significantly correlated with the Social Engagement (r = -.39, p =

.016) and Interpersonal Communication (r = -.45, p = .005) subscales. Social functioning as measured by the SFS was not significantly correlated with emotion regulation as measured by RACERS (r = .08, p = .622), use of suppression as a regulatory strategy (r =-.06, p = .729), or alexithymia (r =-.11, p = .526).

Social functioning as measured by the clinician version of the Multnomah Community Ability Scale was marginally related to difficulties with emotion regulation as measured by the DERS (r = -.29, p = .076) such that individuals with more difficulties with emotion regulation tended to have poorer community ability. Clinician-rated community ability was not correlated with a history of self-injury (r = -.02, p = .913), use of reappraisal as a regulatory strategy (r = .26, p = .119), use of suppression as a regulatory strategy (r = -.12, p = .475), emotion regulation as measured by RACERS (r = .08, p = .645), or alexithymia (r = .18, p = .259).

Social functioning as measured by the self-report version of the Multnomah Community Ability Scale was correlated with measures of emotion regulation. Individuals with a history of self-injury tended to rate their overall community ability as lower (r = -.47, p = .003), and in particular their social skills (r = -.53, p = .001). Self-reported community ability was also strongly correlated with self-reported emotion regulation as measured by RACERS (r = .38, p = .019). This relationship extended to the Adaptation (r = .42, p = .009) and Behavior (r = .33, p = .043) subscales. Difficulties with emotion regulation as measured by the DERS were strongly related to poorer community ability (r = -.64, p < .001), which extended to the Health (r = -.49, p = .002), Social Skills (r = -.55, p < .001), and Behavior (r = -.54, p < .001) subscales of the MCAS-SR. There was no relationship between use of reappraisal or suppression as

regulatory strategies and self-reported community ability (Reappraisal: r = .23, p = .157; Suppression: r = -.05, p = .752). Nor was there a relationship between alexithymia and self-reported community ability (r = -.21, p = .218).

Table 7.5. *Correlation Matrix for Measures of Emotion Regulation and Social and Community Functioning*

	Social and Community Functioning Measure		
Emotion Regulation Measure	SFS	MCAS	MCAS-SR
RACERS	.083	.076	**.380****
DERS	-.313*	-.291*	**-.639*****
ERQ			
Reappraisal	**.492*****	.258	.234
Suppression	-.058	-.119	-.053
BVAQ	-.106	.180	-.205
DSHI			
0 = no history of self-harm	**-.561*****	-.018	**-.468*****
1 = history of self-harm			

Note. Bold values are $p < .05$.

* Correlation is approaching significance at the 0.10 level (2-tailed).

** Correlation is significant at the 0.05 level (2-tailed).

*** Correlation is significant at the 0.01 level (2-tailed).

RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.

DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.

ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.

BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.

DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.

SFS = Social Functioning Scale; higher scores = better social functioning.

MCAS = Multnomah Community Ability Scale; higher scores = better community ability.

MCAS-SR = Multnomah Community Ability Scale – Self-Report; higher scores = better community ability.

Interaction with severity of positive psychotic symptoms. Hypothesis 3b predicted that the relationship between emotion regulation and social and community functioning would interact with severity of positive psychotic symptoms. Multiple regression analyses were conducted to examine the interaction between emotion regulation, social functioning, and positive psychotic symptoms. Table 7.6 summarizes the correlation matrix between positive symptom severity and measures of social and

community functioning. Correlations between positive symptom severity and measures of emotion regulation were noted above, see Table 7.2.

Positive symptom severity was significantly correlated with social functioning as measured by the SFS (r = -.31, p = .030) and MCAS-SR (r = -.29, p = .039), but not as measured by the MCAS (r = -.21, p = .102).

Table 7.6. *Correlation Matrix for Positive Symptom Severity and Social and Community Functioning*

	Positive Symptom Severity
SFS	-.31**
MCAS	-.21
MCAS-SR	-.29**

Note. Bold values are $p < .05$.
* Correlation is approaching significance at the 0.10 level (2-tailed).
** Correlation is significant at the 0.05 level (2-tailed).
*** Correlation is significant at the 0.01 level (2-tailed).
SFS = Social Functioning Scale; higher scores = better social functioning.
MCAS = Multnomah Community Ability Scale; higher scores = better community ability.
MCAS-SR = Multnomah Community Ability Scale – Self-Report; higher scores = better community ability.

In all regression models reported below, variables were centered at the mean prior to the analysis. Each analysis followed a similar pattern of evaluating the interaction hypothesized in Hypothesis 3b: the first model includes a single measure of social functioning as a predictor for a single measure of emotion regulation; the second model adds positive symptom severity as a predictor; and the third model adds the interaction between positive symptom severity and social functioning as a predictor, computed as the product of these variables.

Social Functioning Scale as predictor. As shown in Table 7.7, consistent with the correlation matrix, most of the multiple regression models predicting various measures of emotion regulation from social functioning (as measured by the Social Functioning Scale) produced nonsignficant models. The fit of these models was improved

neither by including positive symptom severity as a predictor nor by including the interaction between positive symptom severity and social functioning.

Table 7.7. *Hierarchical Multiple Regression Analyses Predicting Emotion Regulation from Social Functioning, Positive Symptom Severity, and the Interaction between Social Functioning and Positive Symptom Severity*

Social Functioning Predictor	Criterion	Model 1			Model 2		Model 3	
		R^2	F	p	$R^2\Delta$	p ($F\Delta$)	$R^2\Delta$	p ($F\Delta$)
SFS								
	DSHI	**.315**	**16.571**	**<.001**	.005	.631	.000	.938
	RACERS	.007	0.248	.622	.043	.215	**.120**	**.033**
	DERS	.098	3.914	.056	.079	.076	.080	.065
	Reappraisal (ERQ)	**.242**	**11.480**	**.002**	.000	.892	.000	.947
	Suppression (ERQ)	.003	0.122	.729	.009	.579	.006	.646
	BVAQ	.011	0.411	.526	.019	.411	.001	.836
MCAS								
	DSHI	.002	0.070	.793	.015	.477	.005	.691
	RACERS	.014	0.501	.484	.041	.224	.000	.953
	DERS	.085	3.335	.076	.095	.051	.000	.897
	Reappraisal (ERQ)	.066	2.557	.119	.006	.628	.059	.137
	Suppression (ERQ)	.014	0.521	.475	.007	.618	.011	.545
	BVAQ	.037	1.416	.242	.009	.567	**.102**	**.048**
MCAS-SR								
	DSHI	**.219**	**10.116**	**.003**	.001	.852	.010	.513
	RACERS	**.144**	**6.058**	**.019**	.014	.449	.015	.433
	DERS	**.409**	**24.880**	**<.001**	.035	.148	.005	.573
	Reappraisal (ERQ)	.055	2.094	.157	.005	.684	.068	.113
	Suppression (ERQ)	.003	0.101	.752	.009	.571	.024	.367
	BVAQ	.205	1.571	.218	.015	.507	.003	.760

Note.
Bold values are $p < .05$.
Each row represents a single regression equation with a single predictor (either SFS, MCAS, or MCAS-SR) for Model 1.
In Model 2, each row represents the regression equation from Model 1 with an additional predictor (positive symptom severity).
In Model 3, each row represents the regression equation from Model 2 with an additional predictor (interaction between a single measure of social functioning and positive symptom severity).
Thus, this table represents the results of 54 independent regression equations.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.
ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.
SFS = Social Functioning Scale; higher scores = better social functioning.
MCAS = Multnomah Community Ability Scale; higher scores = better community ability.

MCAS-SR = Multnomah Community Ability Scale – Self-Report; higher scores = better community ability.

There are two noteworthy exceptions. The multiple regression model predicting emotion regulation as measured by RACERS from social functioning (SFS) produced $R^2 = .01$, $F < 1$. Adding positive symptom severity to the model did not significantly improve the model fit ($R^2\Delta = .04$, $p = .215$). However, including the interaction between positive symptom severity and social functioning significantly improved the model fit ($R^2\Delta = .12$, $p = .033$). The final model produced $R^2 = .17$, F (3, 34) = 2.33, $p = .092$. In the final model, social functioning had a non-significant regression weight ($\beta = .14$, $p = .440$) and positive symptom severity had a regression weight approaching significance ($\beta = -.32$, $p = .071$). The interaction between social functioning and positive symptom severity had a significant negative regression weight ($\beta = -.39$, $p = .033$), indicating that the relationship between social functioning and emotion regulation becomes less positive as symptom severity increases. Figure 7.1 shows the interaction graphically.

When social functioning is above average (high), there is a significant relationship between positive symptom severity and emotion regulation such that individuals who have more severe positive symptoms tend to have worse emotion regulation than individuals with less severe positive symptoms ($\beta = -.62$, $p = .016$). The relationship between positive symptom severity and emotion regulation is somewhat smaller for individuals with average social functioning ($\beta = -.32$, $p = .071$), and it becomes zero for individuals with below average social functioning ($\beta = -.02$, $p = .926$).

In general, there is a non-significantly positive relationship between social functioning and emotion regulation for individuals with few positive symptoms ($\beta = .43$,

p = .092), there is a non-significantly positive relationship for individuals with an average level of positive symptoms (β = .14, p = .440), and there is a non-significantly negative relationship for individuals with very severe positive symptoms (β = -.16, p = .377). Overall, positive symptoms have the greatest impact on the relationship between emotion regulation and social functioning when social functioning is high.

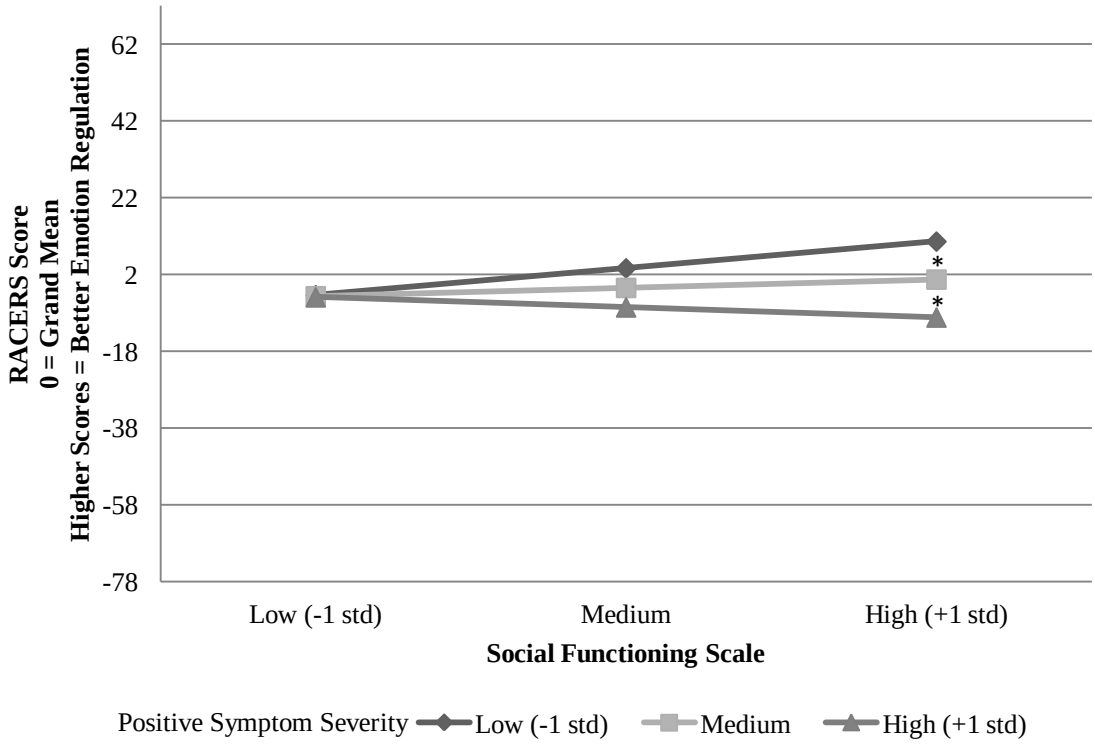

Figure 7.1. Positive Symptoms Moderate Relationship between SFS and RACERS

* Mean difference is p < .05
RACERS scores are centered such that 0 represents the grand mean.
Low = individuals with scores one standard deviation below the grand mean.
Medium = individuals with scores at the grand mean.
High = individuals with scores one standard deviation above the grand mean.
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
SFS = Social Functioning Scale; higher scores = better social functioning.
Positive Symptoms = measured by Thought Disorder Factor of BPRS; higher scores = more severe positive symptoms

Additionally, the multiple regression model predicting difficulties in emotion regulation as measured by DERS from social functioning (SFS) produced R^2 = .10, F(1,

36) = 3.91, p = .056. Adding positive symptom severity to the model improved the model fit to a degree approaching significance ($R^2\Delta$ = .08, p = .076). Similarly, including the interaction between positive symptom severity and social functioning improved the model fit to a degree approaching significance ($R^2\Delta$ = .08, p = .065). In the final model, social functioning had a negative regression weight approaching significance (β = -.32, p = .059), positive symptom severity had a significant positive regression weight (β = .38, p = .026), and their interaction had a regression weight approaching significance (β = .32, p = .065). Figure 7.2 shows the interaction graphically.

When social functioning is above average (high), there is a significant relationship between positive symptom severity and emotion regulation such that individuals who have more severe positive symptoms tend to have worse emotion regulation than individuals with less severe positive symptoms (β = .62, p = .011). The relationship between positive symptom severity and emotion regulation is somewhat smaller for individuals with average social functioning (β = .38, p = .026), and it becomes non-significant for individuals with below average social functioning (β = .13, p = .464).

In general, there is a significant positive relationship between social functioning and emotion regulation for individuals with few positive symptoms (β = -.56, p = .023), such that fewer symptoms are associated with better emotion regulation as social functioning increases. There is also a positive relationship approaching significance for individuals with an average level of positive symptoms (β = -.38, p = .059), such that fewer symptoms are associated with better emotion regulation as social functioning increases. And there is no relationship between emotion regulation and social functioning for individuals with severe positive symptoms (β = -.08, p = .661). Overall, consistent

with the results obtained with RACERS, positive symptoms have the greatest impact on the relationship between emotion regulation and social functioning when social functioning is high.

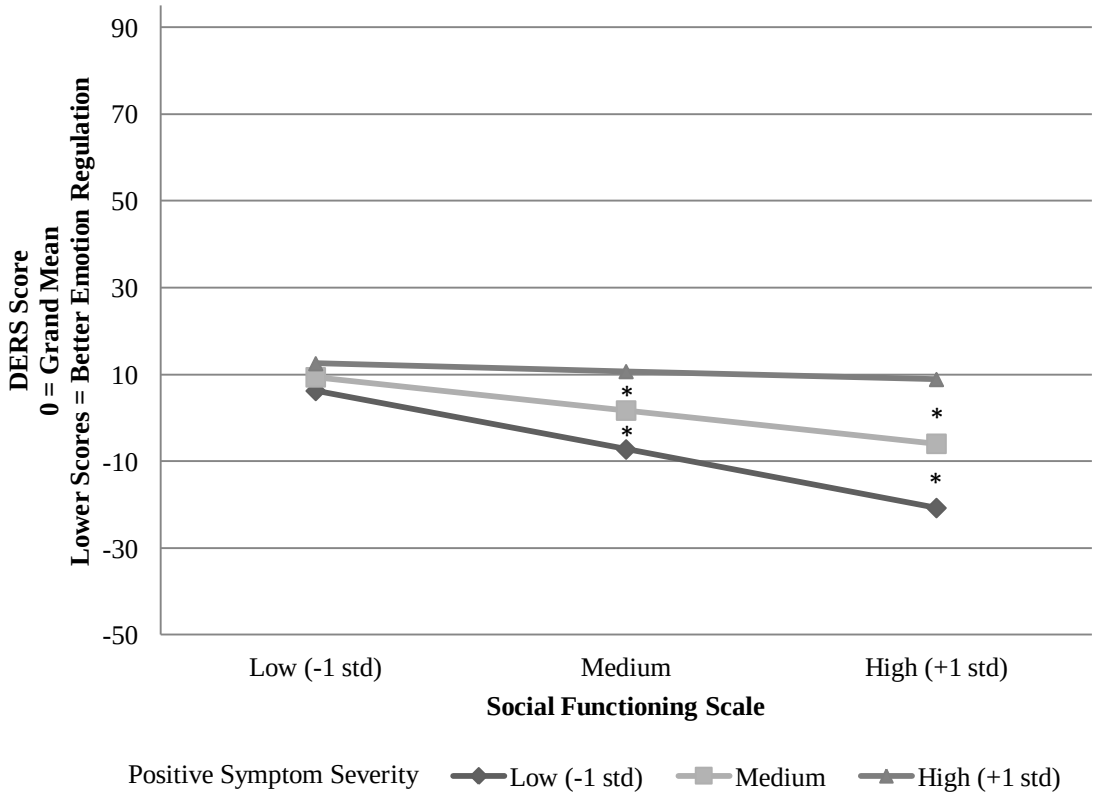

Figure 7.2. Positive Symptoms Moderate Relationship between SFS and DERS

* Mean difference is $p < .05$

DERS scores are centered such that 0 represents the grand mean.

Sx = Positive Symptoms as measured by Thought Disorder Factor of BPRS.

Low = individuals with scores one standard deviation below the grand mean.

Medium = individuals with scores at the grand mean.

High = individuals with scores one standard deviation above the grand mean.

DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.

SFS = Social Functioning Scale; higher scores = better social functioning.

Multnomah Community Ability Scale as predictor. Similar to the results obtained for the SFS as a predictor, most of the multiple regression models predicting various measures of emotion regulation from the MCAS produced nonsignficant models,

the fit of which was not improved by including positive symptom severity as a predictor nor by including the interaction between positive symptom severity and community ability. Again, there are two noteworthy exceptions.

The multiple regression model predicting difficulties in emotion regulation as measured by DERS from community ability (MCAS) produced $R^2 = .09$, $F(1, 36) = 3.34$, $p = .076$. Adding positive symptom severity to the model improved the model fit to a degree approaching significance ($R^2\Delta = .10$, $p = .051$). However, including the interaction between positive symptom severity and social functioning did not significantly improve model fit ($R^2\Delta = .00$, $p = .897$). Nevertheless, positive symptom severity does appear to account for some of the relationship between emotion regulation and community ability.

Additionally, the multiple regression model predicting alexithymia (BVAQ) as a proxy of emotion regulation from community ability (MCAS) produced $R^2 = .04$, $F(1, 36) = 1.42$, $p = .242$. Adding positive symptom severity to the model did not improve model fit ($R^2\Delta = .01$, $p = .567$). However, adding the interaction between positive symptom severity and social functioning did significantly improve model fit ($R^2\Delta = .10$, $p = .048$). In the final model, community ability had a non-significant regression weight ($\beta = .22$, $p = .202$) and positive symptom severity had a non-significant regression weight ($\beta = .20$, $p = .254$). The interaction between community ability and positive symptom severity had a significant positive regression weight ($\beta = .34$, $p = .048$), indicating that the relationship between social functioning and emotion regulation becomes more positive as symptom severity increases. Figure 7.3 shows the interaction graphically.

When community ability is below average (low), there is no relationship between positive symptom severity and alexithymia (β = -.04, *p* = .839). The relationship becomes more positive for individuals with average community ability (β = .20, *p* = .254) and it approaches significance for individuals with above average community ability (β = .44, *p* = .070), such that individuals with more severe positive symptoms tend to have more alexithymia than individuals with less severe positive symptoms.

There is no relationship between community ability and alexithymia for individuals with few positive symptoms (β = -.04, *p* = .917). The relationship becomes more positive for individuals with an average level of positive symptoms (β = .37, *p* = .202), and it becomes significantly positive for individuals with above average levels of positive psychotic symptoms (β = .78, *p* = .029). Overall, consistent with the previous interaction results, positive symptoms have the greatest impact on the relationship between emotion regulation and community ability when community ability is high.

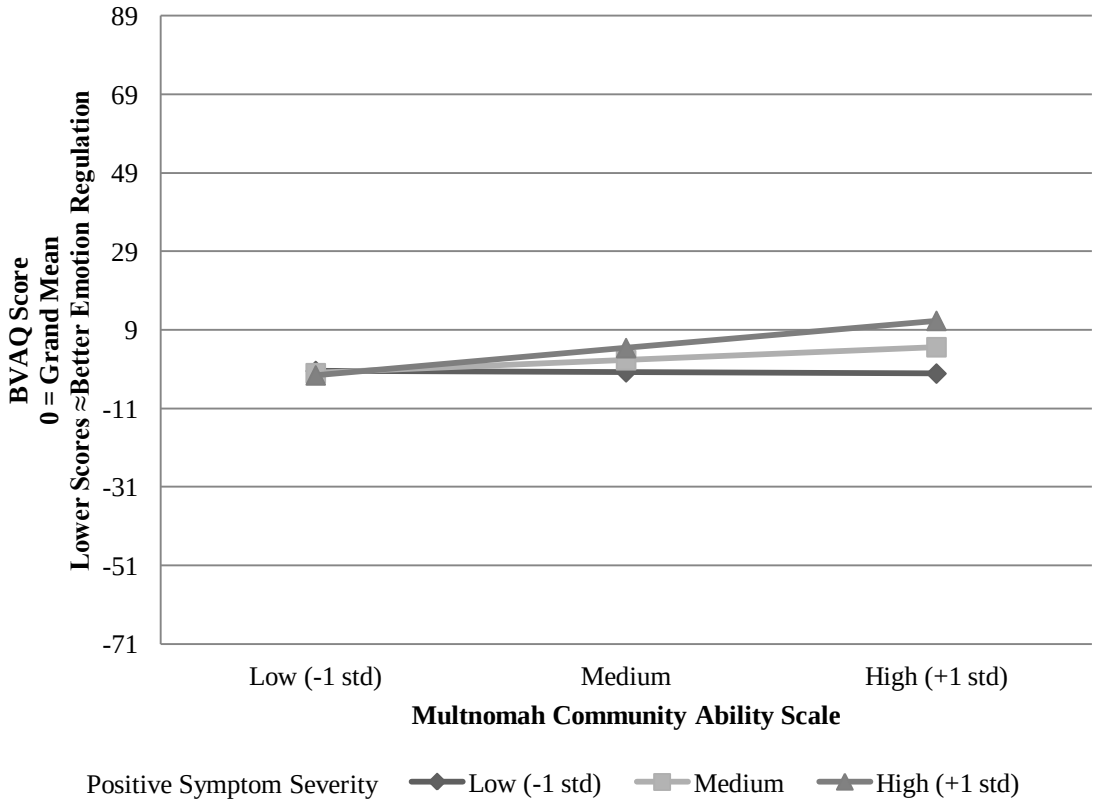

Figure 7.3. Positive Symptoms Moderate Relationship between MCAS and BVAQ
BVAQ scores are centered such that 0 represents the grand mean.
Sx = Positive Symptoms as measured by Thought Disorder Factor of BPRS.
Low = individuals with scores one standard deviation below the grand mean.
Medium = individuals with scores at the grand mean.
High = individuals with scores one standard deviation above the grand mean.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.
MCAS = Multnomah Community Ability Scale; higher scores = better community ability.

Multnomah Community Ability Scale - Self Report version as predictor. Similar to the results obtained for the SFS and MCAS as predictors, most of the multiple regression models predicting various measures of emotion regulation from the MCAS produced nonsignficant models. None of the models were improved by including positive symptom severity as a predictor nor including the interaction between positive symptom severity and community ability.

Summary. Overall, there are strong relationships between emotion regulation and social and community functioning, such that better social and community functioning is associated with better emotion regulation. This holds across multiple measures. Moreover, this relationship interacts with severity of positive symptoms. The relationship between emotion regulation and positive symptom severity becomes greater as social functioning increases. Individuals with below average social functioning have difficulties with emotion regulation, regardless of the severity of their positive symptoms; but individuals with average and above average social functioning have many fewer difficulties with emotion regulation if their symptoms are well controlled than if their symptoms are severe. In addition, the relationship between emotion regulation and social functioning becomes greater as positive symptom severity decreases. People with very severe positive symptoms have difficulties with emotion regulation, regardless of how well they function in the community; but individuals with very few positive symptoms have fewer difficulties with emotion regulation as their social functioning improves. . . . . .

Hypothesis 4: Group Treatment Participation and Emotion Regulation

Hypothesis 4a predicted that individuals who have completed group treatment modalities will have better emotion regulation than those who have not. In the total sample, 26 individuals had completed a group treatment modality (SCIT and/or IPT). A series of one-way ANOVAS were conducted to determine if scores on various measures of emotion regulation differed between these groups. As shown in Table 7.8, there were no significant differences between the groups on any of the measures of emotion regulation.

Table 7.8. *Group Means for Emotion Regulation by Completion of Group Skills Training Modalities*

	Group Skills Training Completion						
	Yes (n = 26)		No (n = 15)		ANOVA		
Emotion Regulation Measure	*M*	*SD*	*M*	*SD*	*F*	*p*	df
RACERS	110.10	16.90	104.85	13.80	< 1		
DERS	80.96	22.54	95.25	24.95	3.086	.087	1, 36
ERQ							
Reappraisal	29.92	7.41	26.92	5.28	1.591	. 215	1, 36
Suppression	16.08	5.18	17.25	2.93	< 1		
BVAQ	111.42	16.16	109.60	12.84	< 1		

Note.
Yes = Participants have completed Social Cognition and Interaction Training (SCIT) and/or Integrated Psychological Therapy (IPT).
No = Participants have completed neither SCIT nor IPT.
RACERS = Researcher and Consumer Emotion Regulation Scale; higher scores = better emotion regulation.
DERS = Difficulties with Emotion Regulation Scale; higher scores = worse emotion regulation.
ERQ = Emotion Regulation Questionnaire; higher scores = more use of that strategy.
BVAQ = Bermond-Vorst Alexithymia Questionnaire; higher scores = more alexithymia.

Hypothesis 4b predicted that individuals who have completed a group treatment modality based on the specific deficit hypothesis (e.g., SCIT) will have better emotion regulation than individuals who have completed a group treatment modality based on the generalized deficit hypothesis (e.g., IPT).

In the total sample, 2 individuals had completed only SCIT, 12 individuals had completed only IPT, and 12 individuals had completed both groups. The group of individuals who had completed a group treatment modality based on the specific deficit hypothesis was insufficient to compare to the group of individuals who had completed a group treatment modality based on the generalized deficit hypothesis.

Overall, there is little evidence to support the hypothesis that group treatment participation is associated with better emotion regulation. However, as the means were all in the expected direction, increasing statistical power may provide evidence in support of this hypothesis.

Hypothesis 5: Emotion Regulation Pathways to Clinical Outcome

A path analysis was conducted in Mplus to test the fit of the hypothesized model (see Figure 5.3) to the obtained data. Mplus allows for specification of simultaneous regression equations and therefore implies a very specific covariance matrix that better approximates the data than sequential regression equations. As composite variables were not created, it was necessary to choose individual measures to represent the constructs being studied.

The NAB-Screener total score was chosen to represent the construct of neurocognition because it measured the broadest range of neurocognitive abilities. The FEIT total score was chosen to represent the construct of emotion perception because it was the only measure of this construct included in the battery. A binary variable was created to represent participation in group treatment modalities. Selection of other representative constructs was informed by the correlation matrix as follows.

There were four predicted pathways to social functioning: from emotion perception, emotion regulation, symptom severity, and participation in group treatment modalities. There was a significant relationship between emotion perception and social functioning as measured by the SFS (r = -.48, p = .003). There were three significant relationships between emotion regulation and social functioning: between RACERS and MCAS-SR (r = .38, p = .019), between DERS and MCAS-SR (r = -.64, p < .001), and between ERQ Reappraisal and SFS (r = .49, p = .002). There were four significant relationships between symptom severity and social functioning: between positive symptoms and MCAS (r = -.37, p = .019), between total symptoms and SFS (r = -.49, p = .002), between total symptoms and MCAS (r = -.423, p = .006), and between total

symptoms and MCAS-SR (r = -.48, p = .003). Finally, there were two significant relationships between participation in group treatment modalities and social functioning, as measured by the SFS (r = .39, p = .016) and as measured by MCAS-SR (r = .33, p = .042). As there were relationships between all four constructs (i.e., emotion perception, emotion regulation, symptom severity, and participation in group treatment modalities) and SFS as a measure of social functioning, this measure was chosen to represent the construct of social functioning. ERQ Reappraisal was the only emotion regulation measure to correlate significantly with SFS, so that measure came to represent emotion regulation in the model. As total symptoms correlated with SFS whereas positive symptoms did not, total symptoms came to represent symptom severity in the model. Figure 7.4 represents the proposed model after the correlation analysis and including the instruments chosen to represent the constructs.

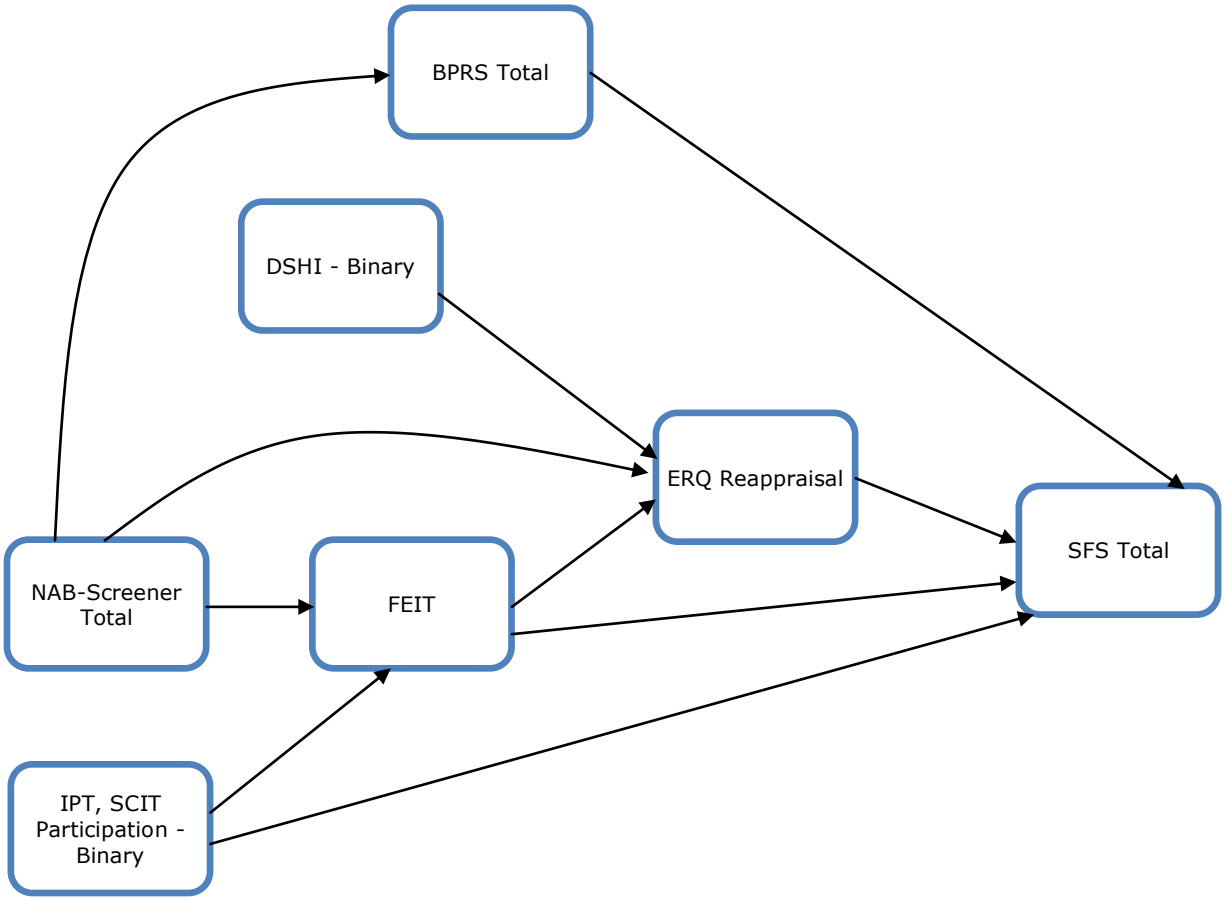

Figure 7.4. Hypothesized Path Model with Representative Measures

NAB-Screener Total = Neuropsychological Assessment Battery Screening Total Score.
FEIT = Facial Emotion Identification Test proportion correctly identified.
IPT, SCIT Participation = Group Skills Training Participation; 0 = Participation in Social Cognition and Interaction Training (SCIT) and/or Integrated Psychological Therapy (IPT); 1 = Participation in neither SCIT nor IPT.
BPRS Total = Brief Psychiatric Rating Scale Total Score.
ERQ Reappraisal = Emotion Regulation Questionnaire Reappraisal Score; higher scores = more use of reappraisal.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
SFS Total = Social Functioning Scale Total Score; higher scores = better social functioning.

Next, a path analysis was conducted to evaluate the proposed model. As shown in Table 7.9, the hypothesized model did not adequately fit the data according to any of the fit statistics. Therefore, the model was revised on the basis of conceptual and theoretical foundations and empirical guidance (i.e., the normalized residuals for the covariances,

correlations, and residual correlations of the models; modification indices; and model estimate results).

Table 7.9. *Parameters of the Hypothesized Path Model*

Independent Variable	Dependent Variable	Estimate	SE	*p*
NAB	BPRS	-.137	.160	.392
NAB	**FEIT**	**.363**	**.139**	**.009**
Group Skills Training	FEIT	.021	.150	.890
NAB	Reappraisal	.212	.160	.186
FEIT	**Reappraisal**	**-.503**	**.164**	**.002**
DSHI	Reappraisal	-.236	.142	.096
BPRS	**SFS**	**-.401**	**.118**	**.001**
FEIT	**SFS**	**-.440**	**.125**	**<.001**
Reappraisal	SFS	.240	.127	.059
Group Skills Training	SFS	.214	.122	.080
	Intercepts			
BPRS		-.101	.161	.533
FEIT		.047	.259	.855
Reappraisal		.297	.182	.103
SFS		-.281	.206	.172
	Residual Variances			
BPRS		**.981**	**.044**	**<.001**
FEIT		**.867**	**.101**	**<.001**
Reappraisal		**.707**	**.135**	**<.001**
SFS		**.467**	**.104**	**<.001**
	R^2			
BPRS		.019	.044	.669
FEIT		.133	.101	.188
Reappraisal		**.273**	**.135**	**.043**
SFS		**.533**	**.104**	**<.001**
	Model Fit			
Number of Free Parameters		18		
Loglikelihood, This model		-505.658		
Loglikelihood, Saturated (unstructured) model		-498.335		
AIC		1047		
BIC		1078		
Chi-Square Test of Model Fit	*df* = 6 (criterion: *p*>.05)	14.648		.066
Root Mean Square Error of Approximation	(criterion: <.05)	.146		
CFI	(criterion: >.95)	.851		
TLI	(criterion: >.95)	.664		
Standardized Root Mean Square Residual	(criterion: <.08)	.122		

Note.
Bold values are $p < .05$.
Estimates represent standardized estimates where appropriate.
Criteria for the model fit statistics represent the accepted standards for adequate fit.

NAB = Neuropsychological Assessment Battery Screening Total Score.
BPRS = Brief Psychiatric Rating Scale Total Score.
FEIT = Facial Emotion Identification Test proportion correctly identified.
Group Skills Training = Group Skills Training Participation; 0 = Participation in Social Cognition and Interaction Training (SCIT) and/or Integrated Psychological Therapy (IPT); 1 = Participation in neither SCIT nor IPT.
Reappraisal = Emotion Regulation Questionnaire Reappraisal Score; higher scores = more use of reappraisal.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
SFS = Social Functioning Scale Total Score; higher scores = better social functioning.

The final model is represented in Figure 7.5 and summarized in Table 7.10. It has 8 direct paths, 4 residual variances, 1 exogenous covariance, and 4 endogenous variable intercepts. The equation of this final model is as follows:

$$BPRS_i = \beta_0^{BPRS} + \beta_{DSHI}^{BPRS} DSHI_i + e_i^{BPRS}$$

$$FEIT_i = \beta_0^{FEIT} + \beta_{NAB}^{FEIT} NAB_i + e_i^{FEIT}$$

$$Reappraisal_i = \beta_0^{Reappraisal} + \beta_{DSHI}^{Reappraisal} DSHI_i + \beta_{NAB}^{Reappraisal} NAB_i + \beta_{FEIT}^{Reappraisal} FEIT_i + e_i^{Reappraisal}$$

$$SFS_i = \beta_0^{SFS} + \beta_{BPRS}^{SFS} BPRS_i + \beta_{DSHI}^{SFS} DSHI_i + \beta_{Reappraisal}^{SFS} Reappraisal_i + \beta_{FEIT}^{SFS} FEIT_i + e_i^{SFS},$$

where the variables represent the intercepts and slopes for person *i*.

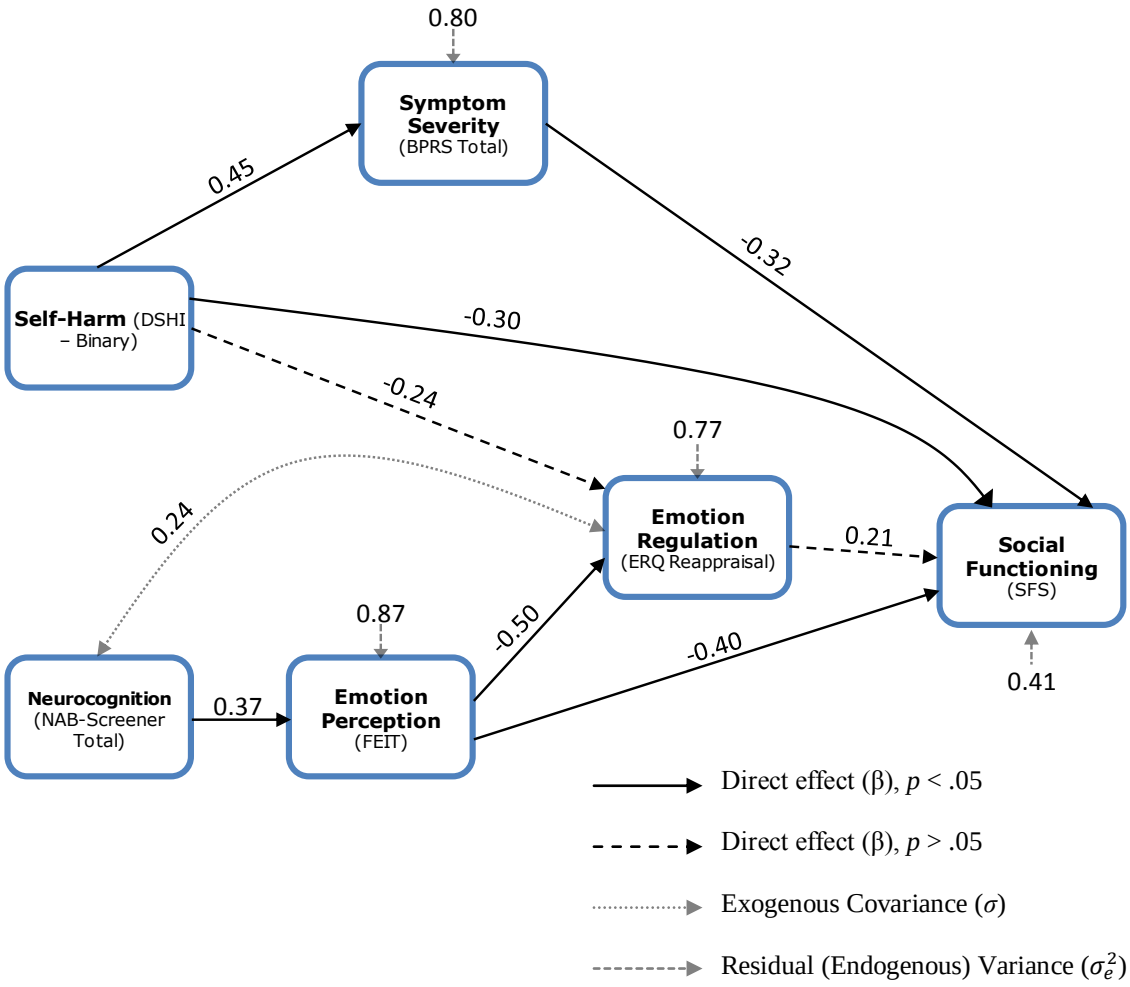

Figure 7.5. Final Path Model

NAB-Screener Total = Neuropsychological Assessment Battery Screening Total Score.
FEIT = Facial Emotion Identification Test proportion correctly identified.
BPRS Total = Brief Psychiatric Rating Scale Total Score.
ERQ Reappraisal = Emotion Regulation Questionnaire Reappraisal Score; higher scores = more use of reappraisal.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
SFS = Social Functioning Scale Total Score; higher scores = better social functioning.

Table 7.10. *Parameters of the Revised Path Model*

Independent Variable	Dependent Variable	Estimate	SE	*p*
DSHI	**BPRS**	**.445**	**.130**	**.001**
NAB	**FEIT**	**.365**	**.139**	**.009**
FEIT	**Reappraisal**	**-.502**	**.164**	**.002**
DSHI	Reappraisal	-.236	.141	.095
BPRS	**SFS**	**-.318**	**.115**	**.006**
FEIT	**SFS**	**-.398**	**.122**	**.001**
Reappraisal	SFS	.205	.120	.088
DSHI	**SFS**	**-.301**	**.122**	**.013**
	Covariance			
NAB, Reappraisal		.242	.182	.184
	Intercepts			
BPRS		**-.494**	**.182**	**.007**
FEIT		.077	.149	.608
Reappraisal		.295	.185	.111
SFS		**.301**	**.152**	**.047**
	Residual Variances			
BPRS		**.802**	**.116**	**<.001**
FEIT		**.867**	**.101**	**<.001**
Reappraisal		**.770**	**.121**	**<.001**
SFS		**.409**	**.095**	**<.001**
	R^2			
BPRS		.198	.116	.087
FEIT		.133	.101	.189
Reappraisal		.230	.121	.058
SFS		**.591**	**.095**	**<.001**
	Model Fit			
Number of Free Parameters		19		
Loglikelihood, This model		**-666.663**		
Loglikelihood, Saturated (unstructured) model		-665.524		
AIC		**1371**		
BIC		**1403**		
Chi-Square Test of Model Fit	$df = 6$ (criterion: $p>.05$)	**2.279**		**.892**
Root Mean Square Error of Approximation	(criterion: <.05)	**<.001**		
CFI	(criterion: >.95)	**1.000**		
TLI	(criterion: >.95)	**1.188**		
Standardized Root Mean Square Residual	(criterion: <.08)	**.037**		

Note.
Bold values are $p < .05$ and fit statistics that meet or exceed the accepted criterion for adequate fit.
Estimates represent standardized estimates where appropriate.

Criteria for the model fit statistics represent the accepted standards for adequate fit.
NAB = Neuropsychological Assessment Battery Screening Total Score.
BPRS = Brief Psychiatric Rating Scale Total Score.
FEIT = Facial Emotion Identification Test proportion correctly identified.
Group Skills Training = Group Skills Training Participation; 0 = Participation in Social Cognition and Interaction Training (SCIT) and/or Integrated Psychological Therapy (IPT); 1 = Participation in neither SCIT nor IPT.
Reappraisal = Emotion Regulation Questionnaire Reappraisal Score; higher scores = more use of reappraisal.
DSHI = Deliberate Self-Harm Inventory; 0 = no history of self-harm; 1 = history of self-harm.
SFS = Social Functioning Scale Total Score; higher scores = better social functioning.

In developing the final model, several pathways remained from the hypothesized model whereas several new pathways were created. The hypothesized pathways from neurocognition to social functioning via emotion perception followed from the results of Brekke et al., 2005 and were supported in the final model. The hypothesized pathway from neurocognition to social functioning via symptom severity followed from the results of Lipkovich et al., 2009. The hypothesized pathway from neurocognition to symptom severity did not remain in the model because of the absence of a correlation among these variables. However, the hypothesized pathway from symptom severity to social functioning was supported in the final model.

The hypothesized pathways from participation in group treatment modalities to emotion perception and social functioning followed from Hypothesis 4. Neither pathway was included in the final model because of a failure to account for a sufficient portion of the variance.

The hypothesized pathway from neurocognition to reappraisal followed from Hypothesis 2 but was eliminated from the model because of the absence of a correlation between the variables. However, a non-significant covariance did improve the fit of the model. Similarly, the pathways from self-harm to reappraisal and from reappraisal to

social functioning had non-significant contributions to the final model. However, both remained in the model because these pathways improved the model fit.

A couple of pathways exist in the final model that were not hypothesized: a direct pathway from self-harm to social functioning and an indirect pathway from self-harm to social functioning via symptom severity. Of note, the hypothesized pathway from emotion regulation to social functioning had a non-significant contribution to the model, but a pathway from self-harm to social functioning did significantly contribute to the model.

The final model predicts two pathways from self-harm to social functioning: a direct pathway (DSHI to SFS) and an indirect pathway via symptom severity. Symptom severity did not significantly mediate the relationship between self-harm and social functioning (p = .068).

The final model fit the data as well as a saturated (unstructured) model, which has all variance, covariances, and means estimated. The loglikelihood of the final model was -666.66, which closely approximates the loglikelihood of the saturated (unstructured) model, -665.52, indicating the final model accounts for the variances and covariances as well as the saturated (unstructured) model. The AIC was estimated to be 1371.33 and the BIC was estimated to be 1402.93, both indicating good fit. The chi-square test of model fit provides a likelihood ratio test comparing the final model to the saturated (unstructured) model and indicated that the final model fit equivalently to the saturated (unstructured) model, $\chi^2(6) = 2.279$, $p = 0.89$. The root mean square error of approximation is an index of model fit based on the approximated covariance matrix, where 0 indicates perfect fit. The estimate for the final model was less than 0.001,

indicating that the final model fits the data well. The Comparative Fit Index, comparing fit to an independence model of uncorrelated variables, was estimated to be 1.000, and the Tucker Lewis Index was estimated to be 1.188, both indicating good model fit (above 0.95). Finally, the standardized root mean square residual provides the average standardized difference between the observed correlations and the model-predicted correlations. The estimate was 0.037, which is less than the criterion of 0.08, indicating the model fit the data well. In sum, all fit indices indicated the model fit the data well.

In the final model, the unstandardized intercepts represent the expected means, centered at 0. Therefore, the mean emotion perception for individuals with average reappraisal, symptom severity, and social functioning is expected to be 0.28 units above average; the mean reappraisal for individuals with average emotion perception, symptom severity, and social functioning is expected to be 2.05 units above average; the mean symptom severity for individuals with average emotion perception, reappraisal, and social functioning is expected to be 4.2 units below average; and the mean social functioning for individuals with average emotion perception, reappraisal, and symptom severity is expected to be 5.17 units above average.

The standardized pathways represent the slopes for predicting the dependent variables. A significant pathway from self-harm to symptom severity was found, such that for every one standard deviation increase in self-harm, symptom severity was expected to be higher by 0.45 standard deviations. The unstandardized coefficient indicates that individuals who have histories of self-harm have an average symptom severity 7.59 units higher than individuals who have no history of self-harm. A significant pathway from neurocognition to emotion perception was found, such that for

every one standard deviation increase in neurocognition, emotion perception was expected to be higher by 0.37 standard deviations. A significant pathway from emotion perception to reappraisal was found, such that for every one standard deviation increase in emotion perception, use of reappraisal was expected to be lower by 0.50 standard deviations, for individuals with no history of self-harm; and a non-significant pathway from self-harm to reappraisal was found, such that for every one standard deviation increase in self-harm, use of reappraisal was expected to be lower by 0.24 standard deviations, for individuals with an average emotion perception ability. The unstandardized coefficient indicates that individuals who have histories of self-harm have an average use of reappraisal 3.29 units lower than individuals who have no history of self-harm. Three significant pathways to social functioning were found, from emotion perception, symptom severity, and self-harm; and one non-significant pathway was found, from reappraisal. For every one standard deviation increase in emotion perception, social functioning was expected to be lower by 0.40 standard deviations, for individuals who have never self-harmed and who have average symptom severity and use of reappraisal. For every one standard deviation increase in symptom severity, social functioning was expected to be lower by 0.32 standard deviations, for individuals who have never self-harmed and who have average emotion perception and use of reappraisal. For every one standard deviation increase in self-harm, social functioning was expected to be lower by 0.30 standard deviations, for individuals who have average emotion perception, symptom severity, and use of reappraisal. The unstandardized coefficient indicates that individuals who have histories of self-harm have an average social functioning 10.38 units lower than individuals who have no history of self-harm, among

those with average emotion perception abilities, symptom severity, and use of reappraisal. Finally, for every one standard deviation increase in use of reappraisal, social functioning was expected to be non-significantly higher by 0.21 standard deviations, for individuals who have never self-harmed and who have average emotion perception and symptom severity.

The model explained 13.3% of the variance of emotion perception abilities; 19.8% of the variance in symptom severity; 23.0% of the variance in use of reappraisal; and 59.1% of the variance in social functioning.

Overall, the hypothesis that emotion regulation can be incorporated in pathways to clinical outcome was supported.

CHAPTER 8

DISCUSSION

Results Summary

Emotion dysregulation was hypothesized to be associated with more severe positive symptoms, poorer neurocognitive functioning, and poorer social and community functioning. The results were mixed across the various assessments of these domains.

Difficulties with processing emotions were observed in the sample, consistent with previous research. These included difficulties expressing emotions, identifying facial emotions, and regulating emotions. Many participants presented with flat affect, indicating a deficit in expressing emotions. However, the sample did not necessarily indicate reduced emotional experience, as an assessment of alexithymia produced a normal distribution comparable to non-clinical populations. Consistent with previous research, participants also had difficulty perceiving facial emotions. Whereas they were relatively successful at identifying positive facial expressions such as happiness, they had the most difficulty with negative facial expressions, particularly ashamed faces. Participants also demonstrated difficulty regulating emotions on a variety of assessments. Of interest, they reported relying more on reappraisal strategies than suppression strategies to regulate their emotions. This appears inconsistent with the literature, which suggests that this population has a preference for strategies like suppression. However, the scores obtained for use of reappraisal and suppression in this study are consistent with earlier studies (e.g., Livingstone et al., 2009; van der Meer et al., 2009). It is important to note that, like healthy controls, this population tends to use reappraisal more than suppression to regulate their emotions. However, this population tends to use reappraisal

less than controls and suppression *more than* controls. Thus, in both populations, the overall preference is for reappraisal, although healthy controls have a much stronger preference for that particular regulatory strategy. This study is consistent with those results.

The possibility remains that participants use emotion regulation strategies to alter their perception of their emotional experiences, thereby perceiving their abnormal emotional experiences as normal. However, the obtained results suggest that the emotion paradox is present in this population (i.e., the population tends to demonstrate reduced emotional expressiveness while reporting normal emotional experiences). The sample included in this study had relatively few psychiatric symptoms, including mood symptoms. It remains unclear whether a pathological excess of negative emotion (e.g., as seen in affective disorders) is associated with underutilization of strategies to induce positive emotional changes or with overuse of strategies to induce negative emotional changes. This important question about how this population regulates emotions during times of affective crisis is a critical finding that will have implications for treatment development.

Contrary to previous research (e.g., Gross & John, 2003; Henry et al., 2008), use of suppression as a regulatory strategy was significantly associated with neither positive symptom severity nor global psychiatric symptom severity. Only one measure of emotion regulation (DERS) predicted positive symptom severity, such that greater emotion dysregulation was associated with more severe positive symptoms. Half of the measures of emotion regulation predicted global psychiatric symptom severity, such that greater emotion dysregulation was associated with more severe psychiatric

symptoms. The evidence was not strong enough to specifically link suppression as a regulatory strategy to psychiatric symptom severity; however, there was some evidence that there may be a relationship between psychiatric symptom severity and broader emotion dysregulation.

In general, the sample had few psychiatric symptoms, especially positive symptoms, and this may have limited the associations that could be found between symptoms and emotion regulation. On the other hand, a sample whose symptoms are well controlled with pharmacological and behavioral interventions may be representative of the larger population, particularly those engaged in services founded on principles of rehabilitation and recovery. In that case, there may be a weaker relationship between these functional domains.

Overall, there were few relationships between emotion regulation and neurocognitive abilities. A measure of attention was associated with use of reappraisal and suppression as regulatory strategies, and a measure of executive functioning was marginally associated with use of suppression, but there were no relationships between memory and any of the measures of emotion regulation. That there were no relationships between memory impairment and reappraisal is consistent with previous research (e.g., Gross & John, 2002; Richards & Gross, 2000). Of note, none of the measures of global emotion dysregulation (e.g., RACERS, DERS) significantly correlated with any of the measures of attention, memory, or executive functioning, suggesting that a relationship between these constructs might be specific to the regulatory strategies used and may not extend to more global indicators of emotion

regulation and dysregulation. In general, overall neurocognition appears to explain very little of the variance in emotion regulation in this population.

With respect to social and community functioning, individuals with poorer emotion regulation, as measured by a variety of instruments, tended to have poorer self-reported social functioning. Consistent with previous work (e.g., John & Gross, 2004), greater reliance on reappraisal as a regulatory strategy was associated with better social functioning, whereas no relationship was found between use of suppression and social functioning. Perhaps most interesting about these findings is that the strongest relationships between emotion dysregulation and social functioning were on the self-report measures of social functioning (i.e., SFS and MCAS-SR). The measure of social functioning completed by practitioners familiar with the individuals' functioning in the community did not significantly relate to any of the measures of emotion regulation. This could reflect an artifact of the instruments used or could represent a modicum of insight on the part of the participants regarding the impact of emotion regulation on social and community functioning.

It was also hypothesized that positive symptom severity would account for some of the variance in the relationship between emotion regulation and social functioning. A few of the models indeed indicated this was the case. The interaction models indicated that positive symptoms have the greatest impact on emotion regulation when social functioning is above average and that social functioning has the greatest impact on emotion regulation when positive symptoms are below average. This suggests that individuals with SMI are best able to regulate their emotions during times of few psychotic symptoms and good social functioning. . . . . .

Finally, contrary to what was hypothesized, having participated in group skills training was not related to better emotion regulation. The failure to find a relationship between skills training and emotion regulation may be attributable to insufficient statistical power. Indeed, the means were in the expected direction, indicating that group skills training may have an impact on improving emotion regulation or the skills required for more effectively regulating emotions.

A path model summarized the relationships among the constructs of interest. Consistent with the theory that functional neurocognition is a prerequisite for any more molar biosystemic domain (including emotion regulation), the path model begins with neurocognition. The hypothesized pathway from neurocognition to emotion regulation did not remain in the model because of the absence of a correlation among these domains. However, that a covariance between these constructs improved the fit of the model indicates that neurocognitive abilities do account for some of the variance in use of reappraisal as a regulatory strategy, which may be mostly attributable to variance in the attention domain. These results are supportive of developing interventions based on the specific deficit hypothesis. Because neurocognition only accounted for a small proportion of the variance in emotion regulation, interventions based on the generalized deficit hypothesis may only result in slight improvements in emotion regulation. These results suggest that a more targeted intervention specifically aimed at remediating emotion processing deficits may demonstrate greater improvements.

The pathway from emotion perception to emotion regulation with a negative regression weight is inconsistent with the emotion paradox, which implies that these constructs might have a positive relationship.

Non-significant pathways from self-harm to social functioning via emotion regulation remained in the model because of contributions to overall model fit, indicating that self-harm accounts for some of the variance in emotion regulation strategies, and that emotion regulation strategies in turn account for some of the variance in social functioning.

In addition, pathways exist in the final model that were not hypothesized. A path between self-harm and social functioning via symptom severity is consistent with the regression models reported above indicating that symptom severity accounts for some of the variance in the relationship between emotion regulation and social functioning. However, symptom severity did not significantly mediate the relationship between self-harm and social functioning, owing to its accounting for a small proportion of the variance. Finally, the hypothesized pathway from emotion regulation to social functioning had a non-significant contribution to the model, but a pathway from self-harm to social functioning did significantly contribute to the model. It may be that a behavioral proxy of emotion regulation (i.e., self-harm) better accounts for variance in social functioning than the cognitive strategies used to regulate emotions (i.e., reappraisal).

Overall, in the final model, better neurocognitive abilities were associated with better emotion perception abilities. However, better emotion perception was associated with poorer emotion regulation (i.e., less use of reappraisal) and poorer social functioning. These unanticipated relationships are in fact among the strongest relationships in the model. A history of self-harm predicted more severe psychiatric symptoms, poorer emotion regulation, and poorer social functioning. The model

indicates that more severe psychiatric symptoms and poorer emotion regulation both predict poorer social functioning, but recall that an interaction was found among these domains that indicates this is a complicated relationship. Such interactions are not adequately represented in the path model.

Limitations

There are two major limitations to this study: the sample size and the assessment instruments. The failure to consistently find the hypothesized relationships between the constructs of interest is likely due in no small part to insufficient statistical power. This is particularly true for the hypotheses regarding the impact of group skills training on emotion regulation. In such a heterogeneous population, a larger sample size will more effectively stabilize estimates of the mean and variance, resulting in more accurate representations of the relationships among the constructs of interest. The multivariate interactions investigated in this study demonstrate that there are important relationships among subgroups of individuals and measures, but insufficient power conceals their significance.

However, there is also a significant problem in validly and reliably measuring these constructs in this population, in particular the construct of emotion regulation. There is only one measure of emotion regulation specifically designed for this population (RACERS), and it has major limitations owing to its being recently developed and not having undergone the rigorous psychometric testing and revision necessary to improve its validity and reliability. The other measures of emotion regulation included in the battery have undergone more rigorous psychometric testing

but were not developed for the SMI population and therefore may not accurately measure emotion regulation in that population.

Further understanding of emotion regulation in SMI will require that methods are developed for measuring the latent construct validly and reliably with enough sensitivity to detect longitudinal changes over the course of the illness. A conceptualization of emotion regulation as a multidimensional and dynamic construct requires that measurement also be multidimensional and dynamic. Integrating measures of multiple aspects of emotion regulation, such as behavioral, cognitive, and emotional measures from multiple sources (e.g., self-report, clinicians, family, direct observation, etc.), would be consistent with this approach.

In addition to those limitations, the statistical analyses, including the path analysis, assumed all variables to be continuous, multivariate normal, and measured with perfect reliability. In reality, none of these conditions were met by the data. Therefore, the analysis presented in this manuscript is intended to be an initial hypothesis reflecting the relationships among the variables of interest. Further analyses using more sophisticated statistical techniques that more accurately represent the limitations of the data will result in a more accurate representation of the connections between and among the functional domains in SMI.

Conclusions

This study evaluated the multivariate relationships between emotion regulation and other biosystemic domains in outpatients with SMI. The results indicate that emotion regulation is a central domain in understanding neurocognitive functioning, social cognitive functioning, psychiatric symptom severity, and social functioning in this

population. Many questions remain unanswered, such as more specifically detailing the nature of the relationships among these domains, including potential interactions among the domains that more accurately represent variance. The population of interest in this study is heterogeneous, and the domain of emotion regulation is heterogeneous. The inconsistencies among the results are indicative of the many individual differences in abilities in this domain.

Although the sample reported using antecedent-focused strategies to regulate their emotions, their preference for these strategies remained below what is found in healthy controls. That they tend to also rely on more maladaptive skills such as response-focused strategies indicates that skill training may be a beneficial treatment target. Nevertheless, it is critical to consider that no single regulatory strategy is inherently adaptive or maladaptive. Therefore, that response-focused strategies tend to be maladaptive in healthy controls does not necessarily indicate this is the case in the SMI population or in any particular individual. This highlights the need for valid assessment.

The path model summarizing the results is consistent with the biosystemic theory of serious mental illness, which posits that the various processes are independent yet casually linked throughout the biosystem. Emotional responses entail coordinated and interrelated systemic changes in thoughts, behavior, and physiology. Thus, dysfunctional emotional responses will likely require intervention throughout the biosystem. The independence of the functional domains, as highlighted by their largely orthogonal relationships in this study, requires individual attention in clinical assessment and compels the development of separate, specific treatment and

rehabilitation interventions. Resolving questions such as the types of emotion regulation that are ongoing during affective crises in this population will also inform the skills such training modalities should target. An effective emotion regulation skills training modality would begin with assessment of adaptive strategies and follow with improving performance of skills individuals already possess while building their competence in alternative antecedent-focused strategies and their flexibility of emotional responses. While innovative and comprehensive modalities are being developed, utilizing existing treatment strategies in the psychiatric rehabilitation toolkit such as exercise and relaxation may prove beneficial.

This research joins previous exploratory research in finding that schizophrenia and related disorders have a major affective component, despite their not being classified as affective disorders. The deficits and impairments in SMI extend to regulating emotional experiences, which has implications independent functioning, particularly interpersonal and occupational functioning. Further characterization of the emotional component of serious mental illnesses will inform treatment development and thereby contribute to the recovery of this population.

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FOOTNOTES

[1] It is important to note that some emotion regulation strategies may overlap with social skills, interpersonal problem solving skills, and so on. Indeed, stress management curricula often include training in those types of skills in addition to skills more obviously linked to managing stress and emotions. The distinction between stress management and emotion regulation is not clearly articulated in the literature. However, it can be argued that stress management is a broader term, including general lifestyle management as well as managing emotional reactions to environmental stressors, whereas emotion regulation refers specifically to strategies intended to change the type, timing, experience, or expression of emotions. This dissertation focuses on emotion regulation, as opposed to stress management.

[2] Note that neurocognition here refers to neuropsychological processes, or the poorly understood processes responsible for performance on particular neuropsychological tasks. In other disciplines of psychology, neurocognition refers more specifically to molecular cognitive processes and their brain-based mechanisms.

APPENDIX

Appendix A: Correlation Matrix for Emotion Regulation by Symptoms, Neurocognition, Social Cognition, and Social Functioning

	RACERS	DERS							ERQ		BVAQ						
	Total	Nonacceptance	Goals	Awareness	Strategies	Clarity	Impulse	Total	Reappraisal	Suppression	Verbalizing	Fantasizing	Emotionalizing	Analyzing	Affective	Cognitive	Total
Symptoms																	
BPRS																	
Total	-.285	**.440****	**.471****	**.346***	**.548****	**.378***	**.398***	**.596****	-.206	.144	.255	-.307	**-.518****	.143	**-.480****	.259	-.078
Neurocognition																	
Trails																	
A	-.134	-.207	.031	-.084	-.079	.130	.223	-.025	**.320***	.300	-.130	.194	.037	.003	.163	-.068	.043
B	.075	**-.389***	-.137	-.141	-.315	-.119	-.021	-.282	.095	-.005	**-.388***	.291	**.371***	-.071	**.389***	-.248	.032
NAB																	
Attention	.052	.206	-.002	.027	-.033	.021	**-.344***	-.016	-.080	-.207	.305	.078	.077	-.066	.095	.155	.194
Lang	.256	-.053	-.068	-.178	-.183	-.088	**-.438****	-.225	-.016	-.128	.137	-.012	.021	-.112	.001	-.076	-.065
Memory	.102	-.083	.029	-.043	-.117	.030	-.212	-.101	.239	-.082	.219	-.148	.036	-.084	-.093	.083	.013
Spt Abil	.209	-.184	-.080	-.176	-.171	-.259	**-.358***	-.271	-.087	-.125	-.103	-.163	.189	-.098	-.031	-.291	-.270
Exe Fnc	.072	.296	.089	.064	.029	-.052	-.207	.068	-.214	-.277	.066	**-.312***	.059	-.015	-.204	.014	-.116
Total	.168	.073	.016	-.051	-.093	-.075	**-.399***	-.111	-.039	-.188	.196	-.168	.085	-.104	-.084	-.016	-.067
Social Cognition																	
FEIT																	
Total	.286	-.130	-.066	-.123	-.229	**-.363***	-.318	-.271	**-.378***	**-.451****	-.141	-.114	.046	-.276	-.063	-.308	-.305
BFRT																	
Total	**.331***	-.194	-.118	-.166	-.039	-.313	**-.345***	-.243	-.036	-.294	.046	-.032	-.031	-.168	-.039	-.128	-.135

	RACERS	DERS							ERQ		BVAQ						
	Total	Nonacceptance	Goals	Awareness	Strategies	Clarity	Impulse	Total	Reappraisal	Suppression	Verbalizing	Fantasizing	Emotionalizing	Analyzing	Affective	Cognitive	Total
	Social Functioning																
SFS																	
SocEng	.128	-.257	**-.354***	-.304	-.244	**-.412***	-.222	**-.389***	.065	-.139	-.287	.082	.232	.076	.171	-.320	-.167
IntCom	.121	**-.344***	**-.351***	-.211	**-.367***	**-.442****	-.260	**-.445****	.165	-.005	**-.483****	.010	.188	.008	.096	**-.437****	-.317
IndepC	.119	-.211	**-.426****	-.023	-.170	-.062	-.319	-.271	.050	-.167	.179	.196	.212	.160	.246	.074	.224
IndepP	.049	-.125	**-.383***	-.057	-.175	-.037	-.223	-.224	.096	-.258	**-.372***	-.049	.160	.021	.039	-.219	-.164
Recreat	.173	-.155	-.039	-.198	-.050	-.290	-.054	-.164	.260	-.188	**-.445****	-.098	-.046	-.247	-.095	**-.327***	**-.345***
Prosoc	-.037	-.122	.132	-.211	-.050	.093	.122	-.026	**.523****	.293	-.107	.117	.141	-.139	.153	-.052	.054
Employ	-.186	.300	-.098	.146	.137	-.080	-.069	.108	.306	.045	.092	-.144	.156	.068	-.033	.005	-.017
Total	.083	-.241	**-.330***	-.232	-.207	-.184	-.212	-.313	**.492****	-.058	**-.323***	.071	.276	-.057	.183	-.259	-.106
MCAS																	
Health	.146	-.105	-.133	.002	-.085	-.195	-.254	-.164	.152	-.157	-.021	.206	**.500****	.060	**.396***	.074	**.312***
Adapt	.229	-.037	-.088	-.221	**-.339***	-.237	-.285	-.277	-.022	-.295	-.102	.012	**.309***	-.132	.158	-.131	-.014
SocSk	-.211	-.029	-.158	-.317	-.154	-.033	-.044	-.164	**.385***	.175	-.251	-.044	**.379***	.111	.151	-.030	.068
Behavior	.276	-.294	-.077	-.233	-.316	-.301	-.198	**-.332***	.150	-.266	-.188	.223	**.432****	-.081	**.376***	-.077	.169
Total	.076	-.125	-.162	-.271	-.276	-.220	-.235	-.291	.258	-.119	-.193	.114	**.539****	.013	**.346***	-.042	.180
MCAS-Self Report																	
Health	.261	**-.393***	**-.420****	-.120	**-.452****	-.295	**-.422****	**-.489****	.121	-.038	-.286	.026	.291	-.025	.157	-.315	-.172
Adapt	**.416****	-.216	-.179	-.138	-.182	-.100	**-.357***	-.267	.085	-.148	-.190	.069	.045	-.171	.073	-.283	-.199
SocSk	.205	**-.602****	-.298	**-.345***	**-.437****	-.309	**-.336***	**-.547****	.286	.001	**-.531****	.170	.117	.024	.182	-.272	-.118
Behavior	**.330***	**-.430****	-.220	**-.456****	**-.432****	-.284	**-.533****	**-.542****	.149	.004	-.213	.201	.094	-.117	.194	-.290	-.126
Total	**.380***	**-.586****	**-.408***	**-.337***	**-.529****	**-.352***	**-.536****	**-.639****	.234	-.053	**-.456****	.147	.211	-.068	.209	**-.392***	-.205

Note: Bold values are $p < .05$.
** Correlation is significant at the 0.01 level.
* Correlation is significant at the 0.05 level.

Spt Abil = Spatial Ability; Exe Fnc = Executive Functions; SocEng = Social Engagement; IntCom = Interpersonal Communication; IndepC = Independence – Competence; IndepP = Independence – Performance; Recreat = Recreation; Prosoc = Prosocial Behavior; Employ = Employment; Adapt = Adaptation; SocSk = Social Skills

Appendix B: Correlation Matrix for Neurocognition by Symptoms, Social Cognition, and Social Functioning

	Trails		NAB-Screener					
	A	B	Attention	Language	Memory	Spatial Ability	Exec Functions	Total
Symptoms								
BPRS								
Total	.037	-.313	.012	-.076	-.058	-.224	-.060	-.109
Social Cognition								
FEIT								
Total	-.167	.021	.154	.307	-.026	**.421****	.171	.308
BFRT								
Total	-.256	-.214	.277	.263	.019	**.391***	.210	**.328***
Social Functioning								
SFS								
Social Engagement	.120	.262	-.216	-.019	.136	.299	-.101	.019
Interpersonal Communication	.137	.210	**-.322***	-.302	-.036	.305	-.153	-.148
Independence – Competence	-.124	-.151	.213	.106	.034	-.002	.058	.114
Independence - Performance	-.240	-.027	-.036	-.121	.015	.057	.255	.016
Recreation	.210	.187	-.312	**-.393***	-.218	-.279	-.123	**-.375***
Prosocial Behavior	**.624****	**.377***	**-.328***	-.293	.019	-.217	-.239	-.299
Employment	-.048	-.332	.145	-.147	-.086	-.083	.293	.014
Total	.287	.218	-.236	**-.336***	-.036	-.121	-.024	-.231
MCAS								
Health	-.235	-.142	.196	.017	-.182	.265	.244	.139
Adaptation	-.086	-.076	.200	.180	.145	.084	**.333***	.224
Social Skills	.102	.216	-.005	-.041	-.077	.097	.092	.011
Behavior	-.089	-.107	.007	-.229	-.251	.163	.203	-.054
Total	-.085	-.013	.124	-.021	-.123	.201	.270	.102

	Trails		NAB-Screener					
	A	B	Attention	Language	Memory	Spatial Ability	Exec Functions	Total
MCAS-SR								
Health	.000	.172	-.015	.021	.200	.302	-.038	.113
Adaptation	**-.347***	-.198	.196	.131	.121	.207	.318	.278
Social Skills	.147	.287	-.214	-.228	-.037	.104	-.152	-.158
Behavior	.122	-.003	.069	.202	.218	**.404***	.014	.223
Total	.003	.151	-.039	-.019	.146	.314	-.002	.094

Note: Bold values are $p < .05$.
** Correlation is significant at the 0.01 level.
* Correlation is significant at the 0.05 level.
Exec Functions = Executive Functions

Appendix C: Correlation Matrix for

Symptoms and Social Cognition by Social Functioning

	Symptoms	Social Cognition	
	BPRS	FEIT	BFRT
Social Functioning Measure			
SFS			
Social Engagement	**-.420****	.021	-.060
Interpersonal Communication	**-.368***	-.065	.162
Independence – Competence	-.294	-.204	-.066
Independence - Performance	-.110	-.212	.133
Recreation	-.188	**-.485****	-.144
Prosocial Behavior	**-.391***	-.071	.150
Employment	-.107	-.272	.061
Total	**-.491****	**-.475****	.010
MCAS			
Health	**-.503****	.140	.135
Adaptation	-.043	.195	-.106
Social Skills	-.267	.164	.002
Behavior	**-.434****	.217	.009
Total	**-.425****	.230	.022
MCAS-SR			
Health	**-.363***	-.008	.005
Adaptation	-.201	.111	.204
Social Skills	**-.512****	-.309	.061
Behavior	-.168	.028	.026
Total	**-.475****	-.109	.087

Note: Bold values are $p < .05$.

** Correlation is significant at the 0.01 level.

* Correlation is significant at the 0.05 level.