Emotion Perception in Schizophrenia:

A Functional Connectivity Study

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THESIS

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OAB

CONTRIBUTION OF AUTHORS

Chapter 1 is a literature review that places my dissertation question in the context of the larger field and highlights the significance of my research question. Chapter 2 includes methodology from a published manuscript (Bjorkquist & Herbener, 2013) for which I was the primary author and major driver of the research. Dr. Ellen Herbener assisted me in the experiment and contributed to the writing of the manuscript. Chapter 3 represents study results of this dissertation. Chapter 4 represents my synthesis of the research presented in this dissertation and my overarching conclusions. The future directions of this field and this research question are discussed.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

ACC	Anterior Cingulate Cortex
AFNI	Analysis of Functional Neuroimages
β	Unstandardized Beta
BOLD	Blood Oxygen Level Dependent
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, 4th Edition
fMRI	Functional Magnetic Resonance Imaging
HC	Healthy Control
IAPS	International Affective Picture System
M	Mean
mPFC	Medial Prefrontal Cortex
MRI	Magnetic Resonance Imaging
n	Number in Group
n.s.	Nonsignificant
OFC	Orbitofrontal Cortex
p	Probability
PANSS	Positive and Negative Syndrome Scale
PPI	Psychophysiological Interaction
QLS	Heinrichs-Carpenter Quality of Life Scale
QLS-SF	Heinrichs-Carpenter Quality of Life: Social Functioning
QLS-WF	Heinrichs-Carpenter Quality of Life: Work Functioning
QLS-M	Heinrichs-Carpenter Quality of Life: Motivation
ROI	Region-of-Interest

SD	Standard Deviation
SZ	Schizophrenia
UIC	University of Illinois at Chicago
WASI	Wechsler Abbreviated Scale of Intelligence
WRAT	Wide Range Achievement Test, 3rd Edition

SUMMARY

Individuals with schizophrenia evidence impairment in multiple aspects of emotional functioning, including emotion expression, perception, and recognition. Neuroimaging research has identified abnormalities in the amygdala as an etiological factor underlying affective impairment in this population. However, the exact nature of amygdala dysfunction remains unclear. The current study utilized psychophysiological interaction analyses to examine functional connectivity between the amygdala and medial prefrontal cortex (mPFC) during an emotion perception task. Participants with schizophrenia (SZ) and demographically-matched comparison participants (HC) viewed and rated positive, negative, and neutral images from the International Affective Picture System (IAPS) library while undergoing functional neuroimaging. Results revealed a significant group difference in right amygdala-mPFC connectivity during perception of negative compared to neutral images. Specifically, HC participants demonstrated positive functional coupling between the amygdala and mPFC, consistent with co-active processing of salient information. In contrast, SZ participants evidenced negative functional coupling, consistent with top-down inhibition of the amygdala by the mPFC. A significant positive correlation between connectivity strength during negative image perception and clinician-rated social functioning was observed in SZ participants. Similar patterns of functional coupling were observed during positive image perception, though the between-group difference failed to reach statistical significance. These results suggest that emotional dysfunction in schizophrenia may be due, in part, to abnormal interactions between the amygdala and mPFC during perception of emotional stimuli. Disturbances in functional connectivity during early stages of emotion processing could lead to impairment in higher order aspects of emotion processing, such as emotion regulation.

I. INTRODUCTION

Emotional dysfunction has long been recognized in schizophrenia, since Kraepelin (1919/1971) and Bleuler (1911) described disturbances in affect. Affective impairment is considered a core feature of the illness, with affective flattening listed in the *DSM-IV* as a negative symptom of schizophrenia (American Psychiatric Association, 1994). Individuals with schizophrenia show deficits in emotional expression, perception and recognition, including identification, discrimination, and intensity ratings of facial emotion expressions (Kring & Moran, 2008; Tremeau, 2006). They also report increased levels of physical and social anhedonia (e.g., Blanchard, Mueser, & Bellack, 1998; Herbener, Rosen, Khine, & Sweeney, 2007; Katsanis, Iacono, & Beiser, 1990). Despite blunted emotional expression and self-reported anhedonia, individuals with schizophrenia report the same emotional responses to affective stimuli in the moment (e.g., Aghevli, Blanchard, & Horan, 2003; Curtis, Lebow, Lake, Katsanis, & Iacono, 1999; Herbener, Song, Khine, & Sweeney, 2008; Kring & Moran, 2008). Gaining a better understanding of this emotional experience-perception disconnect is a current schizophrenia research goal and aim of the current study.

A. Amygdala Dysfunction in Schizophrenia

Neuroimaging techniques have been used to investigate the neural underpinnings of emotional dysfunction in schizophrenia, revealing mixed results. Studies have typically focused on the amygdala given its role in processing emotional information, particularly negative affect (LeDoux, 2000; Phan, Wager, Taylor, & Liberzon, 2002). Anatomical studies note decreased volume (Ellison-Wright, Glahn, Laird, & Thelen, 2008; Joyal et al., 2003) and altered gene expression (Weidenhofer, Bowden, Scott, & Tooney, 2006) in the amygdala in schizophrenia. Functional studies reveal abnormal amygdala response during emotion perception and

recognition, with mixed findings regarding direction of activation differences. Some studies have reported amygdala hypoactivation (e.g., Gur et al., 2002; Hempel, Hempel, Schonknecht, Stippich, & Schroder, 2003; Phillips et al., 1999; Schneider et al., 1998) while others have found amygdala hyperactivation (Holt et al., 2006; Kosaka et al., 2002; Sanjuan et al., 2007; Taylor, Phan, Britton, & Liberzon, 2005) in people with schizophrenia compared to controls. Similarly, some studies report group differences in left amygdala (Gur et al., 2002; Paradiso et al., 2003; Williams et al., 2004), while others report differences in right amygdala activity (Johnston, Stojanov, Devir, & Schall, 2005; Takahashi et al., 2004). Further complicating matters, some studies have found no group differences in amygdala response to emotional stimuli (Anticevic, Repovs, & Barch, 2012a; Dowd & Barch, 2010). Meta-analyses examining amygdala activation in people with schizophrenia indicate modest amygdala hypoactivation in response to aversive stimuli (Anticevic et al., 2012b; Li, Chan, McAlonan, & Gong, 2010; Taylor et al., 2012).

Several factors likely contribute to differing findings regarding amygdala dysfunction in schizophrenia. First, studies differ in patient characteristics and there is evidence that some amygdala abnormalities may be specific to paranoid patients (Russell et al., 2007). Second, studies differ in control condition, with some comparing emotional to neutral stimuli and others comparing emotional stimuli to baseline brain activity. Studies investigating brain response to neutral faces have found hyperactivation in those with schizophrenia in the amygdala (Hall et al., 2008; Holt et al., 2006; Surguladze et al., 2006), suggesting that decreased amygdala response to emotional versus neutral stimuli may reflect heightened response to neutral stimuli. Third, task differences in cognitive load may also contribute to mixed amygdala findings. Most tasks require at least modest cognitive effort (e.g., recognizing, labeling, or matching emotions), and increased cognitive engagement is known to suppress amygdala function in healthy individuals (Pessoa,

Padmala, & Morland, 2005). Moreover, a recent emotion study using minimal cognitive load reported no group differences in amygdala recruitment but abnormal amygdala-prefrontal connectivity in participants with schizophrenia (Anticevic et al., 2012a). Thus, the literature largely points to amygdala dysfunction underlying emotion impairment in schizophrenia, but the exact nature of amygdala abnormality is unclear.

B. Functional Connectivity

The amygdala is a well-connected structure, with known anatomical and functional connections throughout the brain (Kim et al., 2011b). Therefore, emotion deficits in schizophrenia may not be due to isolated amygdala dysfunction, per se, but rather disruptions to functional connections in amygdala circuitry. Of particular relevance to schizophrenia, functional disconnection has been proposed as a key etiologic factor in schizophrenic illness (Friston, 1998). Recent advances in statistical methodology have allowed for the examination of functional connectivity between brain structures, i.e. the statistical associations between blood oxygen level dependent (BOLD) intensity time series in different brain regions. Accordingly, neuroimaging research has recently shifted its focus from activity in isolated brain regions to using a network approach, investigating how brain regions interact with one another. Functional connectivity approaches can be applied to resting state brain activity or activity associated with different task conditions. For example, psychophysiological interaction (PPI) analyses allow for comparison of functional connectivity between specified brain regions in different task conditions. PPI analyses can further be applied to the comparison of task-related connectivity between healthy controls and psychiatric populations. The present study employed PPI analyses to investigate functional connectivity in an emotion perception task in individuals with schizophrenia compared to healthy controls.

1. Amygdala-mPFC connectivity

The amygdala shares connections with a number of cortical and subcortical regions that are involved in emotion processing including frontal, insular, temporal, and occipital cortices (Amaral, Behniea, & Kelly, 2003; Amaral & Price, 1984) as well as striatum (Russchen, Bakst, Amaral, & Price, 1985). Here we focus on connections between the amygdala and medial prefrontal cortex (mPFC) including the anterior cingulate cortex (ACC), given the well-established mPFC and ACC abnormalities in schizophrenia (Goghari, Sponheim, & MacDonald, 2010; Levitt, Bobrow, Lucia, & Srinivasan, 2010) and importance of mPFC/ACC circuits for emotion appraisal, expression, and regulation (Etkin, Egner, & Kalisch, 2011). Strong anatomical connections between the amygdala and prefrontal cortical regions have been identified. Specifically, animal studies have found efferent and afferent connections between the amygdala and prefrontal regions including the mPFC and orbitofrontal cortex (OFC) (Aggleton, Burton, & Passingham, 1980; Carmichael & Price, 1995; Ghashghaei & Barbas, 2002; Ghashghaei, Hilgetag, & Barbas, 2007; Stefanacci & Amaral, 2002). More recently, diffusion tensor imaging studies have confirmed amygdala-mPFC anatomical connectivity in humans (Bracht et al., 2009; Croxson et al., 2005; Kim & Whalen, 2009).

In addition to anatomical connectivity, functional connections between the amygdala and mPFC have been identified in healthy individuals. Functional magnetic resonance imaging (fMRI) studies have revealed significant co-activation between the amygdala and prefrontal regions during emotion processing, with the direction of coupling depending on task characteristics and location within the frontal lobes. Studies utilizing passive emotional picture viewing paradigms with minimal cognitive load largely indicate positive coupling between the amygdala and prefrontal regions including ventromedial PFC (Heinz et al., 2005), dorsomedial

PFC (Kim et al., 2004) and dorsal ACC (Williams et al., 2006). Positive coupling between the amygdala and PFC has also been noted in emotion labeling (Satterthwaite et al., 2011) and matching (Pezawas et al., 2005) tasks. Findings of positive coupling suggest the amygdala and mPFC co-activate during the detection of emotionally salient stimuli that may have survival significance, for example during the perception of threat. In addition, they may co-activate to generate an emotional response, for example during emotion appraisal or expression. On the other hand, emotion studies requiring higher cognitive load such as emotion regulation typically find negative amygdala-mPFC coupling (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Drabant, McRae, Manuck, Hariri, & Gross, 2009; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner & Gross, 2005). Negative coupling results suggest top-down inhibition of amygdala activity by the PFC in order to regulate emotional experience, for example dampening negative affect. Affective labeling (Hariri, Bookheimer, & Mazziotta, 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003) and viewing (Das et al., 2005) tasks have also found negative amygdala-mPFC coupling during negative emotion conditions, which are also interpreted as top-down emotion regulation. Cognitive load is of particular relevance, as even a simple cognitive load can alter emotional experience. For example, one fMRI study found increased amygdala and decreased PFC response during passive viewing compared to labeling of emotional images (Taylor, Phan, Decker, & Liberzon, 2003). Location within the PFC is also important to note, as some studies have found instances of both negative and positive coupling between the amygdala and various PFC regions during emotion tasks (e.g., Pezawas et al., 2005; Satterthwaite et al., 2011; Williams et al., 2006) and at rest (Kim et al., 2011a). Ektin and colleagues (2011) suggested that dorsal PFC regions associated with emotion generation (e.g., appraisal, expression) hold positive connections with the amygdala while ventral PFC regions associated

with emotion inhibition (e.g., emotion regulation) hold negative functional connections with the amygdala. In sum, there is ample evidence of functional connections between the amygdala and mPFC during emotion processing, with data generally indicating positive coupling during low cognitive load tasks and negative coupling during high cognitive load tasks.

2. Amygdala-mPFC connectivity in schizophrenia

Functional connectivity studies indicate altered connectivity between the amygdala and mPFC in schizophrenia that may underlie emotional dysfunction in this population. Resting-state studies largely indicate decreased functional coupling between the amygdala and mPFC in individuals with schizophrenia (Fan et al., 2013; Hoptman et al., 2010; Liu et al., 2014), though one study found hyperconnectivity amongst these regions during rest (Salvador et al., 2010). Amygdala-mPFC hypoconnectivity may hold functional relevance, as lower connectivity strength has been correlated with higher aggression (Hoptman et al., 2010) and poorer emotion regulation task performance (Fan et al., 2013). In addition to altered resting state connectivity, there is burgeoning evidence of aberrant amygdala-mPFC connectivity during emotion processing tasks in people with schizophrenia. The exact nature of functional disconnection is unclear. Some studies have found decreased connectivity strength in people with schizophrenia (Anticevic et al., 2012a; Williams et al., 2004) and those with high proneness to psychosis (Modinos, Ormel, & Aleman, 2010). Similarly, others have found significant task-dependent amygdala-mPFC connectivity in controls but not those with schizophrenia (Das et al., 2007; Fakra, Salgado-Pineda, Delaveau, Hariri, & Blin, 2008; Leitman et al., 2008). Yet another study revealed opposite directions of functional coupling such that healthy controls showed negative coupling and schizophrenia participants showed positive coupling between the amygdala and ventral ACC (Das et al., 2007). Differences in connectivity findings is likely due to task

differences in cognitive engagement (e.g., passive viewing versus emotion labeling) and level of emotion processing (e.g., emotional appraisal versus regulation), variables that are known to influence amygdala-PFC connectivity (Etkin et al., 2011). Though findings differ, they provide convergent evidence of disjunction in amygdala-mPFC circuitry during emotion processing in schizophrenia.

The available literature on amygdala-mPFC functional connectivity in schizophrenia is not without limitations. First, the aforementioned studies largely assess amygdala-mPFC coupling during tasks in which subjects view emotional faces; however, results from these types of tasks are difficult to interpret given that people with schizophrenia often show general deficits in face processing (Marwick & Hall, 2008). Face processing studies also carry limited ecological validity, as emotional stimuli experienced in daily life contain environmental and human body information beyond that conveyed by faces. Only one functional connectivity study to date has used emotion stimuli that included emotional scenes (Anticevic et al., 2012a). Second, the published functional connectivity studies on emotion in schizophrenia have all used negatively-valenced images, with neutral images as a comparison condition. It is unclear whether the results reflect the impact of arousal versus valence, as these two aspects of emotion are confounded in these studies. It is also unclear whether functional disconnection extends to positively valenced stimuli, as the amygdala may also respond to positive affect (Hamann, Ely, Hoffman, & Kilts, 2002; Murray, 2007). Third, the previously mentioned tasks vary significantly in design, including passive viewing, emotional face matching, emotion labeling, gender identification, and cognitive reappraisal. These studies either employed passive viewing or tasks with high cognitive load, with none requiring participants to focus on or rate the intensity of emotional

response they felt while viewing emotionally-valenced images. Such a study could shed light on functional connectivity during self-evaluative emotional perception.

C. The Current Study

We previously examined effects of valence and diagnosis on brain activity in regions associated with emotion processing with this dataset (Nelson, Bjorkquist, Olsen, & Herbener, under review). Results indicated minimal differences between schizophrenia and control participants in brain activation to emotional stimuli, consistent with previous studies (Dowd & Barch, 2010; Harvey, Armony, Malla, & Lepage, 2010). Of note, no group differences were observed in the amygdala, ACC, or medial frontal gyrus. Schizophrenia participants also showed similar ratings of emotional response to images, and both groups reported stronger emotional response to negative than positive images (Nelson et al., under review). While isolated amygdala activity and behavioral response may be intact, it is possible that functional disconnection between the amygdala and other regions may underlie emotional dysfunction in this population. Consistent with this notion, Anticevic et al. (2012a) found no group differences in behavioral or amygdala response, but decreased amygdala-mPFC negative coupling in people with schizophrenia in an emotional perceptual decision making task.

The present study builds upon prior findings in our fMRI emotion perception task (Nelson et al., under review), investigating whether functional connectivity between the amygdala and mPFC differs by valence (positive, negative, neutral) and group (schizophrenia (SZ), healthy controls (HC)). Specifically, context-dependent changes in functional connectivity were examined using psychophysiological interaction analyses, which allow for comparison of task-related connectivity between individuals with schizophrenia and healthy controls. The current study aims to expand our knowledge of amygdala-mPFC functional connectivity during

emotion processing in schizophrenia and address the previously mentioned limitations. First, the present study uses ecologically-valid task stimuli, specifically, IAPS images that include emotional people and scenes in addition to faces. Second, it is the first study to examine functional connectivity during processing of positive in addition to negative stimuli. Finally, the current study is the first to investigate connectivity during self-evaluative emotion perception, requiring participants to rate the intensity of emotional response they felt while viewing emotionally-valenced images.

D. The Hypotheses of the Current Study

We predicted that individuals with schizophrenia would show decreased strength of amygdala-mPFC connectivity during negative compared to neutral image perception, based on the majority of past studies indicating decreased connectivity strength in schizophrenia participants during negative emotion processing (Anticevic et al., 2012a; Fakra et al., 2008; Leitman et al., 2008; Modinos et al., 2010; Williams et al., 2004). Based on the literature suggesting positive coupling between the amygdala and mPFC during negative emotion appraisal/expression (Etkin et al., 2011) and other emotion processing tasks low in cognitive load (Heinz et al., 2005; Kim et al., 2004; Williams et al., 2006) we expected to find positive coupling in mPFC regions across study participants during perception of negative compared to neutral stimuli. However, negative coupling was also possible, as affective labeling (Hariri et al., 2000; Hariri et al., 2003) and viewing (Das et al., 2005) tasks (in addition to those high in cognitive load) have also found negative coupling between the amygdala and mPFC. Finally, exploratory analyses examined connectivity during positive compared to neutral image perception, as well as the relationship between connectivity strength and current functioning in schizophrenia participants.

II. METHOD

Parts of this chapter were previously published in Psychiatry Research: Neuroimaging (Bjorkquist & Herbener, 2013)

A. Participants

Study participants included individuals meeting *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994) criteria for schizophrenia or schizoaffective disorders (n = 14) and healthy controls (n = 14). Schizophrenia participants were recruited from ads in the community, special interest web sites (National Alliance on Mental Illness, National Institutes of Health), and physician referral. Healthy controls were recruited via ads in the community and were matched to schizophrenia participants on the basis of age, sex, ethnicity, years of education, parental education, and intelligence. Exclusionary criteria included history of head trauma with loss of consciousness longer than 15 minutes, substance abuse or dependence within the past 6 months, contraindication for MRI scanning (e.g., metallic implants in the body), or serious medical conditions that could influence brain activity or blood flow (e.g., epilepsy).

Diagnoses for all participants were established with the Structured Clinical Interview for *DSM-IV* diagnosis (First, Spitzer, Gibbon, & Williams, 1997). Schizophrenia participants were clinically stable outpatients and had been on a stable medication regimen for a minimum of 4 weeks prior to testing. Healthy control participants were not taking any psychotropic medications and had no family history of schizophrenia spectrum disorders. Participants were asked to avoid caffeine and tobacco for at least two hours prior to the neuroimaging session. All participants completed the reading subtest of the Wide Range Achievement Test, 3rd ed. (WRAT; Wilkinson, 1993), a measure of premorbid intelligence, and the Wechsler Abbreviated Scale of Intelligence

(WASI; Wechsler, 1999), a measure of current intelligence. See Table I for participant demographics. Written informed consent was obtained from all participants, and the study was approved by the Institutional Review Board at the University of Illinois at Chicago (UIC). Participants received financial compensation for their time participating in the research.

Participants with schizophrenia were additionally administered two clinical rating measures by a Ph.D. level clinician. Severity of symptoms was rated with the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987). Participants with schizophrenia (n = 9) did not differ from those with schizoaffective disorder (n = 5) on PANSS Positive, Negative, or General ratings (p's > 0.05). Schizophrenia participants were also interviewed using the Heinrichs-Carpenter Quality of Life Scale (QLS; Heinrichs, Hanlon, & Carpenter, 1984). The QLS is a semi-structured clinical interview yielding composite scores in several domains, with higher scores reflecting a greater quality of life. The present study assessed social functioning (QLS-SF), a composite score reflecting frequency of social contact as well as capacity for intimacy, active versus passive participation, avoidance or withdrawal tendencies, empathy, and emotional interaction; work functioning (QLS-WF), a composite score measuring the extent of functioning as a worker, student, or housekeeper/parent as well as level of accomplishment, degree of underemployment, and satisfaction with the current role; and motivation (QLS-M), a composite score reflecting sense of purpose, motivation, curiosity, and ability to experience pleasure. One schizophrenia participant did not complete the QLS.

TABLE I

SAMPLE DEMOGRAPHICS FOR SCHIZOPHRENIA (SZ) AND HEALTHY CONTROL (HC) PARTICIPANTS

	SZ [M±SD]	**HC [M±SD]**	***p***
N	14	14	
Age (years)	31.57±7.52	31.36±11.38	n.s.*
Sex (% female)	29	36	n.s.
Race (% African American)	50	71	n.s.
Years of Education	13.89±3.00	13.64±1.65	n.s.
Parental Education Level	< 12th grade 0% High school graduate 36% Some college 18% College graduate 45%	< 12th grade 14% High school graduate 57% Some college 21% College graduate 7%	n.s.
Handedness (% right)	100	100	n.s.
WRAT	102.57±12.47	94.57±10.79	n.s.
WASI	104.43±12.74	99.50±14.87	n.s.
PANSS Positive	17.93±4.75		
PANSS Negative	17.36±5.81		
PANSS General	38.79±9.24		
	Medication		
Number on Medication	11	0	
Typical Antipsychotics (*n*)	3	0	
Atypical Antipsychotics (*n*)	9	0	
Antidepressants (*n*)	4	0	
Stimulants (*n*)	0	0	
Mood Stabilizers (*n*)	1	0	
Sedative/Hypnotics (*n*)	2	0	

*n.s.: nonsignificant, $p > 0.05$

Note. Parental Education Level reflects mean level obtained by both parents. Parental education level was unavailable for 3 SZ participants. WRAT: Wide Range of Achievement Test; WASI: Wechsler Abbreviated Scale of Intelligence; PANSS: Positive and Negative Syndrome Scale.

B. Experimental Design

Positive, negative, and neutral images were selected from the IAPS library (Lang, Bradley, & Cuthbert, 2005). Positive and negative images were equated on arousal and were rated as more arousing than neutral images based on the IAPS normative data. Positive and negative images exceeded a cut-off value of four on a 1-9 scale for arousal ratings. Positive, negative, and neutral images also differed by normative valence ratings (Lang et al., 2005). Positive images exceeded a rating of seven and negative images were below a rating of four for valence ratings on a 1-9 scale. See Figure 1 for example images. Emotionally extreme images from the IAPS library were excluded so the task would be less disturbing to participants and to ensure the ecological validity of the study.

Figure 1. Example task stimuli. Negative (left), positive (middle), and neutral (right) images.

To acclimate participants to the MRI scanner and prevent gross movement, participants completed a mock scan session approximately one week prior to the neuroimaging session. They were also given a computerized practice version of the task outside of the scanner to ensure task comprehension. During the task, participants viewed 108 IAPS images. Images were presented in blocks of 12 in each condition and included 36 positive, 36 negative, and 36 neutral images. Images were ordered using a Latin square design. Each image was presented for four seconds, and 10 second rest periods were included between blocks to allow brain activation to return to baseline before the subsequent block. During the presentation of each image, participants rated the intensity of their emotional response to the image, either low (none to low) or high (moderate to strong), via button press. Following the scan, participants were asked to explain task instructions in order to confirm task comprehension.

C. Data Acquisition and Analysis

Analysis of behavioral ratings of emotional response to images is reported elsewhere (Nelson et al., under review). Structural and functional scans were acquired on a 3 Tesla scanner (Signa, General Electric Medical System, Milwaukee, Wisconsin) at the UIC Medical Center. The acquisition protocol for the functional scan was a gradient-echo echo-planar imaging (EPI) series with TR = 2000ms, TE = 25ms, flip angle = 90 degrees, field of view = 200mm, matrix = 64mm x 64mm, slice thickness = 3mm, gap = 1mm, for 33 axial slices. The structural scan immediately followed the functional scan.

fMRI data were processed using public domain Analysis of Functional Neuroimages (AFNI) software (http://afni.nimh.nih.gov/afni/). Context-dependent changes in functional connectivity were examined using psychophysiological interaction analyses (http://afni.nimh.nih.gov/sscc/gangc/CD-CorrAna.html). We examined whether the amygdala

showed significantly altered correlated activity with the mPFC while participants viewed 1) negative compared to neutral, and 2) positive compared to neutral images. Analyses were run separately for the left and right hemispheres (e.g., right amygdala with right mPFC). Between-group t-tests were then run to determine whether these context-dependent changes in functional connectivity differed between HC and SZ participants.

1. Regions-of-interest

Anatomically-defined masks were created in Talairach space separately for the left and right hemispheres of the amygdala. The amygdala region-of-interest (ROI) mask was obtained from researchers with extensive experience creating masks using AFNI software (e.g., Pavuluri, Passarotti, Harral, & Sweeney, 2009; Pavuluri, Passarotti, Fitzgerald, Wegbreit, & Sweeney, 2012). Specifically, the amygdala ROI was drawn on a merged structural image of 15 healthy subjects and traced in coronal view, a method shown to be reliable and valid (Kates, Abrams, Kaufmann, Breiter, & Reiss, 1997). The anterior boundary was defined at the presence of the anterior commissure and posterior boundary at the presence of hippocampus. The superior boundary was defined by the entorhinal sulcus and inferior boundary by the white matter of the uncus. The medial boundary was defined by the presence of cerebrospinal fluid and lateral boundary by the white matter of the insular cortex. ROIs for the medial frontal gyrus and ACC are available on AFNI, and were merged into a single mPFC ROI for each hemisphere. These ROIs were created by tracing Talairach and Tournoux brain illustrations (Lancaster et al., 1997; Lancaster et al., 2000).

2. Individual subject preprocessing

AFNI's 3D volume registration program was used to detect and correct for motion across individual participants' time series. Individual data were spatially smoothed using a 5mm full-

width half-maximum Gaussian filter, and then normalized by dividing signal intensity of a voxel at each time point by the mean signal intensity of that voxel across the time series and multiplying by 100. The amygdala was selected as the seed region and first transformed into original space using parameters from each individual's structural scan. The average time series in the amygdala was extracted and the trend removed from the time series. The psychophysiological variable was created as the deconvolved seed time series multiplied by a vector coded by condition (1 for negative, -1 for neutral, 0 for all other conditions; 1 for positive, -1 for neutral, 0 for all other conditions). Next, AFNI's 3dDeconvolve program was used to obtain per-voxel fit coefficients corresponding to BOLD response associated with the psychophysiological interaction term. This regression controlled for the seed time series, valence conditions, and individual subject motion. Next, images were normalized to stereotaxic space (Talairach & Tournoux, 1988) and resampled to the original acquisition size of $3x3x3mm^3$ voxels. The mPFC mask was then applied to the data to isolate this region-of-interest.

3. Group analyses

Based on Monte Carlo simulations calculated by AFNI's AlphaSim program, we required 486 mm^3 contiguous voxels significant at the $p < 0.01$ level for mPFC significance of $p < 0.05$. To examine whether the psychophysiological interaction differed between SZ and HC participants, t-tests were used to assess between-group differences in the interaction between valence (negative versus neutral; positive versus neutral) and amygdala—mPFC connectivity. Significant group differences were followed up within each group to determine effects underlying the interaction.

III. RESULTS

A. Amygdala-mPFC Connectivity

We hypothesized that HC participants would demonstrate greater connectivity strength between the amygdala and mPFC during negative compared to neutral image perception than would SZ participants**.** Results revealed one area within the right mPFC that showed altered connectivity strength with the right amygdala in SZ compared to HC participants. This 648mm^3 cluster was anterior to the genu of the corpus callosum and included portions of the medial frontal gyrus and rostral ACC. In order to determine the nature of the observed between-group effect, within-group t-tests compared right amygdala-mPFC functional connectivity during negative compared to neutral image perception. Results indicated opposite patterns of functional coupling between groups, though results failed to reach statistical significance in either group. Specifically, healthy controls demonstrated *positive* functional coupling, while schizophrenia participants demonstrated *negative* functional coupling between the right amygdala and mPFC. See Figure 2.

In contrast to the significant group difference observed in right amygdala-mPFC connectivity, we did not find any significant differences in left amygdala-mPFC connectivity between HC and SZ participants. We also failed to find group differences in amygdala-mPFC connectivity in positive compared to neutral image perception, for either the right or left hemispheres.

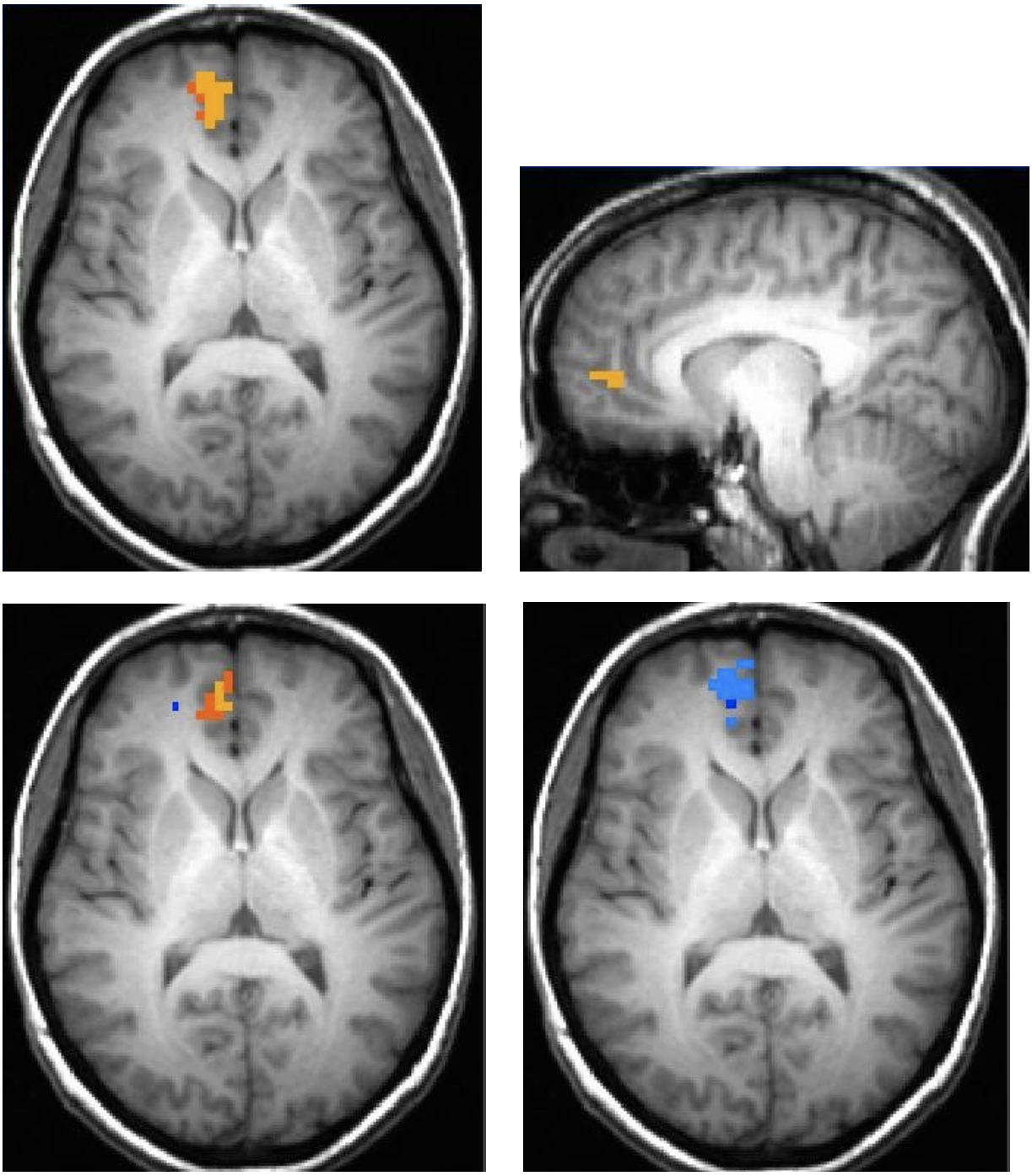

Figure 2. *Top panel:* mPFC region of task- and group-related functional connectivity with right amygdala, in negative versus neutral perception (HC > SZ). Peak Talairach coordinates: x=-7.5, y=-49.5, z=8.5. *Bottom panel:* Within-group negative versus neutral connectivity with right amygdala; left panel, HC; right panel, SZ. Note: In bottom panel, voxelwise $p < 0.05$, uncorrected, for visualization.

B. Quality Assurance

To ensure that results were not due to individual differences in behavioral response, functional connectivity analyses were repeated with the addition of emotion intensity rating as a covariate. Mean intensity ratings were calculated per block for each subject, and then transformed into z-scores and combined into a single covariate file. Individual and group analyses were repeated with the addition of the covariate. Results of initial analyses were replicated in covariate analyses, ruling out the possibility that results were driven by individual differences in emotion intensity ratings.

To determine specificity of results between the right amygdala and mPFC, functional connectivity analyses were conducted between the right amygdala and a region not expected to differ by group: the precuneus. Results revealed no group differences in right amygdala-precuneus connectivity in negative compared to neutral image perception.

C. Functional Significance

We next aimed to determine the functional significance of connectivity results. Specifically, we examined whether right amygdala-mPFC connectivity strength during negative versus neutral image perception was related to symptoms and quality of life in SZ participants. First, the resultant right mPFC cluster demonstrating significant connectivity with the amygdala was extracted and applied to individual subject preprocessed data. Beta weights representing connectivity strength between the mean amygdala and maximum mPFC BOLD response were then obtained per subject for the amygdala x condition interaction term using AFNI's 3dROIstats program. Pearson's correlation analyses were used to determine the relationship between maximum connectivity strength and the following variables: PANSS Positive, PANSS Negative, PANSS General, HQL-SF, HQL-WF, and HQL-M. Results revealed a significant positive

correlation between maximum connectivity strength and HQL-SF in SZ participants, $r(14) = 0.63$, $p = 0.021$, such that greater right amygdala-mPFC connectivity strength during negative compared to neutral image perception was associated with increased social functioning. See Figure 3. No other significant correlations between maximum connectivity strength and functional outcome were observed.

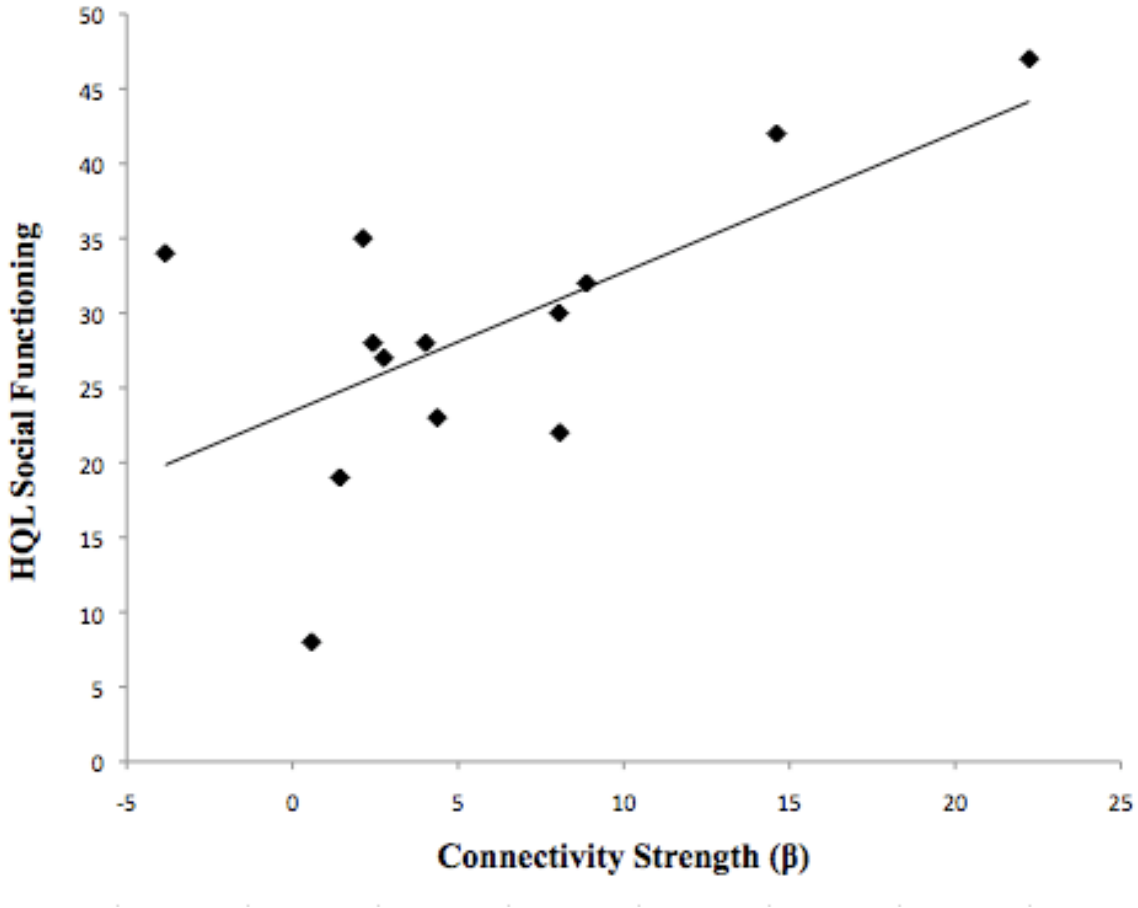

Figure 3. Correlation between clinician-rated social functioning (HQL social functioning) and maximum amygdala-mPFC connectivity strength during negative versus neutral perception.

IV. DISCUSSION

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The current study investigated functional connectivity between the amygdala and mPFC during an emotion perception task, and whether connectivity differed between participants with schizophrenia and healthy controls. Results of psychophysiological interaction analyses indicate between-group differences in connectivity strength in negative compared to neutral image perception, consistent with our predictions. Specifically, we found significant group differences in functional connectivity between the right amygdala and a region within the right mPFC that included the medial frontal gyrus and rostral ACC. Follow-up analyses indicated opposite patterns of functional coupling between groups, such that healthy controls showed positive amygdala-mPFC coupling, while participants with schizophrenia showed negative amygdala-mPFC coupling. Furthermore, groups did not differ in behavioral or amygdala response (Nelson et al., under review), ruling out the possibility that connectivity results reflected group differences in task performance or amygdala responsivity.

Past research on healthy populations indicates both negative and positive functional connections between the amygdala and mPFC during emotion processing. The amygdala and mPFC typically hold positive connections during emotion generation (e.g., expression, appraisal) and in emotion tasks lower in cognitive load (e.g., passive viewing; e.g., Heinz et al., 2005; Kim et al., 2004; Williams et al., 2006). In contrast, the two regions are inversely related during emotion inhibition and in emotion tasks higher in cognitive load (e.g., emotion regulation; e.g., Banks et al., 2007; Drabant et al., 2009; Ochsner et al., 2002; Ochsner & Gross, 2005). In our emotion perception task requiring affective response ratings, healthy controls utilized co-active

processing between the amygdala and mPFC, consistent with salience detection and emotion generation. On the other hand, participants with schizophrenia demonstrated an inverse relationship between the amygdala and mPFC, consistent with top-down inhibition. These findings suggest that individuals with schizophrenia may utilize emotion regulation strategies when perceiving and judging their emotional response to negative stimuli, perhaps to dampen negative affect. Such an interpretation may help explain affective flattening in schizophrenia, at least for negatively valenced information. Alternatively, our findings of positive coupling in controls and negative coupling in schizophrenia participants may relate to cognitive load. While our task was relatively low in cognitive load, it is possible that the participants with schizophrenia required additional cognitive effort to generate emotion intensity ratings, eliciting a high cognitive load negative coupling response.

Our results of altered amygdala-mPFC connectivity during emotion processing in schizophrenia adds to the growing literature on this topic. To date, studies have reported decreased connectivity strength (Anticevic et al., 2012a; Modinos, et al., 2010; Williams et al., 2004) and task-dependent connectivity in controls but not those with schizophrenia (Das et al., 2007; Fakra et al., 2008; Leitman et al., 2008) in tasks eliciting negative amygdala-mPFC coupling in healthy individuals. The current study is the first to examine connectivity in schizophrenia using a task that elicits positive coupling in healthy controls. Our findings of opposite direction of functional coupling complement the findings of Das and colleagues (2007), who reported negative amygdala-mPFC coupling in controls and positive coupling in schizophrenia participants. Our findings also support those of Anticevic and colleagues (2012a), who reported altered functional connectivity in participants with schizophrenia despite intact behavioral and amygdala responses. In sum, our results, along with the extant literature, suggest

that abnormal interactions between the amygdala and mPFC may underlie emotion deficits in individuals with schizophrenia. Disturbances in functional connectivity during early stages of emotion processing could lead to impairment in higher order aspects of emotion processing by influencing the neural resources available for further processing. For example, aberrant perception of emotional stimuli could reasonably lead to abnormal interpretation of such information, guiding decisions and behavior.

A. Symptom Correlations

Symptom correlation analyses indicated that connectivity between mean amygdala and maximum mPFC response during negative image perception predicted clinician-rated social functioning in individuals with schizophrenia. Of note, this composite variable (QLS Social Functioning) reflects an interplay of social and emotional factors, including social contact and participation, capacity for intimacy, avoidance or withdrawal tendencies, empathy, and emotional interaction. Our results indicate that small regions of significant positive coupling between the amygdala and mPFC predicted improved social and emotional functioning in participants with schizophrenia. This suggests that some, albeit limited, capacity to co-activate the amygdala and mPFC relates to a more positive functional outcome in patients. These results demonstrate the functional significance of our task and build upon previous literature demonstrating relationships between brain response and functional outcome in individuals with schizophrenia. For example, one study found a correlation between amygdala-PFC connectivity strength during neutral image perception and flat affect, such that weaker connectivity predicted greater affective flattening (Anticevic et al., 2012a). In addition, studies examining BOLD response have demonstrated positive correlations between social functioning and activity in the ACC (Nelson et al., under review) and amygdala (Pinkham, Hopfinger, Pelphrey, Piven, & Penn,

2008) during social and emotional processing in people with schizophrenia. These findings are consistent with the role of the amygdala in salience detection (e.g., Sander, Grafman, & Zalla, 2003) and ACC in evaluation of affective social information (e.g., Harris, McClure, van den Bos, Cohen, & Fiske, 2007). Taken together, these results suggest that the amygdala and mPFC function in concert during the processing of social and emotional information, and stronger connectivity between these two regions predicts better functional outcome in people with schizophrenia.

B. Valence

The present study was the first to examine amygdala-mPFC coupling during positive emotion processing in schizophrenia. Although we found effects in right amygdala-mPFC connectivity during negative emotion perception, we failed to find effects during positive image perception. Examination of data during positive versus neutral processing revealed similar patterns of functional coupling (positive coupling for healthy controls, negative coupling for schizophrenia participants), but within and between group analyses failed to reach statistical significance. Of note, a previous study with these data indicated that all participants rated negative images as evoking a stronger emotional response than positive images (Nelson et al., under review). This difference in behavioral response to images may have contributed to differences in findings with regard to valence.

The amygdala's role in processing negative emotion, particularly fear, is well-established (for review, see Phan et al., 2002; Phelps & LeDoux, 2005; Zald, 2003). More recently, research has revealed amygdala response to positive stimuli. The amygdala is implicated in reward and positive reinforcement (Murray, 2007), and has been proposed as a salience detector given its role in processing positive and biological stimuli (Sander et al., 2003). However, studies on

positive emotion processing are inconsistent, with many studies failing to detect amygdala response to positive stimuli (Zald, 2003). Several possibilities exist to explain amygdala response to negative but not positive stimuli, including arousal and perceptual intensity. By nature, positive stimuli are less strongly pleasant than negative stimuli are unpleasant, and are typically less arousing and intense than negative stimuli. For example, a picture of an ice cream cone is likely less arousing and less intensely perceived than a picture of a gun pointed at the viewer. In addition, while greater amygdala activity is associated with greater arousal for negative images, highly pleasant stimuli can be either arousing (e.g., beautiful woman) or calming (e.g., beautiful landscape), and there are instances of decreased amygdala response to highly positive emotional stimuli in the literature (e.g., Blood & Zatorre, 2001). It is thus difficult for studies to truly match positive and negative images in terms of valence intensity and arousal. The current study used IAPS normative data to match negative and positive stimuli on arousal, but participants rated their emotional response to negative images as significantly more intense than their response to positive images (Nelson et al., under review). Thus, while our findings of altered connectivity appear to be specific to negative images, it is possible that images in our positive condition were simply not arousing or intense enough to elicit significant group differences in amygdala-mPFC coupling.

C. Laterality

The current study observed effects in right, but not left, amygdala-mPFC connectivity during negative emotion processing. A majority of emotion studies in the literature report lateralized amygdala findings, though laterality results are highly inconsistent across studies. Indeed, in a meta-analysis of emotion studies in healthy individuals, 41 studies found left amygdala activity, 30 found right amygdala activity, and 17 found bilateral amygdala activity,

with no consistent pattern within or between tasks (Baas, Aleman, & Kahn, 2004). Several theories have been proposed regarding amygdala laterality. First, the left amygdala processes verbal information and detailed features, while the right amygdala processes visual information and engages in quick and shallow processing (Markowitsch, 1998). Second, and similarly, laterality may depend on elaboration and interpretation of emotional information, such that the right amygdala is active when the stimulus is visual and emotional valence is obvious, while the left amygdala is active when the emotional property of the stimulus requires verbal elaboration (Phelps, O'Connor, Gatenby, Core, Grillon, & Davis, 2001). Third, laterality may depend on task instructions, i.e., whether participants are asked to explicitly judge the emotionality of a stimulus versus passively view (and implicitly process) the stimulus (Lange et al., 2003). Fourth, the right amygdala may be involved in rapid stimulus detection and habituate quickly, while the left amygdala is involved in sustained response (Wright et al., 2001). The meta-analysis failed to support any of these theories, as it did not find significant laterality effects of stimulus type, elaboration, task instructions, or habituation. Instead, it was proposed that the right amygdala may process global, holistic aspects of a stimulus, while the left amygdala may process local details of the stimulus (Baas et at., 2004). Such a theory is consistent with our findings of right hemisphere activity, as our images largely consisted of complex scenes including multiple stimuli that would require more global processing. Our findings are also consistent with Markowitsch (1998), who proposed that the right amygdala processes visual information.

D. Strengths

The current study addresses several limitations in the literature, adding to our growing understanding of functional connectivity in schizophrenia. Prior studies on emotion processing in schizophrenia have typically utilized emotional faces as task stimuli, which hold minimal

ecological validity and may be confounded by general face processing impairment. The current study addressed this limitation by using ecologically valid stimuli, such as emotional people and scenes. Our results of altered amygdala-mPFC connectivity are consistent with the literature, suggesting that extant findings extend beyond face processing to general emotion processing. Second, to our knowledge, studies of amygdala-mPFC connectivity in schizophrenia have only examined connectivity in processing of negative stimuli, so it is unclear whether altered connectivity extends to positively valenced information. Based on our findings, it appears that altered connectivity in individuals with schizophrenia is specific to negative stimuli; however, this may due to differences in arousal, as our negative images were rated as more emotionally intense than positive. Examination of the data indicated that people with schizophrenia and healthy controls demonstrated similar patterns of connectivity in negative and positive conditions, but with lower connectivity strength in the less arousing positive condition. Finally, emotion processing connectivity studies in the literature vary largely in task design and most require participants to rate some aspect of a stimulus, such as matching emotional faces, labeling valence of the stimulus, identifying gender, or passively viewing. Our study is one of few that are low in cognitive load and require participants to rate their internal emotional response to task stimuli. Our findings thus add to the literature by demonstrating altered functional connectivity during self-evaluative emotional perception.

E. Limitations

This study contributes to the emotion processing literature in schizophrenia by demonstrating abnormal functional connectivity between the right amygdala and right mPFC during negative emotion perception. At the same time, we note that we used an *a priori*, theoretically-driven ROI approach, so analyses were focused on predetermined brain regions that

have previously been associated with emotion processing. It is possible that functional disconnections between other regions outside the scope of this study may also underlie emotion processing deficits in schizophrenia. Second, in contrast to many studies using IAPS stimuli, our affective conditions excluded extreme images such as mutilated bodies or pornographic images in order to not upset our participants or decrease ecological validity; thus, our range of affective valence was restricted. Inclusion of extreme images would likely elicit increased limbic brain response and perhaps lead to different results, such as differences in functional connectivity during positive emotion perception. Third, we did not examine potential effects of neuropsychological, diagnostic, or demographic factors that could have influenced results. However, we attempted to control for these effects by matching the SZ and HC groups on demographics and by comparing conditions that required the same judgment. Finally, a majority of participants in the SZ group were taking antipsychotic medication. Further, the group was heterogeneous in medication type, dosage, and medication history. Our results may have been influenced by effects of psychotropic medication on brain functioning, both between groups and within the SZ group. However, most individuals with schizophrenia who are in psychiatric care are treated with antipsychotic medication. Thus, our results provide important information about brain circuitry in people with schizophrenia in their typical life circumstances.

F. Future Directions

The current study focused on connectivity between the amygdala and mPFC, but we recognize a network of regions work in concert to process emotional information. Future studies should examine functional connectivity between other regions known to be involved in emotion processing, such as the orbitofrontal cortex, dorsolateral prefrontal cortex, insula, hippocampus, and ventral striatum. Future studies should also use data-driven approaches, such as independent

component analysis, to investigate emotion processing networks in schizophrenia. Second, we note that we excluded extreme emotional images from our study, and positive images were rated as less emotionally intense than negative images. Thus, we were unable to determine whether null results in the positive condition were due to differences in valence or arousal. Future studies should match positive and negative stimuli on participant-rated emotional response, perhaps by utilizing more emotionally evocative positive stimuli. Third, we controlled for potential effects of demographic factors by matching groups on demographics but did not investigate contributions of these variables to results of the study. Future studies can examine effects of demographic factors that are known to influence brain response to emotional stimuli, such as sex and race (Brekke et al., 2005; Kring and Moran, 2008). Future studies can also more thoroughly investigate potential differences in connectivity between individuals with schizophrenia and schizoaffective disorder, as our study was unable to do so with our relatively small sample size. Fourth, the current study found that functional connectivity strength in our emotion perception paradigm predicted clinician-rated social/emotional functioning. Future studies can use social cognition tasks to further investigate connectivity during social processing, particularly for affective information. Finally, future studies with larger sample sizes can conduct meaningful analyses on the effects of medication on functional connectivity between the amygdala and mPFC.

G. Conclusions

In sum, our findings provide evidence of altered functional connectivity between the amygdala and mPFC, including the medial frontal gyrus and rostral ACC, in people with schizophrenia during perception of negative stimuli. Aberrant functional connectivity in schizophrenia was observed in light of normal BOLD response in these regions and behavioral

response to images, suggesting altered connectivity as a key etiological factor underlying abnormal perception of emotional stimuli in schizophrenia. Furthermore, connectivity strength predicted clinician-rated social functioning, suggesting a relationship between altered connectivity and functional outcome. This study adds to the growing literature on abnormal functional connections in schizophrenia and supports the functional disconnection hypothesis of schizophrenia. Future studies should continue to take a network approach to investigate the neural correlates of emotion processing in schizophrenia.

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Nelson, B. D., Bjorkquist, O. A., Olsen, E. K., & Herbener, E. S. (under review). Functional correlates of anhedonia and neural responses to emotional stimuli in schizophrenia: An fMRI investigation. *Manuscript submitted to Schizophrenia Research.*

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Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences*, *9*(5), 242-249.

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Pavuluri, M. N., Passarotti, A. M., Fitzgerald, J. M., Wegbreit, E., & Sweeney, J. A. (2012). Risperidone and divalproex differentially engage the fronto-striato-temporal circuitry in pediatric mania: A pharmacological functional magnetic resonance imaging study. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*(2), 157-170.

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Pinkham, A. E., Hopfinger, J. B., Pelphrey, K. A., Piven, J., & Penn, D. L. (2008). Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders. *Schizophrenia Research, 99*, 164-175.

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Russell, T. A., Reynaud, E., Kucharska-Pietura, K., Ecker, C., Benson, P. J., Zelaya, F., ... Phillips, M. L. (2007). Neural responses to dynamic expressions of fear in schizophrenia. *Neuropsychologia*, *45*(1), 107-123.

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Taylor, S. F., Phan, K. L., Decker, L. R., & Liberzon, I. (2003). Subjective rating of emotionally salient stimuli modulates neural activity. *NeuroImage*, *18*(3), 650-659.

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Williams, L. M., Das, P., Liddell, B. J., Kemp, A. H., Rennie, C. J., & Gordon, E. (2006). Mode of functional connectivity in amygdala pathways dissociates level of awareness for signals of fear. *The Journal of Neuroscience*, *26*(36), 9264-9271.

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Zald, D. H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Reviews, 41*, 88-123.

OLIVIA BJORKQUIST

EDUCATION

2011-present **Ph.D. Candidate, Clinical Psychology**, University of Illinois at Chicago
Dissertation**:** *Emotion Perception in Schizophrenia: A Functional Connectivity Study*
Committee: Ellen Herbener (Chair), Robin Mermelstein, Stewart Shankman, James Reilly, and Alessandra Passarotti. Defense passed May 2014.

2008-2011 **M.A., Clinical Psychology**, University of Illinois at Chicago
Master's Thesis: *Social Perception in Schizophrenia: Evidence of Occipital and Prefrontal Dysfunction*
Committee: Ellen Herbener (Chair), Robin Mermelstein, and Stewart Shankman.

2001-2005 **B.S., Psychobiology (Cum Laude)**, University of California, Los Angeles

July 2012 **Continuing Education**, Marquette University
Neuroanatomical Dissection: Human Brain and Spinal Cord

AREAS OF SPECIALIZATION AND INTEREST

Clinical

- Neuropsychological assessment of adults primarily with neurological diagnoses
- Application of empirically-supported treatments (e.g., cognitive behavioral therapy)
- Worked with numerous populations (adults, adolescents) in diverse settings (inpatient, outpatient)

Research

- Neural correlates of social and emotional processing in schizophrenia
- Neuroimaging techniques, including structural MRI, DTI, functional MRI, and functional connectivity
- Neuropsychological test administration in schizophrenia and bipolar disorder

AWARDS/DISTINCTIONS

2013 AACN Annual Meeting Edith Kaplan Award for Best Trainee Poster Presentation ($100)
2012 Letters of Arts and Sciences Ph.D. Student Travel Award (Total Award: $500/year)
2008-2012 UIC Graduate Student Council/Graduate College Travel Award (Total Award: $500/year)
2008-2012 UIC Department of Psychology Travel Award (Total Award: $200/year)
2001-2005 UCLA Alumni Scholar ($500/year)

NEUROPSYCHOLOGICAL CLINICAL EXPERIENCE

2013-present **Adult Neuropsychology Externship (NorthShore Dept. of Psychiatry & Behavioral Sciences)**
NorthShore University Health System, a University of Chicago Medical School affiliate, Evanston, IL

- Provided adult neuropsychological assessment services including test administration, interpretation, and report writing on individuals primarily with neurological diagnoses.
- Attended biweekly didactics, including neurology grand rounds and group supervision.
- Intensive case load with weekly assessments.

Supervisors: Jerry Sweet, Ph.D., ABPP-CN, Leslie Guidotti Breting, Ph.D., ABPP-CN, and Elizabeth Geary, Ph.D., ABPP-CN

2011-2013 **Neuropsychology Service Externship (UIC Dept. of Psychiatry)**
University of Illinois-Chicago Medical Center, Chicago, IL

- Provided adult neuropsychological assessment services including test administration, interpretation, and report writing on individuals primarily with neurological diagnoses.
- Attended weekly didactic training, including neuroanatomy course, behavioral neuroscience seminars, applied neuropsychology course, and group supervision.
- Presenting problems included dementia, traumatic brain injury, epilepsy, chronic pain, stroke, multiple sclerosis, severe mental illness, ADHD, learning disabilities, electrical shock injury, and serious medical conditions such as end-state renal disease.

Supervisor: Neil Pliskin, Ph.D., ABPP-CN

2009-2010 **Office of Applied Psychological Services (UIC Dept. of Psychology)**
University of Illinois at Chicago, Chicago, IL.
- Provided adult and child neuropsychological assessment services, including test administration, interpretation, and report writing on individuals primarily with LD and ADHD diagnoses.
- Provided feedback to clients and their families.

Supervisors: Audrey Ruderman, Ph.D. and Neil Pliskin, Ph.D., ABPP-CN

PSYCHOTHERAPY CLINICAL EXPERIENCE

2008-present **Office of Applied Psychological Services (UIC Dept. of Psychology)**
University of Illinois at Chicago, Chicago, IL.
- Provided individual treatment and conducted intake interviews with patients presenting with a variety of anxiety and mood disorders.
- Provided manualized group treatment for patients with social anxiety disorder.
- Provided manualized individual treatment for panic disorder.

Supervisors: Nancy Dassoff, Ph.D., Evelyn Behar, Ph.D., and Gloria Balague, Ph.D.

SPECIALIZED CLINICAL TRAINING

2013 **Diagnosis in the DSM-5 Workshop** (Presented by Dr. Christopher Hopwood)
2013 **Interviewing Survivors of Torture** (Presented by Marjorie Kovler Center)
2010 **Group Therapy for Social Anxiety Disorder** (Protocol therapy training)
2010 **Performing Clinical Services with Hearing Impaired Clients** (Presented by UIC Disability Center)
2009 **Wechsler Memory Scale - IV (WMS-IV) Workshop** (Presented by Pearson)
2008 **Wechsler Adult Intelligence Scale - IV (WAIS-IV) Workshop** (Presented by Pearson)

PUBLICATIONS

1. Hill, S.K., **Bjorkquist, O.,** Carrathers, T., Roseberry, J.A., Hochberger, W., & Bishop, J.R. (2013). Sequential processing deficits in schizophrenia: Relationship to neuropsychology and genetics. *Schizophrenia Research, 151*(1-3), 91-96.
2. **Bjorkquist, O.A.,** & Herbener, E.S. (2013). Social perception in schizophrenia: Evidence of temporo-occipital and prefrontal dysfunction. *Psychiatry Research: Neuroimaging, 212*(3), 175-182.
3. Behar, E., McGowan, S.K., McLaughlin, K.A., Borkovec, T.D., Goldwin, M., & **Bjorkquist, O.** (2011). Concreteness of positive, negative, and neutral repetitive thinking about the future. *Behavior Therapy, 43*(2), 300-312.
4. **Bjorkquist, O.A.,** Fryer, S.L., Reiss, A.L., Mattson, S.N., & Riley, E.P. (2010). Cingulate gyrus morphology in children and adolescents with prenatal alcohol exposure. *Psychiatry Research: Neuroimaging, 181*(2), 101-107.
5. Fryer, S.L., Schweinsburg, B.C., **Bjorkquist, O.A.,** Frank, L.R., Mattson, S.N., Spadoni, A.D., & Riley, E.P. (2009). Characterization of white matter microstructure in fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research, 33*(3), 514-521.
6. McGee, C.L., **Bjorkquist, O.A.,** Price, J.M., Mattson, S.N., & Riley, E.P. (2009). Social information processing skills in children with histories of heavy prenatal alcohol exposure. *Journal of Abnormal Child Psychology, 37*(6), 817-830.
7. McGee, C.L., **Bjorkquist, O.A.,** Riley, E.P., & Mattson, S.N. (2009). Impaired language performance in young children with heavy prenatal alcohol exposure. *Neurotoxicology and Teratology, 31*(2), 71-75.
8. McGee, C.L., Fryer, S.L., **Bjorkquist, O.A.,** Riley, E.P., & Mattson, S.N. (2008). Deficits in social problem solving in adolescents with prenatal exposure to alcohol. *The American Journal of Drug and Alcohol Abuse, 34*(4), 423-441.

MANUSCRIPTS UNDER REVIEW

1. Nelson, B.D., **Bjorkquist, O.A.**, Olsen, E.K., & Herbener, E.S. (under review). Functional correlates of anhedonia and neural responses to emotional stimuli in schizophrenia: An fMRI investigation. *Manuscript submitted to Schizophrenia Bulletin.*

2. Olsen, E., **Bjorkquist, O.**, Bodapati, A., Shankman, S., Herbener, E. (under review). Clinical correlates of emotional memory in schizophrenia and major depression. *Manuscript submitted to Schizophrenia Research.*
3. Chase, K.A., Rosen, C., Gin, H., **Bjorkquist, O.**, Feiner, B., & Sharma, R.P. (under review). Metabolic inflammation in schizophrenia: Schizophrenia, obesity, immunity, and epigenetics. *Manuscript submitted.*

PROFESSIONAL CONFERENCE POSTER PRESENTATIONS

Bodapati, A., Herbener, E., Olsen, E., **Bjorkquist, O.,** Chase, K., Rosen, C., & Sharma, R. Attentional bias towards nonsocial relative to social stimuli in schizophrenia. To be presented at the annual meeting of the Society for Research in Psychopathology, Evanston, IL, September 18-21, 2014.

*Buehler, S., Botbol, E., Zalizniak, K., Carrión, C., **Bjorkquist, O.**, Leon, A., Gierut, K., Wilson, M., Marreiro, C., Roundfield, K., Faust, K., Greif, T., Calamia, M., Rao, J., Bodapati, A., Rosado, D., Gorka, S., & Pliskin, N. Measurement of outcome following neuropsychological evaluation: The impact of the evaluation on the consumer. To be presented at the annual meeting of the American Psychological Association, Washington, DC, Aug 7-10, 2014. *Blue Ribbon Award Winner for Best Abstract by Division 40.

Rosado, D., Buehler, S., Botbol, E., Zalizniak, K., Carrión, **Bjorkquist, O**., Leon, A., Gierut, K., Wilson, M., Marreiro, C., Roundfield, K., Faust, K., Greif, T., Calamia, M., Rao, J., Bodapati, A., Rosado, D., Gorka, S., & Pliskin, N. Impact of thorough review of neuropsychological testing results on caregiver coping and perceptions of care-recipient functioning. To be presented at the annual meeting of the American Academy of Clinical Neuropsychology, New York, NY, June 25-28, 2014.

Hu, E., Gin, H., Chase, K.A., **Bjorkquist, O.,** Bodapati, A., & Sharma, R.P. Histone methyltransferase inhibitor activity on the restrictive epigenome of schizophrenia. Presented at the annual UIC Department of Psychiatry Research Extravaganza, Chicago, IL, September 18, 2013.

***Bjorkquist, O.A.**, & Herbener, E.S. Emotion appraisal in schizophrenia: A functional connectivity study. Presented at the annual meeting of the American Academy of Clinical Neuropsychology, Chicago, IL, June 20-22, 2013. *Edith Kaplan Award Winner for Best Trainee Poster Presentation.

Bjorkquist, O.A., Olsen, E., Bodapati, A.S., & Herbener, E.S. Aberrant salience may contribute to impaired social perception in schizophrenia. Presented at the annual meeting of the Society for Research in Psychopathology, Ann Arbor, MI, October 4-7, 2012.

Olsen, E., **Bjorkquist, O.**, Bodapati, A., & Herbener, E.S. Neural activity associated with post emotional processing in schizophrenia. Presented at the annual meeting of the Society for Research in Psychopathology, Ann Arbor, MI, October 4-7, 2012.

Herbener, E., **Bjorkquist, O.,** Olsen, E., Bodapati, A.S., & Nelson, B. Medial temporal and frontal activity during encoding of positive, negative, and neutral images and memory accuracy in schizophrenia and healthy subjects. Presented at the biennial meeting of the International Congress on Schizophrenia Research, Colorado Springs, CO, April 2-6, 2011.

Bjorkquist, O.A., & Herbener, E.S. Social perception in schizophrenia: Evidence of occipital and prefrontal dysfunction. Presented at the annual meeting of the Society for Research in Psychopathology, Seattle, WA, October 7-10, 2010.

Nelson, B.D., Herbener, E.S., & **Bjorkquist, O.A**. Is there an association between trait anhedonia and neural activity in response to emotional pictures in schizophrenia? An fMRI investigation. Presented at the 4th Annual Meeting of the Social & Affective Neuroscience Society, Chicago, IL, October 28-31, 2010.

Behar, E., Kerns, G., Zachrel, E., **Bjorkquist, O.**, Goldwin, M., McGowan, S.K., & Bohacz, B. Validity of a coding system for abstractness and concreteness of thoughts. Presented at the annual meeting of the Association of Behavioral and Cognitive Therapy, San Francisco, CA, November 18-21, 2010.

McGowan, S.K., Goldwin, M.A., **Bjorkquist, O.**, Bohacz, B., McLaughlin, K.A., & Behar, E.B. The effects of positive, negative, and neutral preservative thinking on abstractness and concreteness of thought content. Presented at the annual meeting of the Association of Behavioral and Cognitive Therapy, New York, NY, November 19-22, 2009.

Bjorkquist, O.A., & Herbener, E.S. Arousal and anhedonia predict emotional memory in schizophrenia. Presented at the annual meeting of the Society for Research in Psychopathology, Minneapolis, MN, September 10-13, 2009.

Herbener, E.S., **Bjorkquist, O.A.**, & Palmer, M.A. Impairment in long-term retention of preference conditioning in schizophrenia. Presented at the annual meeting of the International Congress of Schizophrenia, San Diego, CA, March 28-April 1, 2009.

Wagner, A.E., McGee, C.L., **Bjorkquist, O.A.**, & Mattson, S.N. Alterations in perceived organization of hierarchical figures in children with heavy prenatal alcohol exposure. Presented at the annual meeting of the Research Society on Alcoholism, Washington, DC, June 28-July 2, 2008.

McGee, C.L., **Bjorkquist, O.A.**, Price, J.M., Mattson, S.N., & Riley, E.P. Social information processing skills in children with histories of heavy prenatal alcohol exposure. Presented at the fetal alcohol spectrum disorders study group at the annual meeting of the Research Society on Alcoholism, Washington, DC, June 28-July 2, 2008.

Riley, E.P., & **Bjorkquist, O.** Could alterations in brain and behavior in children with FASD be related to stress? Presented at the first international meeting "Alcoholism and Stress: A Framework for Future Treatment Strategies," Volterra, Italy, May 6-8, 2008.

Fryer, S.L., Schweinsburg, B.C., **Bjorkquist, O.A.**, Frank, L.R., Mattson, S.N., Spadoni, A.D., & Riley, E.P. Evidence of global white matter microstructural damage in fetal alcohol spectrum disorders. Presented at the 37th annual meeting of the Society for Neuroscience, San Diego, CA, November 3-7, 2007.

McGee, C.L., **Bjorkquist, O.A.**, Riley, E.P., & Mattson, S.N. Impaired language performance in young children with heavy prenatal alcohol exposure. Presented at the 35th annual meeting of the International Neuropsychological Society, Portland, OR, February 7-10, 2007.

RESEARCH EXPERIENCE

2008-present **Graduate Research Assistant**, Herbener Research Laboratory
Departments of Psychology and Psychiatry, University of Illinois at Chicago

- Assisted in fMRI projects examining emotional memory in individuals with schizophrenia including scan acquisition, data preparation, management, and analysis using AFNI.
- Administered behavioral test batteries including WRAT, WASI, and computerized tasks measuring emotion processing and social cognition in schizophrenia.
- Assisted in subject recruitment and coordination of studies.
- Created tasks targeting social cognitive functioning in schizophrenia.

Supervisor: Ellen Herbener, Ph.D.

2010-present **Graduate Research Assistant**, Hill Research Laboratory
Department of Psychiatry, University of Illinois at Chicago

- Performed data analysis for a fMRI project examining cognition in people with schizophrenia, including scan acquisition, image preprocessing, and group analyses using AFNI.
- Managed and analyzed fMRI behavioral data.
- Administered neuropsychological test batteries including MATRICS, WRAT, WASI, and computerized tasks of serial order processing.

Supervisor: Scot Hill, Ph.D.

2012-present **Graduate Research Assistant**, Epigenetics Translational Laboratory

Department of Psychiatry, University of Illinois at Chicago
- Administered MATRICS battery and Heinrichs-Carpenter Quality of Life scale to inpatients and outpatients with schizophrenia spectrum disorders, bipolar disorder, and healthy controls.

Supervisors: Rajiv Sharma, Ph.D. and Cherise Rosen, Ph.D.

2013-present **Graduate Research Assistant**, Neuropsychology Service
Department of Psychiatry, University of Illinois at Chicago
- Administered phone interviews assessing quality of life, levels of stress, caregiver burden, and feedback on services to patients and caregivers for a study assessing the clinical utility of neuropsychological services.

Supervisor: Neil Pliskin, Ph.D., A.B.P.P.-CN

2006-2008 **Research Assistant**, Center for Behavioral Teratology
Department of Psychology, San Diego State University
- Assisted in MRI, fMRI, and DTI projects on children with Fetal Alcohol Spectrum Disorder (FASD) including scan acquisition, data preparation, and image analysis using AFNI.
- Administered and scored neuropsychological test battery measuring social information processing in children with FASD.
- Authored scholarly publications, and edited grants and presentations.

Supervisors: Edward Riley, Ph.D. and Sarah Mattson, Ph.D.

SUPERVISION EXPERIENCE

2013-present **Psychological Assessment**
- Supervised graduate students in neuropsychological assessment, including administration, scoring, and interpretation of the WAIS and WJ-III Achievement & Cognitive Abilities

2013-present **Psychological Interviewing**
- Supervised undergraduates in conducting psychological interviews
- Provided one-on-one clinical supervision

TEACHING EXPERIENCE

2013 **Teaching Assistant-Psychological Interventions**
- Supervised undergraduates in developing, implementing, and writing a research paper on behavioral interventions

2012 **Teaching Assistant-Field Work in Applied Psychology**
- Supervised undergraduates in completing field research and writing senior research theses
- Created and implemented a lecture on writing an introduction section of a research paper, including idea development, literature review, study goals and hypotheses.

2011 **Teaching Assistant-Statistical Methods in Behavioral Sciences**
- Led two weekly discussion sections and graded papers

INVITED ADDRESSES

April 2014 **Frontotemporal Dementia.** University of Illinois-Chicago, Undergraduate Clinical Psychology Laboratory, Chicago, IL

March 2014 **Frontotemporal Dementia.** NorthShore University HealthSystem, Neuropsychology Externship, Evanston, IL

June 2013 **Traumatic Brain Injury in Torture Survivors: Assessment, Diagnosis, and Intervention. Report on Kovler's Pilot Study.** Marjorie Kovler Center, Chicago IL

April 2013 **Neuropsychological Aspects of Epilepsy Surgery.** University of Illinois-Chicago, Neuropsychology Externship, Chicago, IL

Nov 2010 **A Challenging Neuropsychological Assessment Case.** Department of Psychology, University of Illinois at Chicago

Nov 2010 **Social Perception in Schizophrenia: Evidence of Occipital and Prefrontal Dysfunction.** Department of Psychology, University of Illinois at Chicago

AD HOC REVIEWER FOR SCIENTIFIC JOURNALS

Psychiatry Research
*Journal of Abnormal Psychology**
*Biological Psychiatry**
* conducted under the supervision of Ellen Herbener, Ph.D.

SERVICE TO THE UNIVERSITY

2013 **Clinical Psychology Division Liaison,** Department of Psychology, University of Illinois at Chicago

- Work with faculty and students on departmental issues. Update and maintain clinical handbook.
- Organize department sponsored speakers and events.

PROFESSIONAL REFERENCES

Ellen Herbener, Ph.D. (Ph.D. mentor)
Associate Professor of Psychology & Psychiatry
Departments of Psychology and Psychiatry
1007 W. Harrison Street
University of Illinois at Chicago
Chicago, IL 60607-7137
Phone: (312) 413-4612
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Neil Pliskin, Ph.D., A.B.P.P-CN
Professor of Clinical Psychiatry and Neurology
Director, Neuropsychology Service & Neurobehavior Program
Department of Psychiatry
University of Illinois at Chicago
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Chicago, IL 60612
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Jerry Sweet, Ph.D., A.B.P.P-CN
Clinical Professor, Psychiatry & Behavioral Neuroscience
Director, Neuropsychology Service
NorthShore University HealthSystem
Affiliate of University of Chicago, Pritzker School of Medicine
909 Davis Street, Suite 160
Evanston, IL 60201
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Stewart Shankman, Ph.D.
Associate Professor of Psychology and Psychiatry
Director of Clinical Training (DCT)
Departments of Psychology and Psychiatry
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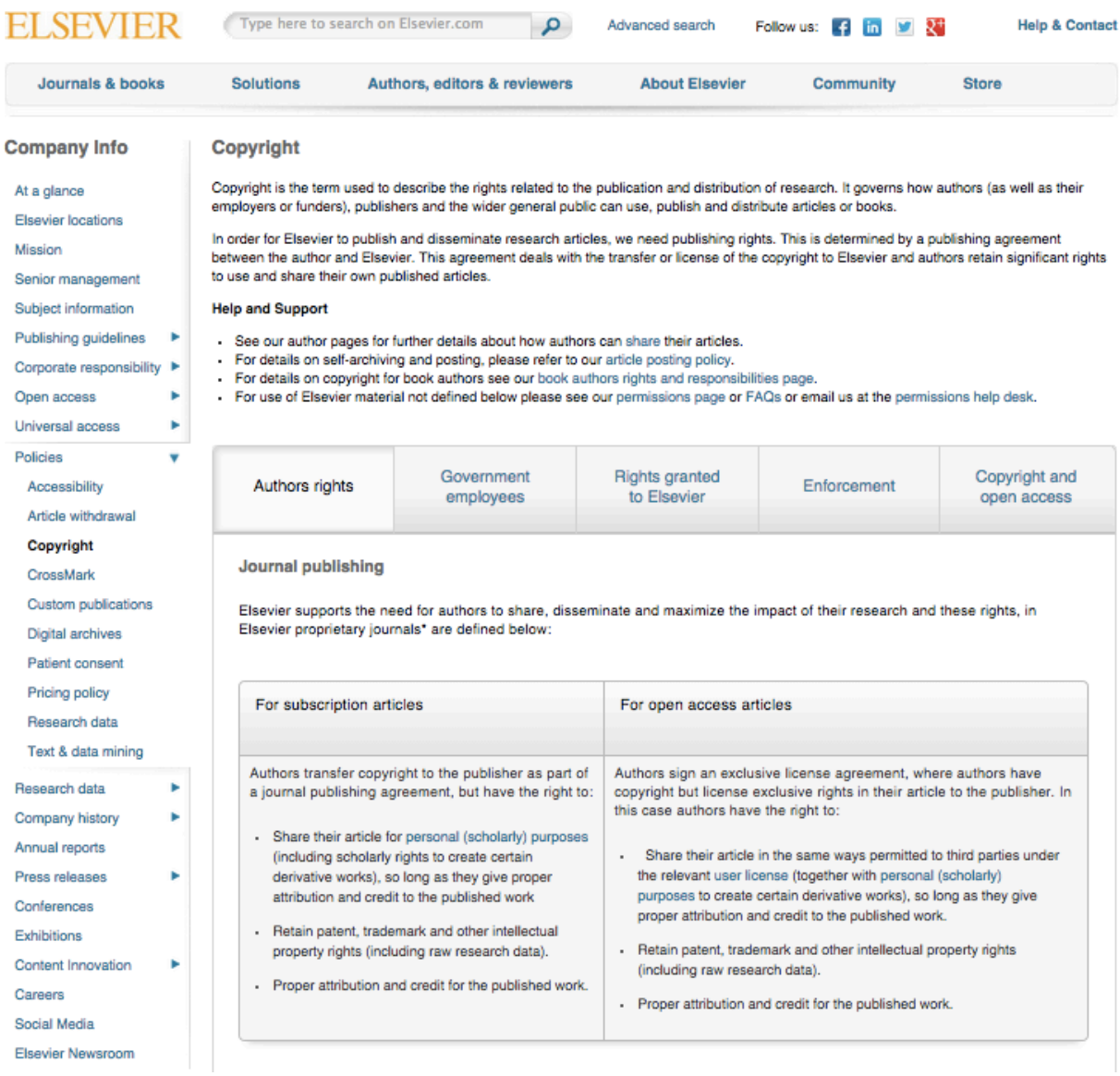

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