Positive emotion processing deficits in schizophrenia

Gregory P Strauss
University of Nevada, Las Vegas

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POSITIVE EMOTION PROCESSING
DEFICITS IN SCHIZOPHRENIA

by

Gregory P. Strauss

Master of Arts
University of Nevada Las Vegas
2004

Bachelor of Science
University of Georgia
2002

A dissertation submitted in partial fulfillment of the requirements for the

Doctor of Philosophy Degree in Psychology
Department of Psychology
College of Liberal Arts

Graduate College
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Gregory Paul Strauss

Entitled

Positive Emotion Processing Deficits in Schizophrenia

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ABSTRACT

Positive Emotion Processing Deficits in Schizophrenia

by

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Dr. Daniel N. Allen, Examination Committee Chair
Associate Professor of Psychology
University of Nevada Las Vegas

Affective impairments were examined in patients with and without deficit syndrome schizophrenia. A battery of tests designed to measure emotional experience, emotional information processing, and emotional perception were administered to deficit (n = 15) and non-deficit syndrome (n = 26) schizophrenia patients classified according to the Schedule for the Deficit Syndrome, and matched non-patient control subjects (n = 22). As predicted, in comparison to non-deficit patients and controls, deficit syndrome patients reported less frequent and intense experience of positive emotion, recalled significantly fewer positive words, and displayed an impaired ability to accurately identify and judge the valence of pleasant odors. Additionally, deficit patients demonstrated a unique failure to have their attention captured by positive information, as well as less accurate and efficient labeling of positive faces than non-deficit patients or controls. Abnormalities were also associated with negative emotions, such that
deficit syndrome patients demonstrated impairment at identifying fearful faces, were less accurate at judging negative smells, had a bias toward recalling anger words, and displayed an elevated attentional lingering effect for negative information. These findings indicate that the deficit syndrome is associated with affective disturbances that impact a number of cognitive and sensory domains, and provide support for the notion that abnormalities may be most severe in relation to the experience and processing of positive emotions. These abnormalities may be due to a mood-congruent processing abnormality, and are consistent with the notion that frontal and limbic system dysfunction may be core to deficit syndrome schizophrenia.
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CHAPTER 1

INTRODUCTION

For years, schizophrenia was viewed as something of an enigma. It has generally been conceptualized as a mental disturbance beyond the reach of science, more of a metaphysical problem than a brain disease (Green, 2000). However, this is no longer the case. Recent breakthroughs in neuroscience and medicine have resulted in the delineation of etiological factors unique to schizophrenia. These factors encompass a wide range of areas, including structural and functional brain abnormalities, cognitive deficits, and neurochemical impairments. Despite significant advances in our understanding of schizophrenia, recent treatments protocols have proven ineffective at remediating some of the more chronic and debilitating psychotic symptoms (Tandon, Jibson, Taylor, & DeQuardo, 1995). This is particularly true regarding emotional disturbances that accompany negative psychotic symptoms, such as flat affect, curbed interests, and diminished emotional experience (Carpenter, Heinrichs, & Wagman, 1988). Although negative symptoms have been shown to be responsive to newer “atypical” antipsychotic medications, it is apparent that some patients evidence residual negative symptoms that are unaffected by treatment (Carpenter et al., 1995). If further advances are to be made in the
treatment of negative symptoms of schizophrenia, researchers will need to investigate its etiology in a fresh new way.

In a recent critique of the field, Seligman and Csikszentmihalyi (2000) proposed that Psychology may benefit from adopting a new perspective. They suggested that mental health has historically focused on remediating psychological symptoms and weaknesses, devoting much of its energy to "curing" disorders associated with negative emotions (e.g., anxiety, depression). As a result, our knowledge of positive emotions and factors that promote them remains largely wanting. Although there are many consequences associated with failing to research positive emotions, the fact that we have little understanding of disorders that experience and process positive emotion abnormally poses a significant problem. This may be particularly true of individuals with schizophrenia who demonstrate deficits in experiencing and perceiving positive emotions. The failure to investigate schizophrenia as a disorder of aberrant positive emotion processing may account for current difficulties in treating chronic symptomatology, such as residual negative symptoms. However, without further knowledge of cognitive and neural substrates that maintain these abnormalities, we are unlikely to remediate unremitting negative symptoms or increase the quality of life of persons with schizophrenia. The current study will provide the first systematic attempt at delineating positive emotion processing deficits in persons with schizophrenia, placing particular emphasis on how such deficits contribute to core negative symptoms.
Clinicians have long noted that individuals with schizophrenia evidence severe emotional disturbances, particularly related to the experience and expression of positive emotion. Original conceptualizations of the disorder proposed by Kraepelin (1919) and Bleuler (1911, 1950) posited that schizophrenia was essentially the splitting of cognition and affect. These conjectures were based upon clinical observations that patients are particularly impaired at producing facial expressions of happiness. Although clinicians largely agreed that patients were deficient at expressing emotion, there was disparity regarding whether difficulties in emotional expression accurately reflected patients’ inner-experience of emotion. Both Kraepelin and Bleuler proposed that patients were capable of experiencing a wide-range of emotions, despite being able to express them. Although these claims were based upon patient self-report, other clinicians contended that some patients were still unable to experience emotion (Rado, 1953). Rado proposed this to be particularly true of positive emotional experience, and described schizophrenia as a disorder characterized by an “integrative pleasure deficiency”.

After several decades of empirical investigation, we now know these conjectures of each of these sets of early clinicians to be largely true. Recent research suggests that individuals with schizophrenia are deficient at both expressing and experiencing emotions. Compared to healthy individuals, patients exhibit less observable positive facial expressions, and report experiencing elevated levels of negative emotion (Aghevli, Blanchard, & Horan, 2003; Berenbaum & Oltmanns, 1992; Kring et al., 1993; Kring & Neale, 1996; Earnst &
Kring, 1999). And some patients, namely those exhibiting negative symptoms, may also experience significant reductions in experiencing positive emotions (Horan & Blanchard, 2003; Suslow et al., 2005). These affective deficits significantly differ from healthy individuals, who typically report being in a moderately positive mood (e.g., 5.50 on a 7.0-point scale), and demonstrate the ability to express positive affect (i.e., smile) from an early age (see Matlin & Stang, 1978). Thus, healthy individuals exhibit a Pollyanna tendency (i.e., tend to be in a positive mood, express high levels of positive emotion, and process positive information accurately and efficiently), while persons with schizophrenia may both express and experience positive and negative emotions abnormally.

Several studies investigating the relationship between emotional expression and experience provide direct clarification of debates posed by early clinicians. Results of multiple studies suggest that the relationship between emotional experience and expression may be disjunctive in patients with schizophrenia (Aghevli, Blanchard, & Horan, 2003; Berenbaum & Oltmanns, 1992; Kring et al., 1993; Kring & Neale, 1996; Earnst & Kring, 1999). That is, patients are often unable to produce emotional expressions, yet report experiencing a wide range of emotions. However, it should be noted that this relationship significantly differs among patients meeting the criteria for deficit syndrome schizophrenia (Horan & Blanchard, 2003). Individuals meeting criteria for this putative schizophrenia subtype evidence a more severe form of negative symptoms, characterized by six specific deficit symptoms: restricted affect, diminished emotional range, poverty of speech, curbing of interests, diminished
sense of purpose, and diminished social drive (Carpenter et al., 1988). Deficit syndrome schizophrenia is characterized by unique etiological, cognitive, and neurological abnormalities (Kirkpatrick et al., 2001). These patients also appear to be uniquely deficient at experiencing positive emotions (Horan & Blanchard, 2003), and display significantly less positive facial expressions than non-deficit syndrome patients (Earnst & Kring, 1999). When recent research is viewed in relation to early clinical theories, it appears that the majority of individuals with schizophrenia display emotional disturbance in line with the views of Kraepelin and Bleuler. However, a distinct group of patients, those meeting criteria for the deficit syndrome, may conform to Rado's (1953) notion that schizophrenia is characterized by positive emotion deficits. Thus, schizophrenia characterized by severe negative symptoms may reflect a more severe form of emotional disturbance.

Several studies also suggest that deficit and non-deficit schizophrenia patients also differ with regard to the perception of emotional information. The recognition of emotional facial expressions has been found to be particularly impaired in patients with schizophrenia (see Edwards et al., 2001 for review). However, results are inconclusive regarding which emotions are most significantly impaired, and whether differences exist between deficit and non-deficit patients. Several studies conducted on non-deficit schizophrenia reported significant impairment in processing happy faces (Archer et al., 1994; Bellack et al., 1996; Loughland, Williams, & Gordon, 2002a; Loughland, Williams, & Gordon, 2002b; Phillips et al., 1998; Sachs et al., 2004; Schneider et al., 1995).
while several others reported intact positive emotion identification and significant impairment for fear (Gaebel & Wolwer, 1992; Johnson, Emde, Scherer, & Kilnert, 1986; Mandal, 1987, 1998; van der Gaag & Haensen, 1990; Wolwer et al., 1996; ) and sadness (Kohler et al., 2000; Edwards, Pattison, Jackson, & Wales, 2001). Only two studies investigated facial affect identification in deficit syndrome patients. Although findings from these two studies are mixed, some evidence indicates that deficit syndrome patients evidence poorer recognition than non-deficit patients, and that these deficits are more severe for positive emotions, such as surprise (Bryson et al., 1998).

Deficit syndrome patients have also been shown to evidence more severe olfactory perception impairments than non-deficit patients and controls (Goudsmit et al., 2004; Malaspina et al., 2001; Secklinger et al., 2002). These smell identification impairments have been demonstrated to be correlated with neurocognitive tests thought to measure frontal and parietal structures, regions that have not been implicated in smell identification abnormalities in non-deficit schizophrenia patients (Secklinger et al., 2002). Considering overlap between neural circuits involved with emotion processing and olfaction, researchers have also attempted to determine whether individuals with schizophrenia are impaired at recognizing olfactory valence. Recent investigations have found that individuals with schizophrenia display impairment at judging the pleasantness of odors (Crespo-Facorro et al., 2001; Hurdy et al., 2002; Moberg et al., 2003), and that these impairments are associated with greater negative symptom severity (Doop & Park, 2006). These findings have yet to be extend to deficit syndrome
patients; however, it would be important to do so considering that deficit patients
display the most severe olfactory impairments noted in patients with
schizophrenia (Goudsmit et al., 2004; Malaspina et al., 2001; Secklinger et al.,
2002), since impaired olfactory hedonic judgment is associated with greater
severity of negative symptoms, and because frontal and limbic system
dysfunction thought to underlie general olfactory identification and valence
judgment are also thought to be core to negative symptoms associated with the
deficit syndrome (Kirkpatrick et al., 2001).

Individuals with schizophrenia also evidence deficits in the cognitive
processing of emotional information, and there is some suggestion that these
impairments may be more severe in patients with negative symptoms.
Impairments have been noted in patients with schizophrenia in relation to
emotional memory (Calev, 1996; Calev & Edelist, 1993; Exner, Boucsein, Degner,
Irle, & Weniger, 2004; Koh, Grinker, Marusarz, & Forman, 1981), with the
majority of studies indicating that deficits are more severe for the recall of
positive than negative information (Calev, 1996; Calev & Edelist, 1993; Exner et
al., 2004; Koh et al., 1981). A recent investigation also demonstrated that these
positive emotion memory impairments may be more pronounced in patients
displaying negative symptoms, particularly anhedonia (Herbener et al., 2007).
Research also suggests that patients evidence aberrant automatic affective
processing, and that these deficits may be more prominent for positive
information (Suslow, Roestel, Droste, & Arolt, 2003) and particularly in patients
with negative symptoms (Suslow et al., 2005). However, it has also been noted
that schizophrenia patients display an inherent attention bias for threatening information (Epstein, Stern, & Silbersweig, 1999; Fear, Sharp, & Healy, 1996), suggesting that negative information may also disrupt automatic cognitive processes. Thus, several studies suggest that patients exhibiting negative symptoms of schizophrenia may display affective disturbances that are more severe than patients without negative symptoms, and that these abnormalities may be more severe for the processing of positive information. Additionally, these positive emotion processing abnormalities appear to be pervasive, extending across multiple cognitive domains, including face perception, smell perception, memory, and attention, as well as emotional experience and expression. However, additional studies are needed to determine whether these findings are core to negative symptoms in and of themselves or the result of secondary factors, such as medication effects, depression, and anxiety.

The current study attempts to address these issues and provide the first comprehensive assessment of affective disturbance in patients with deficit and non-deficit syndrome schizophrenia. Several major questions will be examined to achieve this purpose. First, we aim to determine whether deficit patients, non-deficit patients, and controls differ with regard to emotional experience. Specifically, do groups differ with regard to the experience of state and trait positive and negative emotions? Second, do patient groups and controls differ with regard to the perception and cognitive processing of emotional information? More specifically, a) Are deficit syndrome patients more impaired than non-deficit patients and controls at identifying and judging the valence of positive odors?, b)
Do deficit syndrome patients display a selective impairment in processing positive faces accurately and quickly? c) Are deficit patients more impaired than non-deficit patients and controls at remembering positive words? and d) Does positive information fail to capture attention in patients with deficit syndrome schizophrenia?

Answers to these questions have significant potential to inform us about the nature of aberrant neural mechanisms central to emotion processing deficits in patients with negative symptoms of schizophrenia, and may contribute to the development of novel methods of treatment for patients with core negative symptoms, such as those with the deficit syndrome, who tend to be highly treatment resistant.
CHAPTER 2

LITERATURE REVIEW

In the following sections, literature relevant to the current proposal is reviewed. Discussion involves two primary areas: Deficit Syndrome Schizophrenia and Affective Impairment in Schizophrenia. The Deficit Syndrome Schizophrenia section reviews literature relevant to patients with primary negative symptoms of schizophrenia, including: 1) Symptom Heterogeneity, 2) Clinical Characteristics and Classification, 3) Course of Illness, 4) Risk Factors, and 5) Neuropsychological Impairment. Sections reviewed in the Affective Disturbance section include: 1) Affect Expression and Experience, 2) Facial Affect Processing, 3) Olfactory Affect Impairment, 4) Memory for Affective Information, and 5) Automatic Processing.

Deficit Syndrome Schizophrenia

Symptom Heterogeneity

Schizophrenia is now widely regarded as a disorder characterized by substantial heterogeneity, particularly in relation to symptom presentation. The symptoms of schizophrenia are commonly classified according to positive and negative features. Traits categorized as positive psychotic features are those that involve behavioral excess, and typically include symptoms such as
hallucinations, delusions, and disorganized speech (Andreasen & Olsen, 1982). In contrast, negative features are described as behavioral deficiencies, including symptoms like anhedonia (inability to experience pleasure), flat affect (No outward expression of emotion), asociality (poor social skills), alogia (poverty of speech and thought), and avolition (lack of motivation and energy).

Negative symptoms have received significant attention in the research literature due to difficulty associated with their treatment. It is clear that some patients experience negative symptoms that neither respond to conventional or atypical antipsychotic medications (Carpenter et al., 1995). Due to the high incidence of residual negative symptoms (Carpenter, Heinrichs, & Wagman, 1988), researchers have attempted to develop classification systems that divide symptom profiles into distinct subtypes that are more amenable to treatment.

The most enduring of these classifications is the delineation of primary and secondary negative symptoms. Secondary negative symptoms are those features that are the result of medication, exacerbation, or psychotic processes (Kirkpatrick et al., 1989). In contrast, primary negative symptoms are stable trait-like features, which persist throughout durations of symptom stability (Carpenter et al., 1988). Primary negative symptoms are commonly defined as those negative symptoms (e.g., affective disturbance, alogia, anhedonia) that occur independent of medication side-effects (secondary negative symptom), and are not attributable to clinical features like depression, anxiety, or paranoia (Kelley, van Kammen, & Allen, 1999). Patients who display multiple primary negative
symptoms, which are consistently present for greater than 1 year, are classified as having "Deficit Syndrome" schizophrenia.

Clinical Characteristics and Classification

The deficit syndrome classification has received considerable attention in the research literature, as it has been found to predict a number of outcomes. For example, differences have been noted between deficit and non-deficit syndrome schizophrenia patients with regard to neurological processes, cognitive functioning, emotional disturbance, clinical symptomatology, course of illness, risk factors, structural brain abnormalities, and treatment response (See Kirkpatrick, 2001 for a review). Differentiation between deficit and non-deficit patients provides support for the validity of this classification, and its use in clinical and research purposes.

Although there are a number of negative symptoms common to patients with schizophrenia, those meeting criteria for the deficit syndrome primarily display impairment in relation to volition and emotionality. As detailed by Kirkpatrick et al (2001; SDS Manual), deficit patients display clinical features that are highly similar to individuals meeting criteria for schizoid personality disorder. Like schizoid patients, they tend to have little interest in people and things, and evidence a lack of emotionality and liveliness. They also display significant impairment in behavioral initiation. For instance, despite being capable of doing so, they typically initiate thoughts, emotions, and motor behaviors less frequently than other individuals. Thus, deficit syndrome patients are unique in that they display a lack of liveliness and interest in people and things that is uncommon,
both in comparison to the general population and individuals with schizophrenia alike.

Deficit syndrome status is evaluated in relation to 6 specific negative symptoms: Restricted Affect, Diminished Emotional Range, Alogia, Curbed Interests, Diminished Sense of Purpose, and Diminished Social Drive. Restricted affect refers to a patient's overt expression of emotion. Relative to other people, deficit patients display reduced expression of emotion in facial and vocal channels, and in their use of expressive nonverbal cues. In contrast to the observable emotional phenomenon rated for Flat Affect, the estimation of Diminished Emotional Range reflects a patient's subjective inner-experience of emotion. In comparison to others, deficit patients commonly report experiencing less frequent and intense levels of both positive and negative emotions during clinical interview. They tend to enjoy less in life, are not as easily upset, and seldom experience anger or irritation. Alogia, the third deficit syndrome symptom, has also been termed poverty of speech. This item reflects a patient's proclivity to both use fewer words than what would be considered normal, and convey less information than what is needed to express an idea fully.

Deficit patients also display significant volitional impairments. One of the most core of these symptoms is that of Curbed Interests. In comparison to others, deficit patients display little interest in the external world. They may spend less time thinking about things, rarely attempt to learn new information, and engage in fewer hobbies or activities than what is normal. This may be reflected both in terms of breath or depth of interest. They also display a significant
reduction in relation to experiencing drive for goal-directed activity. Kirkpatrick et al. (2001; SDS Manual) term this symptom, Diminished Sense of Purpose, to reflect a loss in the ability to independently generate goals for one's life, sustain goal-directed activity, and spend time in purposeful activities. Deficit patients therefore spend an inordinate amount of time engaging in aimless activity (e.g., passively sitting and staring into space, smoking cigarettes on a bench), and have little or no desire to improve the daily situation of their lives.

However, a lack of drive to interact with others is perhaps the most core feature of deficit syndrome pathology. In comparison to others, deficit patients do not want substantial amounts of social contact, and importantly, this is not due to anxiety. Unlike Avoidant patients who withdraw from social situations because these situations produce anxiety, even though they long for contact with others, deficit patients simply do not have the normal drive for affiliation inherent to most individuals. Thus, deficit syndrome patients spend less time around others, prefer to complete activities alone, and feel no sense of loneliness even when they go for long periods of time without significant human contact.

It is important to note that the concept of deficit syndrome schizophrenia differs from the traditional view of negative symptoms of schizophrenia in several ways. First, negative symptoms seen in deficit syndrome patients are the most prominent aspect of their clinical presentation. In other words, negative symptoms are the most severe, distressing, and functionally debilitating aspect of the patient's condition. It is not necessarily true that deficit syndrome patients are devoid of positive psychotic symptoms, which are in fact required to be present in
some combination for patients to meet criteria for schizophrenia. These symptoms are simply not as severe or impacting as what is seen in non-deficit patients.

Second, negative symptoms manifested by deficit syndrome patients are primary. That is, negative symptoms are idiopathic and not secondary to factors other than the disease process that are common to schizophrenia. Such secondary factors include: anxiety, medication effect, suspicion (and other positive psychotic symptoms), mental retardation, and depression. The primary/secondary symptom discrimination is often a difficult one considering that prominent negative symptoms can result from a variety of factors. For example, prominent negative symptoms may be brought on by: behavioral withdrawal from the environment, depressive mood, demoralization, antipsychotic medication effects, exposure to an understimulating environment, or neurological processes core to the disease process of schizophrenia. To determine whether these symptoms are caused by primary or secondary factors, it is common to use longitudinal observation or empirical manipulation. It is often necessary to examine patient records during periods of medication change or withdrawal to identify fluctuations in negative symptoms, to interview family members or treatment staff to track the patient’s history of negative symptoms, and assess the effects of antiparkinsonian medication use on the manifestation of flat affect.

Third, negative symptoms of the deficit syndrome also differ in that they must be enduring. In the deficit syndrome classification, as made using the gold standard method of the Schedule for the Deficit Syndrome (Kirkpatrick et al.,
enduring is defined as a period of 12 months prior to interview. Thus, negative symptoms of the deficit syndrome must be stable throughout a 1 year period. Such stability reflects that these negative symptoms are core to the neurological influences of the disease process of schizophrenia in and of itself, and not other causes, such as those previously discussed. During periods of symptom remission or psychotic disorganization, it is still usually possible to diagnose the presence of the deficit syndrome.

Thus, diagnosis of the deficit syndrome of schizophrenia requires the patient to meet several very specific criteria, which are designed to reduce heterogeneity of schizophrenia and identify a homogeneous subgroup of patients with negative symptoms. Specific criteria for the deficit syndrome include (from Kirkpatrick, Buchanan, Ross, & Carpenter, 2001, p 166):

1). At least 2 of the following 6 features must be present and of clinically significant severity: Restricted affect, Diminished emotional range, Poverty of speech, Curbing of interests, Diminished sense of purpose, Diminished social drive. 2.) Two or more of these features must have been present for the preceding 12 months, and always have been present during periods of clinical stability (including chronic psychotic states). These symptoms may or may not be detectable during transient episodes of acute psychotic disorganization or decompensation. 3.) Two or more of these enduring features are also idiopathic (i.e., not secondary to factors other than the disease process. Such factors include: Anxiety, Drug effect, Suspiciousness, Formal thought disorder, Hallucinations and delusions, Mental retardation, Depression. 4.) The patient meets DSM criteria for schizophrenia.

The following sections detail deficit syndrome literature relevant to the current investigation.
Course of Illness

The deficit syndrome has been described as a more severe form of schizophrenia, with a distinct and pervasive course of illness. Compared to non-deficit patients, deficit schizophrenia is characterized by poorer premorbid functioning before the onset of positive symptoms, particularly with regard to social functioning (Fenton & McGlashan, 1994; Kirkpatrick, Ram, & Bromet, 1996; Kirkpatrick et al., 1996; Buchanan, Kirkpatrick, Heinrichs, & Carpenter, 1990). Some evidence suggests that these premorbid impairments may be pervasive, continuing to affect functioning throughout life. Social and occupational abnormalities persist into early and middle adulthood, maintaining presence over multiple assessments and long-term follow-up (Fenton & McGlashan, 1994). However, it has yet to be determined whether the severity of these symptoms progresses with age. This would be an important factor to assess considering that patients with schizophrenia show differential patterns of premorbid impairment throughout adolescence (Allen, Frantom, Strauss, & van Kammen, in press).

Risk Factors

Deficit syndrome schizophrenia is also associated with specific risk factors, which are not prevalent in simple schizophrenia. Four primary risk factors are associated with deficit syndrome schizophrenia: Summer birth, prevalence of borna disease virus antibodies, familial history, and male gender.

Seasonality of birth has been associated with the development of several psychiatric disorders. For example, winter birth is highly associated with Bipolar
disorder and schizoaffective disorder, while spring birth is significantly correlated with major depression and autism (Torrey, Miller, Rawlings, & Yolken, 1997). Winter birth month has also been repeatedly correlated with development of schizophrenia (Torrey et al., 1997); however, the biological substrates governing this relationship are not well understood. It has been hypothesized that this relationship may exist due to multiple factors, including gene combination effects, weather, infectious substances/toxins, and birth complications (Torrey et al., 1997). Considering that winter birth is a significant risk factor for development of schizophrenia, it is of considerable interest that summer birth is highly associated with development of the deficit syndrome (Kirkpatrick et al., 1998, 2000, 2001). The relationship between summer birth and deficit syndrome has been reported in several published studies (Kirkpatrick et al., 1998, 2000), and confirmed by multiple unpublished investigations reviewed by Kirkpatrick et al. (2001). Despite the prevalence of these findings, their biological underpinnings are not well delineated. Regardless of the precise cause, summer birth appears to be a unique marker for deficit syndrome schizophrenia.

Viral acquisition has also been associated with the development of several psychiatric disorders (e.g., dementia of Alzheimer's type; de la Torre et al., 1996), including schizophrenia. The deficit syndrome has been specifically linked to an immune system depleting virus that results in meningoencephalitis, the Borna Disease Virus (Waltrip et al., 1997). Borna disease virus antibodies have been found to be significantly higher in deficit compared to non-deficit patients (Iwahashi et al., 1998; Waltrip et al., 1997). This disease is thought to
significantly affect limbic system regions (e.g., hippocampus, basal ganglia), and potentially disrupt neurotransmitter binding in the DA circuit (Solbrig et al., 1994). Considering distinct emotional and neurochemical abnormalities that accompany the deficit syndrome, it seems plausible that Borna Disease infection is a unique risk factor for the development of schizophrenia with primary negative symptoms.

Familial relation also increases the risk for developing deficit syndrome schizophrenia (Dollfus, 1996; Kirkpatrick, Castle, Murray, & Carpenter, 2000). Genetic-linkage studies suggest that first-degree relatives of deficit patients are at significantly higher risk for developing schizophrenia than relatives of non-deficit patients (Dollfus, 1996). Kirkpatrick et al. (2000) report the risk of developing schizophrenia to be approximately 1.75 times greater in deficit than non-deficit probands. When risk for schizophrenia is compared within families, results suggest that the diagnosis of deficit schizophrenia in one sibling makes the diagnosis of deficit schizophrenia 3 times more likely than developing non-deficit syndrome in another sibling diagnosed with schizophrenia (Ross et al., 2000). Additionally, genes associated with the pathophysiology of deficit syndrome may uniquely relate to the development of social skill impairments, as deficit probands experience significantly more social isolation compared to non-deficit probands. Social skills abnormalities noted in deficit syndrome relatives do not appear to be due to greater incidence of depression. In fact, relatives of deficit patients experience significantly less dysphoria than non-deficit relatives (Ross et al., 2000). These findings suggest that deficit syndrome schizophrenia
has a distinct familial relationship, and that genes associated with this subtype are highly associated with social dysfunction.

Male gender has also been associated with increased risk for deficit syndrome. Roy (2001) conducted a meta-analysis of 23 studies investigating deficit and non-deficit syndrome to determine the relative proportion of male-female patients. In the 23 studies examined, approximately 29% of individuals with schizophrenia met deficit syndrome criteria (Range = 16.5 - 50%). Of those 29% of participants meeting deficit criteria, 63% were male. Roy concluded that the significantly higher proportion of males meeting diagnosis may reflect that sex hormones are related to the etiology of deficit syndrome schizophrenia.

Cognitive Deficits

As previously noted, schizophrenia is characterized by a wide-range of cognitive deficits. Although the majority of persons with schizophrenia evidence significantly poorer mental abilities than controls, some patients have also been reported to appear neuropsychologically normal (Allen et al., 2003). In an attempt to reduce this heterogeneity in cognitive performance, researchers have attempted to study cognition in relation to distinct subtypes. Investigations of the effects of negative symptoms on cognition have revealed significant associations with psychomotor speed, verbal memory (O'Leary et al., 2000), executive functioning (Bell et al., 1997), and semantic fluency (Chen et al., 1996). Since it has been noted that negative symptoms and the deficit syndrome are qualitatively different and maintained by distinct etiological factors, researchers have also investigated cognitive impairment specific to the deficit syndrome.
Although schizophrenia is characterized by a wide-range of cognitive impairment, deficit syndrome schizophrenia is characterized by several distinct deficits. The most consistently reported finding involves deficient executive functioning (Bryson, Whelahan, & Bell, 2001; Buchanan, Strauss, Kirkpatrick, and Holsetin, 1994). Bryson et al. (2001) found that deficit patients were approximately -1.0 SD below the performance of non-deficit patients on the WCST. Similar impairments were noted by Buchanan (1994), using the Stroop Color-Word and Trail Making Tests. These impairments suggest that deficit patients perform more poorly than non-deficit patients on tests sensitive to frontal lobe dysfunction.

Several studies have also reported that deficit patients are impaired in tests measuring parietal lobe functioning (Arango et al., 2000; Buchanan et al., 1994). Like executive functioning, these deficits are significantly more severe than those displayed by non-deficit patients; however, parietal dysfunction may be a unique marker for the deficit subtype, as non-deficit patients typically do not differ from controls on these tasks. Impairment on tasks sensitive to parietal lobe functioning may indicate that deficit patients are particularly deficient at integrating sensory processes (e.g., visual and motor senses). Deficit patients have also been differentiated by attentional impairments, particularly with regard to sustained attention (Buchanan et al., 1997), as well as auditory recognition (Lahti, 2001). Collectively, these deficits suggest that individuals with deficit syndrome have impairment to multiple sensory domains, and that severity of performance may be influenced by frontal and parietal lobe dysfunction.
Neuropathology

Abnormalities to specific neural mechanisms may also differentiate deficit and non-deficit schizophrenia. Results from three separate neuroimaging studies suggest that deficit patients evidence significantly less activation in the dorsolateral prefrontal cortex (DLPFC), thalamus, and inferior parietal cortex (Carpenter et al., 1996; Heckers et al., 1999; Tamminga et al., 1992). Additionally, differences between deficit and non-deficit patients in functional activation were not noted for other brain regions known to be deficient in schizophrenia (e.g., hippocampus; Hecker et al., 1999), suggesting that decreased activity in the DLPFC is a unique finding to deficit syndrome, and is not simply the result of a more global pattern of decreased activation throughout the cerebrum. Structural abnormalities have also been noted in relation to the prefrontal cortex, such that deficit patients are known to have significantly lower prefrontal white matter volume (Buchanan et al., 1993) and smaller prefrontal volume in general (Turetsky et al., 1995). When structural and functional brain abnormalities are viewed together, findings point to a disruption in the dorsolateral prefrontal-basal ganglia-thalamocortical circuit (Kirkpatrick et al., 2001). Impairment to this circuit likely contributes to the significant cognitive (i.e., executive functioning and sensory integration) and social skills abnormalities (Kirkpatrick et al., 2001) inherent to deficit syndrome patients. Irregular communication between structures within this circuit may also explain the fact that deficit patients experience significantly less positive affect than healthy controls. This is likely to be true considering the importance of the prefrontal
cortex, basal ganglia, and thalamus in regulating positive emotion (Thorpe, 1983).

Aberrant neurotransmitter systems may also contribute to the cognitive, emotional, and social processes related to the deficit syndrome. Although schizophrenia is typically characterized by higher levels of DA activity, recent evidence suggests that the deficit syndrome may be uniquely influenced by lower brain DA levels. Lower levels of the DA metabolite, homovanillic acid (HVA), have been reported by multiple investigations (Ribeyre et al., 1994; Thibaut et al., 1998); however, one investigation, Nibuya et al. (1995), reported contradictory evidence, indicating that deficit patients evidenced increased HVA levels. It is likely that results indicating lower HVA levels (Ribeyre et al., 1994; Thibaut et al., 1998) provide the most accurate assessment of functioning, as reports of higher HVA concentrations (Nibuya et al., 1995) may be biased by inappropriate diagnosis of the deficit syndrome (Kirkpatrick et al., 2001). Reduced DA activity may provide another compelling explanation for the diminished inner-experience of positive emotion noted in deficit syndrome patients. This explanation seems plausible considering the relationship between higher DA levels and positive affect in healthy individuals (Benninger 1991; Phillips, Blaha, Pfaus, & Blackburn, 1992); however, this association has yet to be directly tested among deficit syndrome patients.

Summary

The deficit syndrome is a putative subtype of schizophrenia. Patients are considered to meet criteria for the deficit syndrome if they exhibit a chronic
presentation of primary negative symptoms that is not due to the effects of 
medication or other psychotic features. The deficit syndrome is characterized by 
a unique etiology not evident in non-deficit patients, as well as specific cognitive 
and neuropsychological deficits. A critical analysis of the emotion processing 
literature also suggests that the deficit syndrome may be uniquely characterized 
by severe positive emotion processing impairments. Unlike non-deficit patients, 
these individuals are typically unable to experience adequate levels of positive 
emotion. They may also demonstrate unique impairments in the perception of 
positive emotion. Positive emotion processing deficits may be mediated by 
structural and functional brain abnormalities that affect the dorsolateral 
prefrontal-basal ganglia-thalamocortical circuit, as well as hypoactive DA activity. 
Further research is needed to determine whether the deficit syndrome is uniquely 
characterized by pervasive positive emotion processing deficits, and to identify 
the neurological substrates that maintain these impairments.

Affective Impairment in Schizophrenia

Overview

Emotion has been defined in numerous ways throughout the past century. 
Some researchers describe emotion as adaptive functions that have allowed for 
individual survival throughout the evolutionary process (Plutchik, 1980; Izard, 1971). Others regard emotion as dimensions or states of consciousness 
(Tellegen, 1985), complex interactions of self-concept and the environment 
(Arnold, 1960), products of cognitive arousal and the appraisal of the situation
that elicited that arousal (Schacter, 1966), and principal motivational systems that
influence cognition and action (Tomkins, 1962). These views are seen as
complimentary, with each theory representing different aspects of emotion.

Current theories classify emotion according to either valence-arousal
(Russell, 1980; Russell & Feldman Barrett, 1999) or discrete basic emotion
models (Ekman, 1992; Izard, 1991; Turner & Ortony, 1992). The valence-arousal
school of thought holds that emotions can be plotted as points within a two-
dimensional space, with axes representing level of arousal (high and low) and
valence (positive vs. negative affect) (Russell, 1980). The two-dimensional model
has been replicated in numerous studies using self-report data (Feldman Barrett
& Russell, 1999), and supported by some neuropsychological investigations (Lee
et al., 2004). The opposing view suggests that discrete emotions exist within
broader positive-negative classifications. This basic emotion model posits that a
set of universally experienced emotions exist, each possessing unique
physiological arousal patterns, behavioral expressions, and cognitive correlates
(Ekman, 1992). Researchers have identified both discrete negative (e.g., disgust,
sadness, fear, anger) (Ekman, 1992; Izzard, 1991) and positive (e.g., joy,
surprise, love, contentment) (Fredrickson & Branigan, 2001) emotions that meet
these characteristics.

Regardless of how one defines emotion, the ability to experience, express,
and perceive emotion is undeniably important. Without the ability to experience
emotions like happiness, sadness, fear, and guilt, existence would be limited to
simple instincts and reflexes, mere actions and reactions that would determine
major life functions. In several psychiatric disorders, emotional experience is known to be abnormal, with some disorders evidencing a reactive affective experience (e.g., anxiety), and others possibly not even experiencing some or any emotions at all (e.g., alexithymia). Although both abnormalities result in significant distress, it is the later that is hypothesized to have more severe social consequences, which is possibly the case with persons with schizophrenia.

Until recently, relatively little research has investigated cognitive and neurobiological bases of affective disturbances in schizophrenia. Recent studies have indicated that persons with schizophrenia evidence impairment in expressing and experiencing emotion (Berenbaum & Oltmanns, 1992; Dworkin et al., 1993; Earnst et al., 1996; Kring & Earnst, 1999; Kring, Kerr, Smith, & Neale, 1993), identifying emotional facial expressions (Archer et al., 1994; Bellack et al., 1996; Loughland, Williams, & Gordon, 2002a; Loughland, Williams, & Gordon, 2002b; Sachs et al., 2004; Schneider et al., 1995; Gaebel & Wolwer, 1992; Mandal, 1987, 1998; van der Gaag & Haenen, 1990; Wolwer et al., 1996; Kohler et al., 2000; Edwards, Pattison, Jackson, & Wales, 2001), differentiating affective speech productions (Billenber & Johnson, 1965; Bozikas, Kosmidis, Anezoulaki, Giannakou, Karavatos, 2004; Edwards et al., 2001; Fricchione, Sedler, & Shukla, 1986; Jonsson & Sjostedt, 1973; Murphy & Cutting, 1990), integrating audio-visual emotion productions (de Gelder et al., 2005), judging the pleasantness of common odors (Crespo-Facorro et al., 2001; Doop & Park, 2006; Hurdy et al., 2002; Moberg et al., 2003), recalling emotionally laden information (Calev, 1996; Calev & Edelist, 1993; Koh, Grinker, Marusarz, & Forman, 1981; Matthews and
Barch, 2004), and automatically processing emotional words (Epstein et al., 
1999; Fear et al., 1996; Suslow, Roestel, Droste, & Arolt, 2003). Affect 
impairment has been noted for both positively and negatively valenced material, 
with the majority of research suggesting that deficits are more severe for positive 
emotion processing. Although it is well-documented that certain neurological 
processes are crucial for normal experience of positive emotion (i.e., normal 
dopamine and left hemisphere activity), and that these brain regions are impaired 
in schizophrenia, it is unknown whether neurological impairments result in 
widespread (i.e., experience, expression, perception, cognition) disturbance 
related to positive emotion, and how such abnormalities are associated with 
clinical characteristics of schizophrenia. The current section discusses emotion 
processing impairments in individuals with schizophrenia, and contrasts those 
deficits with performance of healthy individuals. Specific attention is given to 
 studies investigating negative symptoms of schizophrenia, particularly those 
making distinctions between deficit and non-deficit schizophrenia. 

_Affect Expression and Experience_

Although schizophrenia has been characterized by positive and negative 
symptoms (Andreasen, 1999), neurocognitive abilities have been more recently 
identified as core features of the disorder (Antonova, Sharma, Morris, & Kumari, 
2004), so much so that some have suggested neurocognitive variables be used 
to develop subtypes of schizophrenia (e.g., Turetsky et al., 2002). Recent 
research has also provided evidence confirming the presence of emotional 
disturbances (Berenbaum & Oltmanns, 1992; Knight & Roff, 1985; Kring, Kerr,
Smith, & Neale, 1993), leading some to suggest that affective disturbances too should be considered core features of the disorder (Kring et al., 1993).

Clinical observations provided the impetus for investigations into the affective abnormalities in schizophrenia, as clinicians have historically noted that patients evidence an inability to express affect through facial and vocal channels (Bleuler, 1950; Kraepelin, 1911). Both Kraepelin (1856-1926) and Bleuler (1857-1939) proposed that affective disturbances are central to schizophrenia symptomatology, and considered features like delusions and hallucinations secondary to emotion processing deficits. Although clinicians and researchers alike agree that emotional abnormalities are central to schizophrenia, debate exists regarding the nature of these deficits.

Bleuler (1911, 1950) is credited with proposing a model focusing on the presence of an expressivity deficit. Through clinical observation, Bleuler noted that patients were typically unable to produce adequate displays of affect. Although these deficits appeared to be pervasive, affecting both facial and vocal expressions, patient’s self-reported descriptions of inner-experience were often inconsistent with their observable expressions. That is, although outward facial and vocal affective displays were blunted, patients often reported experiencing a wide-range of emotions, despite being unable to express them.

In contrast to Kraepelin (1911) and Bleuler (1950), other clinicians suggested that deficits in affective expression result from an inability to experience emotion (Rado, 1953). In this sense, Rado (1953) suggested that the inability to outwardly express emotion accurately reflects the inner-experience of
persons with schizophrenia. He particularly thought this true of pleasurable affective experiences, and posited that the inability to experience positive affect (e.g., joy, affection, pride, interest) was a core deficit of the disorder; however, the experience of negative affect (e.g., sadness, fear) was proposed to be intact.

Schizophrenia researchers have focused on elucidating affective experience and expression deficits both in conjunction and individually. In the current section, discussion of both study types is condensed to provide a clearer exploration of valence related deficits. In facial affect expression studies, participants are typically asked to view a short film clip, while their facial movements are recorded using video tape and/or electromyography (EMG). In the former technique, facial actions are coded using standardized systems aimed at monitoring affect displays, and in the later, electrodes are placed on the participant's face and muscle activity is recorded for relevant sites (e.g., eyes, brow, corners of mouth). Studies examining both experience and expression use a combination of the aforementioned procedures. In these studies, mood is typically induced via emotional film clips or social role-play, and participant inner-experience and facial activity are simultaneously monitored using self report methods (e.g., questionnaires, arousal while viewing film clip) and facial recording techniques (e.g., video-recording, EMG), respectively.

Disturbances in facial affect expression are perhaps the most well-delineated emotional impairments evidenced by individuals with schizophrenia. Results of multiple studies consistently suggest that individuals with schizophrenia are deficient in producing expressions of facial affect, both while
viewing mood induction stimuli (e.g., film clips) (Berenbaum & Oltmanns, 1992; Dworkin et al., 1993; Earnst et al., 1996; Kring & Earnst, 1999; Kring, Kerr, Smith, & Neale, 1993; Kring & Neale, 1999) and while engaged in social interaction (Aghevli, Blanchard, & Horan, 2003; Krause, Steimer, Sanger-Alt, & Wagner, 1989; Mattes, Schneider, Heimann, & Birbaumer, 1995). Deficits have typically been discussed in relation to overall emotional expression (i.e., collapsing both positive and negative emotions); however, a closer look at results from previous studies suggests that impairments may be more severe, and perhaps selective to, positive emotions.

All studies reporting valence-specific deficits have indicated that patients evidence more severe impairments in positive affect expression. These deficits may be mediated by distinct physiological abnormalities. For example, patients display significantly fewer zygomatic (lower face) facial movements than orbicular movements (upper face) while viewing positively valenced film clips. (Kring, Kerr, Smith, & Neale, 1993; Earnst et al., 1996) Similar deficits have been reported in social contexts, where positive affect expression is related to fewer movements of the lower face in comparison to controls (i.e., less zygomatic activity/smiling) (Mattes et al., 1995; Aghevli, Horan, & Blanchard, 2003). These deficits do not appear to be due to medication effects, as patient response is reported to be equally diminished during medicated and unmedicated states (Earnst et al., 1996). Additionally, deficits may be unique to positive affect, as patients evidence normal or perhaps greater facial activity (particularly around the brow) than controls while viewing negative film clips (Mattes et al., 1995). These findings
may suggest that positive affect expression deficits are a core characteristic of schizophrenia.

Deficits in emotional expression are not limited to facial display. Studies also suggest that the vocal production of emotion is disrupted in schizophrenia (Cohen & Docherty, 2004; Docherty, 1996; Docherty et al., 1994, 1998; Rosen, Welkowitz, Slobin, & Borod, 1989). In these studies, participants are typically required to produce a speech sample that consists of reading aloud a positively or negatively valenced paragraph. During this procedure, vocal recordings are made, and later analyzed for the number of communication errors (e.g., ambiguous words, errors in reference) produced for differently valenced passages, under stressful or nonstressful conditions. Impairment on measures of affective reactivity have been noted in several studies of schizophrenia (Cohen & Docherty, 2004; Docherty, 1996; Docherty et al., 1994, 1998), suggesting that the ability to produce effective verbal communication is impaired when patients are asked to read emotionally valenced material aloud. Deficits may be most marked for emotionally negative speech, as communication errors are greatest during these conditions (Cohen & Docherty, 2004).

Vocal intonation has also been found to be abnormal in patients with schizophrenia. Rosen et al. (1989) investigated the speech of patients and controls while reading a neutrally valenced passage, and later measured the acoustical properties of that speech (e.g., rate, fundamental frequency) to determine whether speech patterns differentiated groups based upon symptomatology. It was demonstrated that patients evidencing flat affect
maintain a distinct pattern of speech not found in healthy individuals. While reading emotionally neutral material, vocal correlates of flat affect included a lower percentage of total talking time, shortened utterances, and lengthened pauses. Additionally, these acoustic correlates were predictive of flat affect in schizophrenia, but not other clinical groups. These findings implicate the role of brain structures known to be associated with negative symptoms in the vocal production of flat affect.

Due to the consistency of observed deficits in affect expression, clinicians and researchers alike have posed the question: Does diminished emotional expression signify that individuals with schizophrenia have diminished emotional experience? Several studies have attempted to address this question. Results of multiple investigations suggest a disjunctive relationship between affective expression and experience in which persons with schizophrenia show equal, or perhaps greater, emotional experience than healthy controls, despite producing significantly less affective expressions (Aghevli, Blanchard, & Horan, 2003; Berenbaum & Oltmanns, 1992; Kring et al., 1993; Kring & Neale, 1996; Earnst & Kring, 1999).

When emotional experience is evaluated independently in relation to valence, results suggest that persons with schizophrenia experience emotion equally to controls (Aghevli, Blanchard, & Horan, 2003; Berenbaum & Oltmanns, 1992; Earnst & Kring, 1999; Kring et al., 1993; Kring & Neale, 1996; Mykin-Germeys, Delespaul, deVries, 2000). In fact, several studies suggest that patients experience negative affect more intensely than controls (Aghveli et al.,
Patients also appear to experience positive emotions equally to controls (Aghevli, Blanchard, & Horan, 2003; Berenbaum & Oltmanns, 1992; Kring et al., 1993; Kring & Neale, 1996); however, several studies suggest that this finding may significantly differ among patients experiencing negative psychotic symptoms, as these individuals typically experience significantly less positive emotion than controls and patients who are not exhibiting severe negative symptomatology (Earnst & Kring, 1999; Horan & Blanchard, 2003; Mykin-Germeys, et al., 2000).

Suslow, Roestel, Ohrmann, and Arolt (2003) compared the self-reported experience of multiple discrete emotions among patients experiencing negative symptoms (flat-affect, anhedonia), patients not experiencing negative symptoms, and healthy controls. Results suggest that patients experiencing negative symptoms report experiencing significantly less positive emotions (i.e., joy, interest, surprise) than controls and patients without negative symptoms. Additionally, healthy controls experienced significantly less negative emotions (e.g., fear, sadness, contempt, disgust) than patients with schizophrenia, regardless of psychotic symptomatology. These findings suggest that schizophrenia characterized by negative symptoms may uniquely result in an inability to experience positive emotions.

However, studies classifying patients into deficit and non-deficit forms of schizophrenia are mixed regarding whether patients with primary negative symptoms display a selective impairment in experiencing positive emotion, or even a diminished capacity to experience emotion at all. In comparison to non-
deficit patients, deficit syndrome patients have been found to report lower levels of trait positive affect (Horan & Blanchard, 2003) and higher levels of physical anhedonia (Kirkpatrick & Buchanan, 1990; Herbener, Harrow, & Hill, 2005; Loas et al., 1996). These findings are consistent with a diminished capacity to experience positive emotion. However, impairments have also been noted in relation to negative emotions, as deficit patients have received less severe ratings on clinical scales measuring negative emotions, such as anxiety, guilt, and hostility (Kirkpatrick et al., 1993; Subotnik et al., 2000; Tek et al., 2001). Subotnik (2000) also found evidence for a diminished capacity to experience negative emotions, as indicated by decreased scores for MMPI variables related to emotional distress, such as anxiety, suspiciousness, and social distress. Additionally, despite significant cognitive, social and occupational impairments, deficit patients do not appear to be at higher risk for developing depression (Fenton & McGlashan, 1994; Kirkpatrick et al., 1996; Loas et al., 1996). In fact, deficit patients are not only at decreased risk for major depression, but also six times less likely to have suicidal ideation or behavior than non-deficit patients (Fenton & McGlashan, 1994). Experimental paradigms also suggest that deficit syndrome patients experience a reduced capacity to experience negative emotions, as they have been found to experience lower levels of stress during mood induction procedures paradigms (Cohen & Docherty, 2004; Cohen et al., 2003). Together, these findings appear generally consistent with the notion that deficit syndrome patients report a diminished experience of both positive and negative emotion. However, contrary findings have been reported, as Earnst and
Kring (1999) found that deficit patients did not report experiencing less positive or negative emotion for film clips. Horan and Blanchard (2003) also failed to find differences in trait negative affect using a self-report questionnaire, despite finding diminished trait positive affect. Discrepant findings may be due to differences in affective measurement. In Earnst and Kring’s (1999) study, which failed to find diminished experience, affective experience was measured in relation to momentary state experience. Studies finding diminished emotional experience in deficit patients, as well as Kirkpatrick et al.’s (1989) clinical criterion of reduced emotional range for a period of 1-year, point to enduring trait deficits in emotional experience. Thus, it is possible that the diminished capacity for emotional experience found in deficit syndrome patients may be specific to trait emotional experience.

Aberrant neurotransmitter systems may also contribute to the diminished emotional range reported to occur in the deficit syndrome. In healthy individuals, higher dopamine (DA) levels have been associated with greater experience of positive affect and reward (see Ashby, Isen, & Turken, 1999 for a review.). Although schizophrenia is typically characterized by higher levels of DA activity, recent evidence suggests that the deficit syndrome may be uniquely associated with lower brain DA levels. Lower levels of the DA metabolite, homovanillic acid (HVA), have been reported by multiple investigations (Ribeyre et al., 1994; Thibaut et al., 1998); however, one investigation, Nibuya et al. (1995), reported contradictory evidence, indicating that deficit patients evidenced increased HVA levels. It is likely that results indicating lower HVA levels (Ribeyre et al., 1994;
Thibaut et al., 1998) provide the most accurate assessment of functioning, as reports of higher HVA concentrations (Nibuya et al., 1995) may be biased by inappropriate diagnosis of the deficit syndrome (Kirkpatrick et al., 2001). Reduced DA activity may provide another compelling explanation for the diminished inner-experience of positive emotion noted in deficit syndrome patients. This explanation seems plausible considering the relationship between higher DA levels and positive affect in healthy individuals (Benninger 1991; Phillips, Blaha, Pfaus, & Blackburn, 1992); however, this association has yet to be directly tested among deficit syndrome patients.

In sum, findings are generally consistent with Kirpatrick et al.’s (1989) original conceptualization of the deficit syndrome, which considers diminished emotional range to be a core symptom of the condition; however, additional studies are needed to determine whether deficit syndrome patients display a generalized diminished capacity to experience emotion, or a positive emotion specific impairment that is similar to patients with general negative symptoms of schizophrenia. It will also be important to determine whether differences exist between state and trait experience of positive and negative emotions, considering that findings to date are consistent with trait, but not state impairments. The current study attempts to fill some of these needs by examining both state and trait emotional experience in relation to a range of positive and negative emotions.
Facial Affect Processing

Researchers have also investigated whether affective disturbances extend to emotional perception in patients with schizophrenia. The majority of these studies have examined the perception of facial affect. In healthy individuals, a set of discrete emotional faces have been found to be highly recognizable across genders and cultures (Ekman & Friesen, 1975). These universally identified emotions typically include: happiness, surprise, sadness, anger, fear, disgust, and possibly contempt (Ekman & Friesen, 1975). The ability to discriminate among these emotions is present in infancy (Ludemann, 1991; Soken & Pick, 1999), and typically an automatic task for healthy adults. However, facial affect identification may differ between genders, and among individual emotions. Recent research suggests that women are more accurate in perceiving facial affect than men, and that both genders are more accurate at identifying happiness and surprise faces (i.e., positively valenced faces) than neutral or discrete negative emotions (e.g., sadness, anger, disgust) (Hall & Matsumoto, 2004). Accurate facial affect perception is thought to be critical to the development of normal social functioning (Leppänen & Hietanen, 2001), and may be a core feature of social skills deficits found in several psychiatric disorders (Brozgold et al., 1998).

Over 30 published studies have indicated that persons with schizophrenia evidence impairment in facial affect recognition, discrimination, or matching (for reviews see Kohler & Brennan, 2004; Edwards, Jackson, & Pattison, 2002). Results are inconclusive regarding whether perceptual deficits differ in relation to
valence, as some studies indicate significant deficits in processing happy faces (Archer et al., 1994; Bellack et al., 1996; Loughland, Williams, & Gordon, 2002a; Loughland, Williams, & Gordon, 2002b; Phillips et al., 1998; Sachs et al., 2004; Schneider et al., 1995), while several others report intact positive emotion identification and significant impairment for negative faces, especially fear (Gaebel & Wolwer, 1992; Johnson, Emde, Scherer, & Kilnnert, 1986; Mandal, 1987, 1998; van der Gaag & Haensen, 1990; Wolwer et al., 1996; ) and sadness (Kohler et al., 2000; Loughland, Williams, & Gordon, 2002, 2003; Edwards, Pattison, Jackson, & Wales, 2001). Variability in findings may be due to several factors, including methodological variables, medication status, and symptom profiles (i.e., presence or absence of negative symptoms) (Edwards, Jackson, & Pattison, 2002).

Although findings are variable regarding which facial expressions are processed abnormally, studies consistently indicate that deficient cognitive abilities contribute to affect perception deficits. Several studies have found that facial affect impairments are correlated with the cognitive abilities of abstraction-flexibility, selective attention, sustained attention, verbal memory, visual organization, and language ability (Bryson, Bell, & Lysaker, 1997; Horan & Blanchard, 2003; Kohler et al., 2000; Sachs et al., 2004; Schneider, Gur, Gur, & Shtasel, 1995; Silver & Shlomo, 2001). Research by Allen, Strauss, Gilbertson, & van Kammen (unpublished manuscript) suggests that these relationships may differ in relation to the processing of positive and negative faces. Specifically, negative affect labeling is predicted by attention switching/executive functioning,
as well as disorganization, thought disorder, and anergia. Positive affect labeling was not found to significantly correlate with neurocognitive or symptomatological variables. These results suggest that cognitive deficits may differentially determine the nature of affect perception impairments in individuals with schizophrenia.

Recent eye-tracking research also indicates that positive and negative affect identification deficits are maintained by separate neurocognitive mechanisms. Results from several studies suggest that patients evidence differences in visual-scanning for happy and sad faces (Loughland, Williams, & Gordon, 2002a; Loughland, Williams, & Gordon, 2002b). Specifically, individuals with schizophrenia display a restricted scanning pattern for happy faces, where they fixate upon isolated and irrelevant facial regions (e.g., nose, ears, hair), and fail to attend to informative aspects of the face (i.e., eyes, mouth, brow). Negative affect processing facilitates a much wider visual scanning behavior, as patients typically attend to multiple facial regions, including those that are most informative. Additionally, in each of the studies conducted by Williams et al. (2002ab), patients only differed from controls in their ability to identify positive emotions. This may suggest that difficulties in identifying positive faces are uniquely associated with a failure to attend to relevant facial features (Williams et al., 2002a). As studies investigating visual scanning did not find impairments relative to negative emotions, it is possible that alternate cognitive systems underlie those deficits.
Positive and negative symptoms of schizophrenia may also influence the perception of facial affect. Patients with paranoid schizophrenia have been found to exhibit better overall facial affect recognition than non-paranoid patients and seem particularly good at identifying negative emotions, including anger, fear, disgust, and sadness (Kline, Smith, & Ellis, 1992). A small body of evidence also suggests that deficit syndrome patients are particularly deficient at perceiving emotional faces. Bryson et al. (1998) investigated facial affect identification in a sample of deficit and non-deficit patients using video-taped vignettes designed to represent 7 basic emotions. Results suggest that deficit patients were significantly poorer than non-deficit patients at recognizing the emotions of sadness, disgust, surprise, and neutral. Although impairment was noted for both positively and negatively valenced faces, deficits noted for recognition of surprise are of particular interest, considering that this emotion is typically identified with high accuracy among controls and non-deficit patients alike. Contrary findings were reported by Horan and Blanchard (2003), who failed to find differences in facial affect identification between deficit and non-deficit patient groups; however, these findings should be viewed with caution, since emotions in this study were collapsed into a general “emotional” category. Such procedures may preclude the observation of noticeable differences by compounding variance across multiple emotional conditions. These studies suggest that schizophrenia characterized by negative symptoms is more strongly associated with facial affect recognition deficits compared to schizophrenia characterized by paranoia and possibly positive symptoms.
Collectively, these findings suggest that facial affect identification deficits may be mediated by several variables, particularly neurocognition and psychotic symptoms. Although valence-specific deficits have been inconsistently found in schizophrenia, the identification of positive and negative emotions appears to be maintained by separate neural mechanisms. Further research is needed to determine the nature of these mechanisms in relation to discrete facial emotion identification, and to examine whether individual emotions are differentially influenced by symptom presentation.

**Olfactory Affect Perception**

Adequate olfactory processes are thought to have significant survival value, as they are one of our primary windows to the environment (Ache, 1991). That is, smell identification allows us to both avoid things that would be harmful to the body and consume things that would be beneficial (Scott & Giza, 2000). Although the process of assigning an affective label of “pleasant” or “unpleasant” to a smell seems relatively straightforward, it likely involves a complex emotional appraisal that calls upon emotional memory networks. Several brain regions are thought to underlie complex processes involved in smell perception, including the orbito prefrontal cortex, medial-temporal gyrus, and thalamus. Interestingly, these regions are also central to normal emotion processing, particularly for positive emotions (Thorpe et al., 1983). Given that the aforementioned regions have been consistently implicated in the neuropathophysiology of schizophrenia (see Antonova et al., 2004), researchers have recently begun investigating the relationship between olfaction and emotion (Moberg et al., 2003).
Several studies have found smell identification impairment in persons with schizophrenia (Brewer et al., 1996; Good et al., 1994; Hurwitz et al., 1988; Kohler et al., 2001; Kopala et al., 1989, 1992, 1994; Malaspina et al., 1994, 2003; Moberg et al., 2003; Seidman et al., 1992; Warner et al., 1990). Deficits extend to all aspects of olfactory perception, including quality discrimination, detection threshold (i.e., sensitivity), and odor recognition (see Rupp, 2003 for review). Impairments do not appear to be due to the influence of gender (Koapla et al., 1994), smoking behavior (Moberg et al., 2003), and medication (Pinkhas et al., 1999), nor do they reflect the presence of a general sensory impairment (Kopala et al., 1995). However, researchers have recently found relationships between smell identification deficits (SID) and neurocognitive functioning. SID in schizophrenia have been related to verbal abilities (Malapina & Coleman, 2003), executive functioning (Saoud et al., 1998; Stedman & Clair, 1998), and attention/processing speed (Goudsmit et al., 2004). Separate relationships have been noted in deficit syndrome patients (i.e., patients with severe negative symptoms), who tend to evidence more significant impairments than non-deficit patients in smell identification (Goudsmit et al., 2004; Malaspina et al., 2001; Secklinger et al., 2002). Relationships noted in deficit patients involve visuospatial processes, such as visual-motor processing speed (Goudsmit et al., 2004), eye movements (Malaspina et al., 2001), and visuomotor organization (Secklinger et al., 2002). This pattern of correlations is consistent with both a frontal and parietal dysfunction within deficit syndrome patients (Secklinger et al., 2002).
Considering overlap between neural circuits involved with emotion processing and olfaction, researchers have recently attempted to determine whether individuals with schizophrenia are impaired at recognizing olfactory valence. In a comprehensive assessment of olfactory perception, Hurdy et al. (2002) found that patients were more deficient than controls at several olfactory processes, including the assignment of pleasantness ratings. Similarly, Moberg et al. (2003), found patients to be impaired at rating pleasant stimuli, despite being nearly identical to controls with regard to intensity judgment. These findings were supported by Crespo-Facorro et al. (2001) who furthered results of behavioral studies by identifying neural systems involved with pleasantness judgments using positron emission tomography. In this study, patients and healthy comparison subjects were not found to differ in intensity ratings; however, they did evidence differences in judging the valence of pleasant smells. These deficits were associated with aberrant prefrontal cortex and limbic system activation. Doop and Park (2006) also found schizophrenia patients to display abnormal pleasantness judgments, and reported that these abnormalities are correlated with negative symptoms, such that more severe flat affect was associated with more aberrant hedonic judgment.

Although relatively few studies have investigated the nature of olfactory affect perception in schizophrenia, results from initial studies suggest that patients may be particularly deficient at processing positively valenced smells and that these impairments may be more severe in patients with negative symptoms. Further research is needed to determine whether positive affect impairments are more
pronounced in deficit syndrome patients, who tend to have more severe olfactory impairment in general (Goudsmit et al., 2004; Malaspina et al., 2001; Secklinger et al., 2002). The current study attempts to address this need by examining both accuracy and valence judgments for pleasant and unpleasant odors in deficit and non-deficit schizophrenia.

Memory for Affective Information

Memory processes are critical for normal emotional functioning. A large body of research has investigated recall and recognition memory for affectively valenced information in healthy individuals. Results from numerous studies have shown that healthy individuals evidence increased memory performance for positive information in free recall and recognition measures of long-term memory (Amster, 1964; Barrett, 1938; Bradley, Greenwald, Petry, & Lang, 1992; Colombel, 2000; Hayward & Strongman, 1987; Libukman, Stabler, & Otani, 2004; Lishman, 1972a; Matlin, Stang, Gawron, Freedman, & Derby, 1979; Phelps, LaBar, & Spencer, 1997; Rychlak & Saluri, 1973). These findings reflect a memory bias for positive information. Matlin and Stang (1978) termed this memory bias the Pollyanna Principle, to reflect a general tendency to select pleasant information over unpleasant information, and described the phenomenon as resulting from increased accuracy and efficiency relative to the encoding and retrieval of positive information. With regard to memory, the tendency to remember positive information not only occurs in studies examining experimenter generated lists, but also encompassed the recall of daily experiences and participant generated-lists (Matlin & Stang, 1978). This
suggests that the memory bias inherent to positive information is a pervasive phenomenon that extends to virtually all facets of emotional memory.

A significant body of research suggests that memory performance is impaired in persons with schizophrenia (Paulsen et al., 1995; Randolph et al., 1994; Saykin et al., 1991). However, relatively few studies have investigated memory for emotional information in schizophrenia. In a study conducted by Koh, Grinker, Marusarz, & Forman (1981) patients and healthy controls were required to first sort a list of words repeatedly, and then to unexpectedly recall those words. Results suggest that healthy controls recalled a greater number of positive than negative words (i.e., Pollyanna Principle); however, this finding was not evident in patients with schizophrenia. Patients showed an overall reduction in memory for emotional words in comparison to controls, and relatively equal recall for positive and negative words. In the second experiment of Koh et al. (1981), patients and controls were asked to make judgments of whether emotional faces were pleasant or unpleasant, and later required to perform a test of recognition memory. Results were similar to the verbal memory task, as controls recognized a significantly higher number of positive than negative faces, and patients recognized significantly fewer positive stimuli than controls. Additionally, patients recognized a significantly greater number of negative than positive faces, potentially signifying that patients have a bias toward recognizing negative information.

Calev (1996) found similar results for verbal information, as patients recalled significantly less emotional words in general, and recalled a greater
number of negative than positive words. Calev and Edelist (1993) also found that patients recall significantly fewer emotional words than controls, and extended previous research by finding that negative emotional words were forgotten less rapidly after a 2 day delay period. Thus, results are consistent with the notion that emotional memory is impaired in schizophrenia; however, contrary findings have also been reported, where some studies have failed to find differences in emotional memory between patients and controls. A recent study conducted by Matthews and Barch (2004) failed to find differences between controls and persons with schizophrenia in verbal recall and recognition tasks. In this study, patients and controls were asked to rate words based upon arousal, and subsequently tested on recall and recognition. Results suggest that patients and controls recalled significantly more high-arousal than low arousal words, and neither group displayed the Pollyanna effect typically seen in healthy individuals. However, the discrepant findings of Matthews and Barch may be explained by methodological factors, as positive and negative words included likely vary in intensity (but not valence-arousal).

More recently, studies have examined the association between negative symptoms and emotional memory impairment, focusing specifically on the influence of anhedonia on recall for emotional events and information. In a recent study conducted by Horan, Green, Kring, and Nuechterlein (2006), patients and controls were presented with a series of pleasant and neutral film clips and asked to rate their level of emotional experience. After a four hour delay period, they were given an unannounced recall task where they were asked to rate how
pleasant they felt in relation to previously presented stimuli. Results indicated that although substantially anhedonic, patients did not report experiencing less positive emotion in the moment, nor did they recall experiencing stimuli as being any less pleasant than they did at initial presentation. These findings appear to suggest that positive emotional memory impairment is unassociated with negative symptoms of schizophrenia. However, somewhat contrary findings have been reported by Herbener, Rosen, Khine, and Sweeney (2007). Herbener et al. (2007) presented patients and controls a series of positive, negative, and neutral images, and required participants to indicate how emotionally intense each image made them feel. After a 24-hour delay period, an unannounced recognition session occurred, where participants indicated whether they had seen each image on the previous day. By requiring participants to initially focus on the intensity evoked by each stimulus, the paradigm is thought to assess memory consolidation after initial encoding, and therefore whether pleasant and unpleasant emotional experiences have beneficial effects on memory. With regard to initial emotional experience, results indicated that patients experienced both positive and negative images as being more intense than controls. Recognition findings indicated that patients were poorest at recognizing positive stimuli, whereas controls displayed a bias toward recognizing positive scenes more accurately than negative or neutral stimuli. Correlational findings indicated that trait anhedonia was significantly correlated with recognition of positive images in controls but not schizophrenia patients, suggesting that a failure in the memory consolidation process, such that patients are unable to use the
experience of positive emotion to enhance their memory for emotional information. These findings are consistent with the notion that faculty memory contributes to symptoms of anhedonia. Contrary findings reported by Herbener et al. (2007) and Horan et al. (2006) may be due to several factors. First, Herbener et al employed a 24 hour delay, rather than a 4-hour period. It is possible that the effects of initial positive emotional experience may only fade in memory after significant time delay. Second, Herbener and colleagues used a paradigm that examined recognition for emotional information, rather than recall of their initial feelings themselves. Thus, differences may indicate that anhedonia is associated with reduced memory for positive information, rather than feelings in and of themselves. It is possible that patients may experience little variability in their daily affective lives, and that they subsequently have little difficulty recalling their feelings. However, they appear to have difficulty recalling positive information, which may cause higher symptoms of anhedonia by creating difficulty in recalling specific events experienced over recent weeks.

Considering that anhedonia has been proposed to be a core symptom of deficit syndrome schizophrenia (Kirkpatrick et al., 1996; Loas et al., 2001), it will be important to determine whether faculty memory for positive feelings or information contributes to deficit syndrome symptoms. More specifically, considering that deficit patients have been found to display diminished trait (Horan & Balnchard, 2003) but not state (Earnst & Kring, 1999) positive emotional experience, studies could examine whether these patients evidence impaired encoding and/or retrieval of positive information that leads them to
recall pleasurable experiences as less pleasurable when asked to think back over the last few weeks, despite being able to experience normal levels of positive emotion in the moment.

Collectively, results from studies investigating emotional memory in schizophrenia suggest that patients 1) remember less emotional information than controls, 2) do not exhibit the same Pollyanna tendency found in healthy individuals, 3) have a bias toward recalling negatively valenced material, and 4) have greater impairment for positive information when anhedonia is present. However, further research is needed to determine whether these findings apply when patients are presented with multiple discrete emotional categories and whether negative symptoms of the deficit syndrome are associated with impaired verbal recall.

**Automatic Processing**

Recent emotion research has also focused on automatic cognitive processes. Cognitive processes are described as automatic when information is processed quickly and without effort (Logan, 1978). The rapid detection of emotional information has been proposed to facilitate survival within the environment (Mandler, 1975; Oatley & Johnson-Laird, 1987). Our attentional system may play a central role in this process, as it has been proposed to regulate the process of diverting focus away from goal-directed behavior and toward more adaptive functions that promote survival (i.e., fight or flight) during times of danger. Several studies have found that threatening information significantly disrupts ongoing cognition in healthy individuals (McKenna &
Sharma, 1995, 2004; Myers & McKenna, 1995; Sharma & McKenna, 2001; Watts, McKenna, Sharrock, & Trezise, 1986), signifying that human-beings have an inherent attentional bias for threat.

Interference tasks provide a valid means of assessing disruption caused by emotional material. The Emotional Stroop (ES) task has been widely used to assess emotional interference. Similar to Stroop's (1935) original color-naming task, the ES task requires participants to identify the ink color in which a word is printed, while ignoring the meaning of a written word. However, rather than manipulating color-congruent and incongruent words, the ES task compares RT for emotional and neutral words. The theory behind the ES task states that words more related to an individual's preoccupations, concerns, or mood state will significantly interfere with the target process (i.e., color-naming), and thereby produce longer RTs when compared to neutral words. The ES task has consistently demonstrated that healthy individuals evidence an attention bias for threatening information (McKenna & Sharma, 1995, 2004; Myers & McKenna, 1995; Sharma & McKenna, 2001; Watts, McKenna, Sharrock, & Trezise, 1986); however, several factors may enhance threat detection.

One factor includes the manipulation of time pressure. It has been hypothesized that negative aspects of stimuli are highly influential during situations eliciting time pressure (Wright & Weitz, 1997). McKenna and Sharma (2001) examined the role of time pressure using an ES task by varying the length of interstimulus interval (ISI) time (i.e., length of fixation point that separates stimuli). Participants were presented with blocks of negative and neutral words at
ISI lengths of 32, 80, 160, 240, 400, and 1000 ms, to determine whether RT differed as a function of ISI length. Results suggest that ISI length (i.e., time pressure) impacted RT, such that interference effects for negative words were significantly greater than for neutral words at the shorter ISI length (e.g., 160 ms). Additionally, differences for negative and neutral words were nonsignificant at longer ISIs (> 400 ms). These findings have significant implications for the automatic processing of emotional information, as they suggest that threat stimuli presented under time pressure may rapidly redirect attention, potentially enabling an organism to prioritize focus and respond more effectively in times of danger (Sharma & McKenna, 2001).

Recent research by Strauss and Allen (unpublished manuscript) replicated and extended the findings of Sharma and McKenna (2001). Strauss and Allen investigated the influence of time pressure in relation to multiple discrete positive and negative emotions. Results from this study suggest that time pressure differentially impacts discrete emotions. Specifically, negative emotions are significantly interfering only under periods of time pressure, and positive emotions only become interfering in the absence of time pressure. These findings suggest that our attentional system is predisposed to automatically detect information that is most adaptive based upon situational factors. Under periods of high time pressure (which ostensibly signifies danger), negative emotions are most salient; however, under periods of normal functioning, self-relevant positive information grabs attention. Additionally, not all discrete negative emotions are interfering under periods of time pressure. Sadness words
produced the most interference under the most time pressure, while anger and anxiety words were significantly less interfering than neural words. This may suggest that highly threatening information (i.e., anger and anxiety words) bypasses cortical pathways during processing, and moves quickly to subcortical structures specialized for threat detection (i.e., amygdala), allowing a faster response to the threatening stimuli (LeDoux & Armony, 1999).

Although our inherent bias for detecting threatening information may promote survival during times of danger, this mechanism may also facilitate psychological distress when active in “every-day” situations. Recent research suggests that several psychiatric disorders evidence a heightened sensitivity for or preoccupation with emotional information. This heightened awareness is typically referred to as an “attention bias”, and is proposed to occur in psychiatric populations when individuals encounter environmental information relevant to their unique concerns (Williams, Matthews, & MacLeod, 1996). Attention biases have been proposed to occur in several psychiatric conditions, including: Social Phobia (Hope et al., 1990), Obsessive Compulsive Disorder (OCD) (Foa, Ilai, McCarthy, Shoyer, & Murdock, 1993; Lavy, Oppen, & Van den Hout, 1994), Panic Disorder (McNally, Amir, Louro, Lukach, Riemann, & Calamari, 1994), Mood Disorders (Gotlib & McCann, 1984), and Specific Phobia (Lavy, Van den Hout, & Arntz, 1993).

Relatively few studies have examined attention biases in individuals with schizophrenia. Epstein, Stern, and Silbersweig (1999) presented individuals with schizophrenia with neutral and inter-personally threatening (e.g., whisper, follow,
stare) words. Results suggest that interpersonally threatening words significantly interfered with color-naming, particularly among paranoid patients. However, implications of these findings are limited due to the lack of a control group comparison. Since healthy individuals are also known to evidence an attention bias for threatening (i.e., general negative) words, interference effects found by Epstein et al. (1999) may reflect an attention bias inherent to all individuals; however, diagnosis related differences may suggest that paranoid patients are uniquely predisposed to detecting threat. Findings of Fear, Sharp, & Healy (1996) may further delineate the nature of threat detection and symptom profiles. Fear et al. (1996) presented individuals with delusional disorder words representing sadness, anxiety, and threat in an ES task. Results suggest that individuals with delusional disorder evidenced significantly greater interference for threatening words in comparison to healthy controls. Additionally, individuals meeting the criteria for the non-persecutory delusion subtype showed significantly greater interference than controls for sadness and anxiety words. The fact that these results were not found in the persecutory delusion group suggests that psychosis without paranoid features may also be related to heightened sensitivity for sadness and anxiety words.

Affective priming tasks provide another means of assessing automatic processing. Priming tasks typically require participants to respond to a target word that is preceded by a very brief presentation (e.g., 50 ms) of a prime word, which is either emotionally congruent or incongruent with the target word. RT for the identification of target stimuli serves as the dependent variable. One study
investigated affective priming for verbal stimuli in schizophrenia (Suslow, Roestel, Droste, & Arolt, 2003) and found that patients and controls did not significantly differ regarding the automatic processing of emotional words. However, significant relationships were found between mood state and priming, such that priming based upon positive words was positively correlated with positive mood, and negatively correlated with negative mood. Based on this finding Suslow et al. (2003) suggest that deficits in the automatic processing of positive information may contribute to the development of negative emotions in schizophrenia. In a similar priming paradigm, Suslow et al. (2005) also found that negative symptoms were associated with automatic processing impairments for positive, but not negatively valenced faces. Thus, findings of Suslow et al. (2003, 2005) suggest that patients with schizophrenia display significant automatic processing impairments, which are associated with both emotional experience and negative symptoms.

Research has yet to investigate whether individuals with schizophrenia process discrete emotional information abnormally. Considering the high rate of depression in schizophrenia (Hegalson, 1990), and significant abnormalities in positive emotion processing (Suslow et al., 2003, 2005), it seems relevant to investigate attention bias in relation to the processing of multiple discrete emotions. Additionally, since negative symptoms of schizophrenia have been associated with automatic processing abnormalities in relation to positive emotions using priming paradigms, additional studies are needed to determine whether patients with core, primary negatives symptoms, such as those with the
deficit syndrome schizophrenia, evidence a failure to have attention automatically oriented to emotional information using interference tasks, such as the Emotional Stroop.

Summary

Normal emotion processing is critical for the development of social skills and appropriate social interaction. Clinicians have historically noted that persons with schizophrenia evidence an inability to express affect through facial and vocal channels. Research suggests that this inability to express emotion may be most severe for the expression of positive affect. However, deficits in expressing emotion may not reflect a diminished ability to experience emotion. In fact, patients have been shown to experience an equal amount of positive emotion, and a significantly greater amount of negative emotion than normal comparison samples. Emotional disturbance has also been noted in relation to the perception of emotion. Patients are particularly deficient at identifying emotional faces, detecting emotional intonation in speech, remembering emotional words, ignoring negative emotional information, and integrating audio-visual affective productions. Emotional impairments may be most severe for the processing of positive emotion. However, deficits have also been noted for negative emotions, particularly sadness and fear. A critical analysis of the emotion processing literature also suggests that patients with deficit syndrome schizophrenia may be uniquely characterized by severe positive emotion impairments. Unlike non-deficit patients, deficit syndrome patients are typically unable to experience adequate levels of emotion in general, and may display a unique impairment in
experiencing positive emotion. Deficit patients have also been found to demonstrate unique impairments in the perception of positive emotion. Several additional abnormalities related to the cognitive processing (e.g., attention, memory) or perception (e.g., olfaction, face identification), have been associated with more severe negative symptoms; however, it has yet to be determined whether these impairments also extend to deficit syndrome patients. Positive emotion abnormalities may be mediated by structural and functional brain abnormalities that affect the dorsolateral prefrontal-basal ganglia-thalamocortical circuit, as well as hypoactive DA activity. Further research is needed to determine whether the deficit syndrome is uniquely characterized by pervasive positive emotion processing deficits, and to identify the neurological substrates that maintain these impairments.

Hypotheses

The current investigation attempts to answer 3 major questions regarding affective disturbance in deficit and non-deficit syndrome schizophrenia: 1) Do deficit patients, non-deficit patients, and controls differ with regard to emotional experience? 2) Do deficit patients, non-deficit patients, and controls differ with regard to the perception of emotional information? 3) Do deficit patients, non-deficit patients, and controls differ with regard to the cognitive processing of emotional information?

Based upon previous research indicating that deficit syndrome patients experience significantly less positive affect than non-deficit patients and healthy
controls (Horan & Blanchard, 2003; Suslow et al., 2003), it is hypothesized that
deficit and non-deficit syndrome patients will differ with regard to self-reported
emotional experience. Specifically: 1) individuals meeting criteria for deficit
syndrome will report experiencing significantly less positive emotion than non-
deficit patients and healthy controls, and 2) deficit syndrome patients will indicate
experiencing negative emotion equally to non-deficit patients, and significantly
more than controls.

Based upon previous research indicating that deficit syndrome patients
are impaired at perceiving facial expressions of surprise (Bryson et al., 1998), it
is hypothesized that deficit syndrome patients will be more impaired than non-
deficit patients and healthy controls at identifying positive emotional expressions.
Additionally, healthy controls will conform to the Pollyanna Principle and identify
positive faces more accurately and efficiently (quickly) than discrete negative
emotions, and deficit and non-deficit syndrome patients will evidence positive
emotion processing impairments. However, impairments will be significantly more
severe among individuals meeting criteria for the deficit syndrome (i.e., slower
RTs and poorer accuracy).

Consistent with previous research indicating that deficit syndrome patients
evidence more severe olfactory impairment than non-deficit patients (Goudsmit
et al., 2004; Malaspina et al., 2001; Secklinger et al., 2002), it is hypothesized
that deficit syndrome patients will have poorer smell identification than non-deficit
patients and healthy controls. Additionally, considering that patient deficits are
known to be more severe for pleasant smells (Crespo-Facorro et al., 2001; Hurdy
et al., 2002; Mober et al., 2003), it is hypothesized that deficit and non-deficit syndrome patients will be poorer than controls at recognizing pleasant smells. Furthermore, the recognition of pleasant smells is expected to be more severe for deficit than non-deficit patients.

Based upon previous research indicating that individuals with schizophrenia evidence impaired memory for positive emotional words (Calev, 1996; Calev & Edelist, 1993; Koh et al., 1981), it is hypothesized that deficit and non-deficit patients will recall significantly less happiness words than healthy controls. Impairments in positive emotional memory are expected to be more pronounced for deficit than non-deficit groups, while healthy controls are expected to display a Pollyanna tendency consistent with previous research (Matlin & Stang, 1978).

Based upon previous research indicating that persons with schizophrenia are impaired at automatically processing positive information (Suslow et al., 2003), it is hypothesized that deficit syndrome patients will display less of an attention bias for happiness than using the Attention Grabbing Task, non-deficit patients and controls (i.e., deficit patients will have significantly shorter RTs for happiness).

Additionally, consistent with evidence suggesting that individuals with schizophrenia have a greater attention bias for threatening material than controls (Epstein et al., 1999; Fear et al., 1996), it is hypothesized that both deficit and non-deficit patients will display a greater lingering effect for negative words than controls (i.e., deficit and non-deficit patients will have longer difference scores for
negative words). Deficit patients are not expected to differ from non-deficit patients regarding lingering effects for negative words, as previously research indicates that both patient groups experience similar levels of negative emotion, which are considerably higher than controls (Earnst & Kring, 1999; Horan & Blanchard, 2003). Additionally, no differences are expected among groups with regard to lingering effects for positive or category conditions, as research on healthy individuals has found these conditions incapable of rendering the lingering effect on the attentional system (McKenna & Sharma, 2005).
CHAPTER 3

METHODOLOGY

Participants

Data was collected from two participant groups: 1) 41 patients meeting DSM-IV-TR criteria for schizophrenia, and 2) 22 psychologically and neurologically healthy controls. Participants were selected for inclusion in the patient group if they met criteria for current Axis-I diagnosis of schizophrenia. The healthy control group consisted of individuals with no lifetime diagnosis of schizophrenia, current Axis-I psychiatric disorder, or history of neurological conditions. Exclusionary criteria for individuals with schizophrenia included: 1) English as a second language; 2) history of traumatic brain injury or any other medical condition or neurological disease/damage that could cause cognitive deficits (e.g., seizure disorder, Parkinson’s disease); 3) history of alcohol or drug abuse or dependence diagnosis within the past six months; 4) current DSM-IV Axis I condition other than schizophrenia; 5) diagnosis of mental retardation or other cognitive dysfunction; 6) use of prescribed or over-the-counter medications that could produce significant cognitive effects, other than those medications used to treat schizophrenia. In addition to the aforementioned criteria, exclusionary criteria for the control group also included: 1) current use of psychotropic medication, 2) corrected vision greater than 20/50 (determined
using a standard wall-chart), 3) biological familial relation to an individual with diagnosed or suspected psychosis (as determined through standardized interview with the control subject).

Deficit syndrome classification was then applied to patients using the Schedule for the Deficit Syndrome (SDS) rating scale. This classification resulted in identification of 15 deficit and 26 non-deficit syndrome patients. Patient and control groups were matched on age and education, as these demographic variables are known to effect cognitive functioning. Basic demographic data are presented for deficit, non-deficit, and control participants in Table 1.

Considering the importance of establishing that deficit syndrome patients included in the current study were similar to those identified in the research literature, deficit and non-deficit patients were evaluated on clinical and demographic variables that are recommended for comparison by the SDS authors. In line with these recommendations, deficit and non-deficit patients did not differ on length of illness, age of onset, medication dosage, extrapyramidal symptoms (see Table 4), or severity of positive symptoms (see Tables 5 and 7). The deficit group also had a higher percentage of male than female participants (see Table 1), received lower ratings on BPRS scales for suspicion and emotionality (depressed mood, anxiety, guilt, hostility) (see Table 5), had lower premorbid IQ estimates (see Table 1), and less frequent prevalence of current or previous marriage (see Table 2). Additionally, none of the deficit patients manifested marked disorganization (see Table 5), depressive symptoms (see Tables 5 and 6), or severe sedation due to the effects of medication. These
clinical and demographic characteristics are very consistent with specifications made by the SDS authors (Kirkpatrick et al., 2001; SDS Manual), and provide support for the validity of these patients classified as “deficit syndrome” in the current study.

Several additional classification procedures were used to account for concerns that the SDS authors have raised regarding establishing intergroup reliability within deficit syndrome diagnosis. These procedures were based upon recommendations published within the SDS administration manual. First, following initial SDS interviews performed by one of 3 trained graduate-level researchers, consensus SDS interviews were conducted by the first (G.S.) and senior authors (D.A.) to ensure accuracy of deficit classification and symptom severity ratings. Consensus ratings were performed on all patients initially identified as having the deficit syndrome, or who exhibited significant negative symptoms. One-hundred percent deficit/non-deficit classification agreement was obtained on all consensus interviews, and patients not identified as deficit syndrome were consequently eliminated from the group following consensus ratings. Second, treatment providers (psychiatrists, counselors, case managers) were consulted and psychiatric charts were reviewed to verify stability of symptom presentation and accuracy of primary vs. secondary negative symptom distinction. Finally, one of the SDS authors (B. Kirkpatrick) was consulted on cases where deficit classification was questionable, and relevant clinical, demographic, and historical information were reviewed to assist in making deficit diagnosis judgments.
Procedure

Individuals with schizophrenia were recruited from a local community outpatient mental health center (Mojave Adult, Child, and Family Services). A clear diagnosis of DSM-IV-TR (American Psychiatric Association, 1994) schizophrenia was identified by a staff psychiatrist for all patients, and confirmed using the Structured Clinical Interview for DSM-IV-TR. To further ensure accuracy of diagnosis and suitability for selection criteria, a thorough review was conducted on patient medical records and psychiatrist progress notes. All patients provided release of medical records for this review, and for medication use and dosage at the time of testing to be extracted from charts at the time of testing. Consistent with the schizophrenia literature, medication data was converted to Chlorpromazine equivalent doses using procedures delineated by Woods (2003), allowing patient groups to be compared on neuroleptic dose (see Table 1).

Healthy controls were recruited from the local community using posted flyers and within the UNLV Psychology Department Subject Pool. Participants recruited from the Subject Pool received compensation of extra credit points and/or partial fulfillment of their course requirements, equivalent to one credit hour for each hour of participation. Patient and control participants recruited from the community received monetary compensation for participation in the amount of $10.00 per hour, with an average total of $60.00 for completion of the entire study. Participants who failed to complete all study procedures, and those who
were excluded based upon selection criteria, were compensated on a pro-rated basis (approximately $2.50 for each half-hour of participation).

Total completion time for the current study was approximately 6-8 hours for patient groups. All individuals interested in completing the current study first underwent an initial screening. Individuals from the patient group that met initial selection criteria were scheduled for an appointment and completed study procedures in an average of two to three testing sessions lasting approximately 1-4 hours in length. For individuals with schizophrenia, session one lasted approximately 3 hours, and included the administration of demographic and medical history questionnaires, SCID-IV interview, clinical symptom interview, and self-report emotion measures. Session two consisted of computerized emotional and neuropsychological testing. Patients were allotted breaks as needed. For control subjects, testing was generally completed in one sitting that lasted approximately 4 hours. A 30 minute break was implemented at the midpoint of testing and as needed for controls.

Detailed descriptions of all tests and testing procedures are provided in the measures section. Measures included in the first testing session were administered in the following order: Demographic and medical history questionnaire, SCID-IV, Clinical Symptom Interview (used to obtain information for SDS, SAPS, SANS, BPRS), Extrapyramidal Symptom Measurement (used to obtain information for ratings on the EPS, AIMS, Rockland Rating Scale, and Barnes Akathisia scales). Participants also typically completed several self-report measures of emotional experience and well-being during the first session,
including: Differential Emotions Scale, Positive and Negative Affect Scale, Psychological Well-being Scales, Social Well-Being Scale, and the Satisfaction with Life Scale. During the second portion of testing participants received a battery of emotion processing tasks, the Brief Smell Identification Test, WAIS-III Subtests (Vocabulary, Information, and Block Design), and neuropsychological measures. The order of task administration within this portion of testing was designed to control for carry-over effects and account for necessary periods of verbal and nonverbal testing. Neuropsychological tests were administered as part of a larger study and not described here. These tests included: California Verbal Learning Test, Wechsler Memory Scale, Third Edition Digit Span and Spatial Span subtests (forward and backward), Trail Making Test (A & B), Finger-tapping test, Purdue Pegboard, Controlled Oral-Word Association Test, Visual Backward Masking Test, Biber Figure Drawing Test, and Wisconsin Card Sorting Test.

All testing was conducted by the primary author and two trained doctoral-level graduate students, and occurred in a quiet setting (office within laboratory or community mental health center). Time was allotted for questions after the examination, and participants were given a debriefing form containing experimenter contact information and information regarding the nature of the study.

Measures

Measures used in the current study assessed 4 facets of psychological functioning: 1) clinical symptomatology, 2) emotional experience, 3) emotional...
information processing, and 4) intellectual functioning. Description of the format of each test and its procedures is provided below. Psychometric properties of all tests are also provided where relevant.

Clinical Symptomatology

Several measures were included to assess clinical symptomatology relevant to schizophrenia. Measures included the Structured Clinical Interview for DSM-IV-TR (SCID), Schedule for the Deficit Syndrome (SDS), Scale for the Assessment of Negative Symptoms (SANS), Scale for the Assessment of Positive Symptoms (SAPS), Brief Psychiatric Rating Scale (BPRS), and the Calgary Depression Scale for Schizophrenia. The SCID was used to verify DSM-IV Axis-I diagnosis of schizophrenia. The SDS was used to determine deficit syndrome classification, while the SANS, SAPS, BPRS, and Calgary Depression Scale were included to assess positive and negative psychotic symptoms and depression specific to individuals with schizophrenia.

Several measures were also included to assess patient extrapyramidal symptoms, including the Rockland Rating Scale (RRS), Abnormal Involuntary Movement Scale (AIMS), Extrapyramidal Symptoms Scale (EPS), and the Barnes Akathisia scale. These measures are included to assess a broad sampling of the physical domains affected by antipsychotic medications (e.g., face, neck, extremities).

Structured Clinical Interview for the DSM-IV-TR.

The Structured Clinical Interview for DSM-IV-TR (SCID) is a structured clinical interview designed to determine psychiatric diagnosis and identify clinical
symptoms. The interview process utilizes open-ended questions and "skip-structure" procedures that allow the administrator to investigate content areas based upon examinee response. Questions are designed to assess Axis I (SCID-I) and Axis II disorders (SCID-II), with separate forms for the assessment of inpatient (SCID-P), outpatient (SCID-OP), and nonpatient groups (SCID-NP). In order to account for the SCID's lengthy administration time, examiners commonly administer modules of primary concern. The SCID-P will be administered in the current study.

When assessing schizophrenia, it is important to rule out the presence of several other conditions that present symptoms similar to those in psychosis. The SCID-I will be used to allow for differential diagnosis of several Axis-I disorders, including schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic disorder, substance abuse and dependence, and bipolar disorder. Inter-rater reliability of the SCID-I has been shown to be excellent (kappa = .85, range = .71 to .97), and diagnostic accuracy, as compared to consensus diagnosis, was very accurate (82%) (Ventura et al., 1998). A study conducted by Fennig et al., 1994), suggests that the SCID-I is a valid instrument for the diagnosis of schizophrenia, as SCID schizophrenia diagnosis displayed good sensitivity (.89), specificity (.96), and agreement (.86) when compared to best estimate diagnosis made by psychiatrists on first-admission psychotic patients.

Graduate level researchers in the current study were trained in SCID administration using standard video-tapes marketed by the SCID developers. Reliability assessments were also conducted on standardized SCID reliability
patient videos from the psychosis and mood disorder modules prior to beginning the study. Student raters were able to obtain 100% diagnostic agreement on patient case videos from both modules.

*Schedule for the Deficit Syndrome (SDS).*

The Schedule for the Deficit Syndrome (SDS) (Kirkpatrick et al., 1989) was used to classify patients according to deficit/non-deficit status. The SDS is a semi-structured clinical interview designed to assess severity of negative symptoms in relation to six domains: restricted affect, diminished emotional range, poverty of speech, curbed interests, diminished sense of purpose, and diminished social drive. Severity ratings are made on a 0 (normal relative to characteristic) to 4 (very severe decrease relative to all aspects of characteristic) rating scale. For each symptom domain, symptoms are further classified as being primary/secondary (i.e., idiopathic, not due to secondary negative symptom factors) and stable/unstable (lasting > 1 year). To be classified as a deficit syndrome case, patients must: 1) meet DSM criteria for schizophrenia, 2) evidence moderate or higher (SDS severity of 2 or >) symptom severity on at least two of the six symptom domains, 3) have at least two of these symptoms considered primary, and 4) demonstrate a stable symptom presentation during periods of relative remission over the past year.

*Brief Psychiatric Rating Scale.*

The Brief Psychiatric Rating Scale (BPRS) is commonly employed in the assessment of clinical symptoms associated with schizophrenia. The original BPRS scale consisted of 16 symptom constructs, most of which were derived
from factor analysis of psychiatric rating data (Overall and Gorham, 1963). The current scale consists of 18 items, having added Excitement and Disorganization, that are to be rated. Each item includes a symptom description and a severity rating that consists of a seven-point Likert scale, ranging from not reported/observed (1) to extremely severe (7). Half of the items are rated on the basis of observations made during the 15 to 30 minute interview, while the other half are rated on the basis of self-report data regarding the client’s functioning over the past seven days. Items which predominantly reflect observed behavior include Motor retardation, Uncooperativeness, and Mannerisms and posturing. Items based upon subjective reports from the client include Unusual thought content, Conceptual disorganization, Anxiety, Depressive mood, and Blunted affect. Based upon the most comprehensive factor-analysis of the BPRS, three BPRS syndrome scales were calculated: positive syndrome (grandiosity, suspiciousness, unusual thought content, hallucinatory behavior), negative syndrome (blunted affect, uncooperativeness, emotional withdrawl, motor retardation), and disorganized syndrome (tension, disorientation, excitement, conceptual disorganization, and odd mannerisms and posturing). For the current study, ratings were made in relation to symptoms manifested over the past week.

*Scale for the Assessment of Positive Symptoms.*

The Scale for the Assessment of Positive Symptoms (SAPS; Andreason, 1986) is designed to assess positive symptoms that occur in schizophrenia. Positive symptoms include hallucinations, delusions, bizarre behavior, and positive formal thought disorder. Symptoms were rated over the past week on a
six-point scale, where 0 reflects that the symptom is not at all present and 5 represents a severe presentation. Global ratings are used to represent overall severity within each of these 5 domains, taking into account both the nature and severity of all symptoms observed. A total score is also derived by summing all SAPS items (range 0 to 170).

**Scale for the Assessment of Negative Symptoms.**

The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982) is designed to assess negative symptoms of schizophrenia in relation to 5 core domains: affective flattening, alogia, avolition, anhedonia, and attentional impairment. Examiners rate patients on several clinical domains relevant to each of these core symptoms, and patients are also rated on their subjective evaluation of the symptom as a whole. A global rating is also made for each core domain that takes into account the nature and severity of items within that scale. Ratings are made on a six-point scale (0 not at all to 5 severe) and a total score is also derived by summing all SANS items (range 0 to 150). Ratings were made on a 1-week time frame in the current study.

**The Calgary Depression Scale for Schizophrenia.**

The Calgary Depression Scale for Schizophrenia was designed to assess severity of depression in individuals with schizophrenia. The nine item rating scale is based upon the Hamilton Depression Rating Scale and the Present State Examination, and has been reliably shown to measure depression specific to individuals with schizophrenia. The scale has been shown to assess depression independently of other symptoms present in schizophrenia, in both acute and
residual phases of the disorder. In the current investigation depression scores will serve as a covariate allowing for an assessment of emotion processing without the influence of depression.

Each item consists of several questions designed to assess nine content areas (depression, hopelessness, self-deprecation, guilty ideas of reference, pathological guilt, morning depression, early wakening, suicide, and observed depression), that are each rated on a 0 (Absent) to 3 (severe) scale, based upon the individual's subjective report of thought, feeling, and behavior over the past two weeks. Global depressive symptomatology is typically evaluated using a total severity rating, which is determined by summing each of the nine items.

*Simpson-Angus Extrapyramidal Symptom Scale (SAESS).*

A modified version of the Simpson-Angus scale was administered to measure extrapyramidal side effects. This modified version, used by members of our research group in previous studies (Kelley, van Kammen, & Allen, 1999), contained 8 of the original 10 Simpson-Angus items, excluding leg pendulousness and head dropping, which are not commonly rated. The 8 items adopted from the original scale included gait, arm dropping, shoulder shaking, elbow rigidity, wrist rigidity, glabella tap, tremor, and salivation. Six additional items were also added to obtain a more comprehensive assessment of EPS. These additional items included spontaneity of speech, use of gestures, dystonia, akathisia, akinesia, and cog wheeling. Ratings were made on a 0 to 4 scale, and a total score was tabulated from all items (range 0-56).
Abnormal Involuntary Movement Scale.

The Abnormal Involuntary Movements Scale (AIMS; Guy, 1976) was used to assess involuntary movements occurring in facial/oral, extremity, and trunk domains. The AIMS consists of 12 individual items, which are summed to compute a total score (range = 0 - 42). Higher ratings reflect greater abnormal involuntary movements. These symptoms are common to individuals with tardive dyskinesia.

Rockland Rating Scale (RRS).

The Rockland Rating Scale (RRS) is a 14 item movement rating scale designed to assess extrapyramidal symptoms. Ratings are made on a 1-6 scale across 14 domains measuring abnormality relative to facial and oral movements, neck and trunk movements, extremity movements, and entire body (i.e., akathisia) movements. Scores from the 14 individual items are summed to compute a total score (range 0-84).

Emotion Processing

Emotion processing measures assess either self-reported emotional experience or cognitive/neuropsychological emotion processing. Tests are included to investigate both valence-arousal and discrete basic emotion models, and include several positive (e.g., happiness, surprise) and negative (e.g., sadness, anger, disgust) emotions to assess a full range of human affect. Self-report questionnaires were selected to assess intensity of subjective emotional arousal (positive/negative and basic emotions), and global psychological well-being and positive affect. Cognitive measures are included to assess
performance in general aspects of cognition, such as auditory, verbal, and visual processing, as well as specific domains, including: facial affect identification, hemispheric lateralization, auditory recognition memory, verbal recall and recognition memory, attention bias, and auditory-visual integration. All measures of emotion processing are commonly used in affect assessment, or similar to those used in other studies in the case of computerized tasks, and are suitable for research purposes. Individual measures are detailed in the following section.

**Positive and Negative Affect Scale.**

The Positive and Negative Affect Scale (PANAS) (Watson, Clark, & Tellegen, 1988) is a self-report measure of subjective emotional experience. Participants respond by indicating how intensely they generally experience 20 distinct emotions. The measure includes 10 positively valenced emotions and 10 negatively valenced emotions, which comprise Positive Affect (PA) and Negative Affect (NA) factors. Positive affect items include emotions such as: interested, excited, and strong. Negative affect items include emotions such as: afraid, guilty, and nervous.

Analyses of the PANAS's psychometric properties suggest that the measure displays adequate reliability and validity (Watson, Clark, & Tellegen, 1988). Eight-week test-retest reliability coefficients were found to be reliable for both PA (r = .68) and NA (r = .71) scales, suggesting that test scores obtained on the PANAS are highly reliable. Principle factor analyses yield two distinct factors, which accounted for approximately 63% of the common variance among emotionality ratings, indicating that the measure accurately assesses the
underlying component of emotionality. Additionally, all items have strong primary loadings on their designated factors (r = .50 and above for all items), which suggests that all items are accurate representations of their intended emotions. Validity analyses suggest excellent convergent validity with other emotion measures (.90 and above) and low discriminant validity, providing further evidence that the PANAS is a valid measure of emotion processing. In the current investigation, the PANAS will be used to assess trait positive and negative affect experience in relation to valence-arousal theories of emotion (Russell, 1980).

_Differential Emotions Scale._

The Differential Emotions Scale (DES) (DES; Izard, Dougherty, Bloxom, & Kotsch, 1974) is a 30 item self-report measure designed to assess the frequency with which individuals experience discrete basic emotions in everyday life. The questionnaire yields 10 basic emotion scales, with 3 representing positive emotions (joy, interest, & surprise) and 7 representing negative emotions (sadness, anger, fear, disgust, contempt, guilt, & shame). Examinees are required to identify the frequency with which they experience 30 emotional terms/adjectives on a 4-point scale (0 = not experienced; 3 = highly experienced). DES items were selected to represent verbal labels commonly applied to identify facial expressions, as well as current theories of basic emotion, which describe emotions in terms of discrete categories (e.g., Izard, 1971). The 10 DES factors have been found highly stable across multiple factor-analytic investigations (Fuenzalida, Emde, Pannabecker, & Strenberg, 1981;
Kotsch, Gerbing, & Schwartz, 1982), suggesting that the DES adequately measures the experience of discrete emotions. In the current study, the DES will be applied in its trait form to assess emotional experience on the day of testing. Additionally, this measure has been used in previous schizophrenia investigations, and demonstrated differences in experience among discrete emotions (Suslow et al., 2003).

Facial Affect Labeling Task.

The computerized facial affect labeling task designed for the current study is based upon previous investigations of facial affect in schizophrenia (Loughland et al., 2002). Administration time was approximately 5 minutes. Facial affect stimuli were taken from the Matsumoto and Ekman (1988) stimulus set. All stimuli are proven to be highly namable by healthy individuals (Matsumoto & Ekman, 1988). Stimuli are digitized color images of Caucasian and Asian males and females. Emotions represent 8 primary basic emotions of: happiness, surprise, sadness, anger, fear, disgust, contempt, and neutral. Eight individual stimuli (2 Caucasian males; 2 Asian males; 2 Caucasian females; 2 Asian females) were presented once for each of 6 emotions (happiness, surprise, sadness, anger, fear, neutral), resulting in a total of 48 experimental stimulus presentations. Participants were provided with a list of all 6 possible emotions, and asked to identify the emotion perceived for each face by speaking aloud into a voice activated microphone. Six practice stimuli were also presented, one stimulus from each targeted emotional condition.
A PC and monitor were used to present all facial affect stimuli, which appeared as color images on a grey background. Stimuli will be preceded by a fixation point that remained on the computer screen for 1000 ms. Individual facial affect presentations were presented until participants indicated the perceived emotion by speaking aloud into the voice-activated microphone or until a duration of 5000 ms passed, after which a blank screen appeared prompting participant response. After participant response triggered the voice-activated microphone, a subsequent screen appeared allowing the experimenter to record participant accuracy. Dependent variables for the facial affect identification task included accuracy (% correct) and RT (ms) for emotional conditions.

**Emotional Stroop Task.**

In the current study, two E-Stroop task versions are used to isolate distinct components of attention. Administration time approximated 5 minutes per task. The first version, the Attention Grabbing Task, measures the extent to which emotional information disrupts attentional focus relative to neutral information. This task is designed to allow for a comparison of the extent to which various types of emotional information (happiness, sadness, anger, anxiety) capture or fail to capture the attention of patients with schizophrenia. The second task, the Lingering Effect task, assesses the extent to which an emotional word has a carry-over effect on a string of subsequent neutral words. The lingering effect task therefore allows for a comparison of the extent to which various types of emotional information continue to linger and disrupt on-going cognitive processes, even after they are no longer present.
Similar to J.R. Stroop's (1935) original color-naming task, the E-Stroop task as most commonly used, requires participants to identify the color of ink in which a word is printed, while ignoring the meaning of a written word. However, unlike the Stroop's original task, which manipulates color-congruent and incongruent words, the E-Stroop contrasts emotional and nonemotional (i.e., neutral) words. The theory behind the E-Stroop task states that more influential words cause longer color naming latencies, due to the interfering qualities of the word, which are thought to occur due to an individual's “attention bias” for that particular emotion (Williams, Matthews, & MacLeod, 1996).

The attention grabbing task was modeled after published studies investigating the attention grabbing power of basic emotions (Strauss, Allen, Jorgensen, and Cramer, 2005; Strauss & Allen, 2006). A total of 36 stimuli were presented, six practice and 30 experimental. Practice stimuli consisted of 6 emotional and neutral words not included in the experimental session. In the experimental condition, a total of 30 stimuli were presented, with six words taken from each of five emotional categories (happiness, sadness, anger, anxiety, and neutral). Stimuli were presented via personal computer, and each stimulus appeared on the computer screen for five seconds or until a verbal response was given into a voice-activated microphone. The interstimulus interval (ISI) was set to 1000 ms to ensure that the stimulus onset asynchrony was sufficiently long enough to produce the desired attention grabbing effect that occurs within each trial.

Participants were instructed to name one of four ink colors (red, yellow, green, blue), while ignoring the meaning of the written word. Stimuli were block
presented in a semi-random order with the restriction that no two colors could appear in consecutive trials. This was done to ensure that response time was not biased by the preceding stimulus. All stimuli were presented in uppercase, Arial font (regular), size 18 points, and appeared on the computer screen as color-words presented against a black background.

Words were taken from normative emotional intensity and categorization data developed by Strauss and Allen (in press). All words included evidenced categorization ratings of 70% or higher, indicating that all words are highly representative of their designated categories. Across all 5 categories, words do not statistically differ with regard to emotional intensity, categorization, frequency, or word length. Our research group has previously demonstrated that scores obtained in this test version have high classical test score test-retest reliability (Strauss, et al, 2005).

The commonly employed difference score method is applied to index emotional interference (i.e., attention grabbing effect) using the formula: emotional condition RT - Neutral condition RT. Using this difference score method, higher scores reflect that a given emotional condition has interfered, or captured attention, to a greater extent than neutral words.

The lingering effect task was modeled after parameters reported in a study on non-patients conducted by McKenna and Sharma (2005). A total of 125 stimuli were presented, with 50 practice stimuli consisting of random letter strings, and 75 experimental presentations. The practice session was repeated in instances where participants had difficulty learning button location until a high degree of
accuracy was achieved. Stimuli were presented using a standard PC and responses input using a serial response box that relayed accuracy and RT data to an E-prime data-aid software output file. Stimuli appeared as color words on a black background, and were presented in red, blue, green, and yellow ink. Four response box buttons were covered with colored caps, and a paper overlay was attached that extended up from each button of the box to assist participants from having to look down under their fingers while learning button location.

Participants were instructed to press the button of the color in which the letter string (practice) or emotional/neutral word was printed as quickly and accurately as possible, while trying to ignore reading the written word. Each stimulus appeared on the computer screen for five seconds or until a verbal response was given into a voice-activated microphone. The interstimulus interval (ISI) was set to 32 ms to ensure that the stimulus onset asynchrony was sufficiently long enough to produce the desired "lingering effect". This interval was selected as it was previously found to produce the lingering effect in McKenna and Sharma (2004).

Three blocks (positive, negative, category/neutral) of word stimuli were presented in a counterbalanced order within the experimental condition. Each block included a total of 25 words, which were presented sequentially in 5 series of 5 words. Within each sequence of 5 words, an emotional word was presented in the first position, and was followed by 4 subsequent neutral words (positions 2,3,4,5) that were matched to the emotional word for frequency and length. Thus, there were 5 such sequences of an emotional word followed by four consecutive
neutral words. This presentation resulted in a 3 emotional condition (positive, negative, neutral) x 5 serial position (1 through 5) within-subjects factorial design.

The neutral word in position 2 (i.e., the neutral word immediately following the emotional word in position 1) serves as the primary target in this task, and is used to assess the presence of the lingering effect using a difference score method of: neutral word position 2 - emotional word position 1. Using this difference score, higher RTs reflect a greater lingering effect, suggesting that the emotional content of the word is capable of disrupting on-going attention, even after that stimulus is no longer present.

Positive and negative words included in this task were selected from the normative word set developed by Strauss and Allen (in press). Negative words selected for inclusion were taken from the blended emotional category within the Strauss and Allen norms. This category reflects words that are clearly negative, but not highly representative of a single discrete emotional category. General negative words were selected to avoid an over-sampling of words from any given category, allowing a valid comparison of valence that is not biased by the effects of individual basic emotions. Neutral words used in the positive and negative blocks were selected from the Francis and Kucera normative word frequency manual, and matched to given positive or negative words based upon both length and frequency. Additionally, positive and negative words used within position 1 did not differ with regard to emotional intensity or word frequency, as measured through Strauss & Allen intensity ratings and Francis and Kucera word frequency ratings, respectively. Intensity ratings were not available for neutral words used.
within position 2-5 in the positive and negative blocks; however, their content is clearly neutral, and they thus appear to be a valid neutral comparison.

Similar to procedures used by McKenna and Sharma (2004), a block of category/neutral words were also used as a contrast condition to the positive and negative blocks, and to ensure that interference effects are not due to participants anticipating a target word within the sequence of 5-word presentations. In this category/neutral only block, the word selected for position 1 consisted of a word neutral in emotional content that is semantically related to travel. The words in positions 2-5 were also neutral in content, but were semantically un-related to travel.

*Brief Smell Identification Test.*

Olfactory discrimination will be assessed using the University of Pennsylvania Brief Smell Identification Test (BSIT) (Doty et al., 1996). The measure has been used in schizophrenia investigations (e.g., Goudsmit et al., 2003), and found capable of detecting olfactory processing deficits. The BSIT is a standardized measure that requires examinees to identify 12 common microencapsulated odors by selecting one of 4 multiple-choice answers representing various odor names. Smell identification scores are calculated by totaling the number of correct responses. Raw scores are compared to age and gender based norms (Doty, 1995). Doty et al. (1996) reported that the BSIT has adequate reliability in the normative sample (r=.71). The BSIT also has adequate validity, as indicated by satisfactory separation of test items, suggesting that the SIT is able to
distinguish between individuals with average ability and those with moderate impairment.

Participants also provided valence ratings for each BSIT item. In a procedure similar to that adopted by Doop and Park (2005), odor valence was rated on a 7-point scale (1 = extremely pleasant; 4 = neutral; 7 = extremely unpleasant).

*Emotional Verbal Learning Test.*

The Emotional Verbal Learning Test (EVLT) (Strauss & Allen, unpublished manuscriptb) is a measure of learning and memory for emotional words. All procedures, parameters, and score calculations are modeled after the CVLT-II (Delis, 2000), allowing for a direct comparison between learning and memory performance for emotional and neutral words. The EVLT is a standardized measure with psychometric properties comparable to the CVLT-II (internal consistency, $r = .88$).

The task first requires the experimenter to orally present 16 words (List A) over five immediate-recall trials. The list consists of 4 words from each of four "basic emotion" categories (Happiness, Sadness, Anger, and Anxiety). Individual words of the same category are never presented successively to allow for the assessment of semantic clustering. Following the 5 immediate recall presentations, a second "interference" list (List B) is presented for a single trial. Immediately following List B, a short delay free and category cued recall of List A is administered. A 20 minute delay then occurs between the presentation of the short-delay and long-delay free recall assessments. Long delay free and cued
recall is then assessed. Recognition of List A is measured using a yes-no recognition format immediately proceeding the administration of the long delayed recall. In the test of recognition, there are 28 distractors consisting of: List B emotional words semantically related to List A words, List B emotional words semantically unrelated to List A words (disgust words), novel words that are prototypical of semantic categories presented in list A, and emotional words semantically unrelated to List A words (fear and surprise words).

Administration time is similar to the CVLT-II (Delis et al., 2000), taking approximately 30 minutes to complete. Time estimates include a 20 minute delay interval in-between short and long-delayed recall sections. Unlike the CVLT-II, the EVLT does not contain a Long-delay forced choice recognition test section, which subsequently results in a shorter administration time since the 10 minute delay interval is not included.

The EVLT measures both recall and recognition of two separate word lists, comprised of emotional words, in several short and long-delayed memory trials. As such, the test can measure a number of different aspects of learning and memory for emotional words, including: total recall and recognition, primacy-recency effects in recall, differences in retention after short and long delays, and recognition memory. Measurements of emotional word learning can also be obtained, including measures of learning strategies (semantic, serial, and subjective clustering), emotional learning slope, and the amount of new learning per trial over the first five presentations of List A. Normative data collected on the EVLT suggest that psychologically healthy individuals evidence a memory
bias for happiness words (Strauss & Allen, unpublished manuscript). This memory bias is not accounted for by list order, word frequency, word length, or emotional prototypicality.

California Verbal Learning Test-II.

The CVLT-II (CVLT-II; Delis et al., 2000) is an individually administered clinical instrument designed to quantify various aspects of verbal learning and memory. The CVLT-II is an updated version of the first edition of the CVLT (Delis et al., 1987), which was one of the first clinical instruments to utilize theories of cognitive psychology to measure multiple facets of learning and memory with clinical populations. It has been used in studies of numerous clinical disorders, including schizophrenia (Paulsen et al., 1994).

The task first requires the experimenter to orally present 16 words (List A) over five immediate-recall trials. The list consists of 4 words from each of four categories (furniture, vegetables, ways of traveling, and animals). Individual words of the same category are never presented successively, to allow for the assessment of semantic clustering, which is regarded as the most efficient strategy for learning verbal information. Following administration of the five trials, a second 16 word “interference list” (List B) is presented for a single trial. Immediately following List B, a short delay free recall trial and a category cued recall trial of List A are conducted. Twenty minutes later, long delay free recall, category cued recall, and recognition trials occur for List A. Recognition of List A is measured using a yes-no recognition format immediately in which List A is presented with 28 distractors consisting of: List B words semantically related to
List A words, List B words semantically unrelated to List A words, novel words that are prototypical of semantic categories presented in list A, and novel words semantically unrelated to List A words.

The CVLT-II (Delis et al., 2000) was standardized on a sample of 1,087 individuals between the ages of 16 to 89. Administration time for individuals under 60 years is approximately 47 minutes, while administration time for individuals over 60 years is approximately 51 minutes. Nine measures of memory are selected for analysis in the current study: Trial 1 total recall correct, Trials 1-5 total recall correct, List A short delay recall correct, List A long delay recall correct, Total recognition hits, Trials 1-5 total repetitions, trials 1-5 total intrusions, primacy recall correct, and recency recall correct.

Premorbid Intelligence.

Two subtests from the WAIS-III (Wechsler, 1997a), Vocabulary and Information, will be used to estimate premorbid intelligence in persons with schizophrenia and verbal IQ in healthy controls. These subtests are commonly used to estimate premorbid IQ in schizophrenia, as scores do not markedly change over time or in the presence of brain insult, and because reliability estimates are highest among all WAIS-III verbal composite subtests (Vocabulary = .89; Information = .96) (Vanderploeg, Schinka, & Axelrod, 1996).

The Vocabulary subtest from the Wechsler Adult Intelligence Scale III (Wechsler, 1997) consists of 33 items that require the subject to provide definitions for words of varying degrees of difficulty. Item scores can range from 0-2, with a two point score indicating a full credit response, one point indicates a
partial credit response, and 0 points represent an incorrect response or lack of a response.

The Information subtest from the Wechsler Adult Intelligence Scale III (Wechsler, 1997b) consists of 28 items that require a general knowledge of current and historical events. Items are scored as either correct or incorrect and the total number of correct items serves as the raw score.
CHAPTER 4

RESULTS

Data Entry and Screening

All data was double scored and entered to ensure accuracy of findings reported. Several preliminary analyses were conducted prior to the evaluation of main hypotheses. First, raw test data were inspected to ensure that they meet the assumptions for ANOVA. Descriptive statistics and box plots were calculated for each of the variables, and skewness and kurtosis were examined to ensure that all variables were normally distributed. Box plots were used to evaluate the presence of outliers. In cases where variables were not normally distributed, transformations were used to increase the normality of the distribution. Transformations were selected in accordance with the recommendations of Tabachnik and Fiddel (2001). For individual tests, scores that were 3.0 standard deviations above/below the mean were considered outliers. Score transformations are described in the following results section in relation to individual tests and DVs.

Descriptive statistics and box plots were also calculated for raw computerized task data in order to eliminate individual RT outliers. In cases where mechanical errors were present in computerized tasks (i.e., microphone does not signal upon voice onset), these data were eliminated from analyses as invalid data. Individual
RTs greater than 2.0 SDs above each condition's mean (calculated per group), or less than 150 ms, were considered outliers and eliminated from the data set. Once individual RT outliers and mechanical errors were removed from the data set, means were calculated for each level of each independent variable. These means were used in all subsequent main analyses conducted on RT data. Outlier procedures were adopted from a study conducted by Ratcliff (1993), which suggested that statistical power is considerably impacted when outliers are ignored in RT data. The SD rule adopted was one of the recommended procedures for eliminating outliers in investigations where there is high variability among subject responding (which is certainly true for patients with schizophrenia). In the simulation, Ratcliff tested cutoffs only up to 1.5 SD from the mean. Our elimination of outliers 2.0 SD above the mean is even conservative based upon these standards, and was applied to ensure that too much data was not discarded by using a liberal outlier rule. Further, the total amount of data eliminated approximated 1% of the raw data, which is what would be expected based upon a 2.0 SD cut-off. Although an even more conservative 3.0 SD rule was considered, this procedure was found to include RTs exceeding 4 seconds, which is an absolute eon by typical RT standards. Thus, outlier procedures adopted provide greater assurance that overall subject means exclude individual trials where extraneous conditions, such as momentary difficulty focusing on the task, influenced task performance.
Demographic and Clinical Symptom Analyses

Groups were also evaluated for differences on basic demographic variables including age, education, estimated IQ, race, and gender. ANOVA (age, education, IQ) and chi-square (race, gender) analyses were used to make these comparisons. Means, standard deviations, and statistical test data are presented in Table 1. ANOVA indicated that the 3 groups (deficit schizophrenia, non-deficit schizophrenia, control) did not significantly differ on age and education. As such, age and education were not used as covariates in the main analyses.

Patient and control groups also did not differ with regard to the proportion of right handed individuals within the sample. However, groups significantly differed with regard to race and education, such that the deficit syndrome group had a greater proportion of African-American participants than non-deficit patients or controls, and that both patient groups had a significantly higher proportion of males than the control group.

Patient and control groups significantly differed in intellectual functioning, such that both patient groups were significantly lower than the control group on the WAIS-III Vocabulary, Information, and Block Design subtests. Statistically significant differences were also found between deficit and non-deficit patients with regard to Information and Block Design subtests, but not Vocabulary.

Given these differences IQ was included as a covariate in the analyses of the main hypothesis. These analyses were accomplished primarily for comparisons purposes, although their significance is questionable as it pertains
to neurocognitive and emotion processing differences between the schizophrenia groups. The reason for this was highlighted recently by Goldstein, Seidman, and Tsuang (1999), who suggested that matching diagnostic groups on variables that are not independent of the illness being examined can result in a matching fallacy, whereby variance directly attributable to the variable of interest is removed due to overmatching on a variable, such as IQ. In studying groups like patients with schizophrenia, which are known to display significant neurodevelopmental abnormalities, where the illness in and of itself effects variables such as premorbid IQ would arbitrarily attenuate differences among groups. This may be particularly problematic when studying deficit syndrome patients, as these individuals are known to display lower premorbid IQ and functioning than non-deficit patients. As such, although main hypotheses were conducted using IQ as a covariate, covarying out the effects of IQ may be inappropriate in this case, as it arbitrarily removes variance natural to the neural basis of the deficit syndrome, and results are primarily interpreted without regarding the influence of IQ.

Analyses were also conducted on clinical and demographic variables recommended for comparison by SDS authors. These comparisons are designed to reduce problems related to intergroup reliability, and to ensure that deficit syndrome patients reported here validly represent the concept of deficit syndrome schizophrenia. Based upon recommendations outlined in the SDS manual, analyses were first conducted on clinical characteristics to ensure that deficit syndrome validating variables are not confounded by longer length of
illness, earlier age of onset, higher daily dosage of antipsychotic medication, a different frequency of medication use, or severity of extrapyramidal symptoms. ANOVA indicated that deficit and non-deficit patients did not differ with regard to length of illness, age of onset, daily antipsychotic medication dosage (chlorpromazine equivalent dosage; Woods, 2003), or severity of extrapyramidal symptoms (see Table 4). Additionally, deficit and non-deficit patients were prescribed a similar regiment of conventional, atypical, anti-depressant, mood-stabilizer, anti-parksinsonian, and extrapyramidal medications (see table 4).

Second, analyses were conducted on symptom measures for which deficit patients are known to significantly differ from non-deficit patients. Based upon the literature, it would be expected that deficit patients would display less severe symptoms of emotionality, depression, suspicion, and general positive symptoms. Deficit patients should also evidence significantly higher scores on most negative symptoms, and less or at least equal levels of disorganization. ANOVA indicated that deficit patients demonstrated a symptom profile consistent with the published literature. In comparison to the non-deficit group, deficit patients evidenced significantly lower scores on BPRS scales of emotionality (anxiety, depressed mood, guilt, hostility) and suspicion (see Table 5), and the SAPS total score (see Table 7). Deficit patients also showed a trend (p = .062) toward less global depressive symptoms as indexed by the Calgary Depression Scale total score (see Table 6). Additionally, deficit patients received higher ratings on BPRS measures of emotional withdrawal and blunted affect, as well as the SANS total score (see Tables 5 and 8), suggesting that they evidence higher
levels of negative symptoms than non-deficit patients, as would be expected. No differences were found between deficit and non-deficit groups on the SAPS Disorganization Global Rating or the BPRS Disorganization Syndrome Factor, although the deficit group received lower ratings on both scales (see Tables 5 and 7). These findings are consistent with clinical characteristics reported for deficit and non-deficit patients in the published literature.

Third, descriptive statistics were analyzed for basic demographic variables known to differentiate deficit and non-deficit patients based upon multi-site clinical field studies. Consistent with previous reports, the deficit group displayed a higher proportion of male than female subjects, lower estimated IQ (see Table 1), and less frequent rate of marriage than non-deficit patients and controls (see Table 2).

Fourth, diagnostic groups were compared on smoking behavior, as this variable may affect olfactory performance (see Table 3). Chi Square and ANOVA results indicated that groups did not significantly differ in the proportion of smokers within each group or the number of cigarettes smoked per day.

Finally, deficit patients were compared on SDS characteristics to ensure that severity ratings, and the relative percentage of patients displaying primary and enduring negative symptoms is consistent with patients in the published literature (see Table 9). Deficit patients evaluated in the current study displayed symptom severity ratings of a similar magnitude to most published studies, including those conducted by SDS authors. The percentage of deficit patients displaying symptoms which are at least moderate in severity was also
comparable to previous reports, as were the percentage of patients with primary and enduring negative symptoms.

Primary Analyses

Emotional Experience

Hypothesis 1.

Three repeated measures ANOVAs were conducted to examine the effects of diagnosis on state and trait subjective emotional experience. In the first analysis examining trait experience, a repeated measures ANOVA was calculated in which diagnostic group served as a between-subjects factor, and emotion valence (positive, negative) as a within-subjects factor, in order to determine whether there were group differences in trait experience of positive and negative emotion. Total trait affect scores from the Positive and Negative Affect Scale (PANAS) served as the dependent variable (range 20-100). A significant within subjects effect was found for emotion, $F (2, 60) = 92.03, p < .001$ (eta square = .63); however, the between-subjects effect for diagnostic group was nonsignificant, $F (2, 60) = 1.81, p = .17$ (eta square = .06). As hypothesized, a significant trait emotion X group interaction, $F (2, 60) = 3.56, p = .04$ (eta square = .12) was present, which is presented in Figure 1. One-way ANOVAs were conducted to determine the significance of the overall emotion X group interaction, and to explore hypothesized differences among deficit and non-deficit patients. A statistically significant difference was found for trait positive, $F (1, 60) = 4.44, p = .016$, but not negative experience, $F (1, 39) = 0.37$. 

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Post hoc Scheffe analyses indicated that deficit syndrome patients reported experiencing significantly less intense positive emotion than non-deficit patients and a trend toward less experience than controls ($p = .060$). Thus, the significant emotion X group interaction reflects that deficit patients experience less positive emotion than non-deficit patients or controls, and relatively equal levels of trait negative emotion. Means, standard deviations, and post hoc results are presented in Table 10.

Given the IQ difference present among the groups, the repeated measures ANOVA was conducted again, with IQ entered as a covariate, in order to determine whether the group differences in trait experience of positive and negative emotion were accounted for by IQ differences among the groups. Repeated Measures ANCOVA indicated that the emotion X IQ interaction was nonsignificant, $F(2, 60) = 0.13, p = .717$ (eta square = .002), indicating that IQ differences among the groups did not influence the differences in trait emotional experience resulting from diagnostic status.

The second analysis examined intensity of state emotional experience. Repeated measures ANOVA was conducted with diagnostic group entered as a between-subjects factor and emotion valence (positive, negative) as a within-subjects factor. Total state affect scores from the Positive and Negative Affect Scale (PANAS) served as the dependent variable (range 20-100). Results indicated a statistically significant main effect for emotion, $F(2, 60) = 123.27, p < .001$ (eta square = .67) and a significant between-subjects effect for group, $F(2, 60) = 3.32, p = .044$ (eta square = .11). These significant findings are presented
in Figure 1, and reflect that a greater intensity of state affect was experienced for positive than negative emotions across groups. Also, controls experienced significantly less overall state emotion than both patient groups, and the patient groups experienced higher level of state negative affect than controls. Contrary to hypotheses, there was only a trend toward a statistically significant state emotion x group interaction, $F(2, 60) = 1.84, p = .17$ (eta square = .06).

ANCOVA was conducted to assess the effects of IQ on state emotional experience. Results indicated a nonsignificant emotion X IQ interaction, $(2, 60) = 0.13, p = .724$ (eta square = .002), indicating that IQ is not accounting for differences in state emotional experience.

The third analysis examined frequency of emotional experience in relation to basic emotion theories (e.g., Ekman, 1996). Using diagnostic group as a between-subjects factor, and emotion (12 levels: joy, interest, surprise, sadness, anger, fear, disgust, contempt, guilt, shyness, hostility, and shame) as a within-subjects factor, a repeated measures ANOVA was calculated to determine whether there were group differences in the experience of individual basic emotions. The 12 levels of emotion were from the Differential Emotions Scale (DES), and the scores obtained for each level served as the dependent variable. Mauchly's Test of Sphericity indicated that assumptions of sphericity were violated. As such, the Greenhouse-Geisser correction was applied, and significance levels reflect this correction. A significant main effect of emotion was found, $F(11, 60) = 26.72, p < .001$ (eta square = .32). However, the between subjects effect for group was nonsignificant, $F(2, 60) = 1.33, p = .272$ (eta
square = .63). Consistent with the hypothesis, repeated measures ANOVA also indicated a statistically significant emotion X group interaction, $F(22, 60) = 2.48$, $p < .001$ (eta square = .08), which is presented in Figure 2. As will be noted from the Figure, the interaction effect reflects that diagnostic groups largely did not differ with regard to the experience of discrete negative emotions, while deficit syndrome patients displayed a unique diminished capacity to experience individual positive emotions. This significant interaction was followed by individual one-way ANOVAs to further determine the nature of these differences. Results indicated statistically significant differences in frequency of experience for the positive emotions of interest, $F(11, 60) = 4.28$, $p = .019$, joy, $F(11, 60) = 6.76$, $p = .002$, and a trend toward a difference in surprise, $F(11, 60) = 2.60$, $p = .083$. Significant differences were also found for the negative emotion shame, $F(11, 60) = 3.36$, $p = .042$, and there was a trend toward significant differences in reported experience of fear, $F(11, 60) = 2.82$, $p = .068$. Differences were nonsignificant for all other negative emotions. Post hoc Scheffe analyses were conducted for emotions where significant differences were found. Results indicated that deficit patients experience both joy and interest significantly less than non-deficit patients and controls. Means, standard deviations, and post hoc results are presented in Table 10.

An ANCOVA conducted to determine whether IQ influenced the experience of discrete emotions was nonsignificant, $F(22, 60) = 0.54$, $p = .877$ (eta square = .01). This indicates that IQ did not account for differences found across diagnostic groups with regard to the frequency of experienced emotion.
Emotional Perception

Hypothesis 2 and 3 were concerned with potential differences in emotion perception among the groups. These differences were assessed using two types of tasks, a facial affect identification task (Hypothesis 2) and a test of olfaction (Hypothesis 3).

Hypothesis 2.

Two separate repeated measures ANOVAs were conducted to assess the effects of diagnosis on facial affect perception. For these analyses, performance on the facial affect identification task was examined. Using diagnostic group as a between-subjects factor and facial affect (6 levels: happiness, surprise, sadness, anger, fear, neutral) as a within-subjects factor, a repeated measures ANOVA was calculated to determine whether there were group differences in the mean accuracy (% correct) for emotional faces. Mauchly’s Test of Spericity indicated that assumptions of sphericity were violated. As such, the GreenHouse-Geisser correction was applied, and significance levels reflect this correction. The ANOVA indicated a significant main effect of emotion, $F(5, 36) = 40.8, p < .001$ (eta square = .41), and a significant between subjects effect for group, $F(2, 36) = 9.79, p < .001$. However, contrary to hypotheses, there was a nonsignificant emotion X group interaction, $F(5, 59) = 0.95, p = .487$, indicating that all diagnostic groups had similar levels of difficulty and accuracy at identifying individual emotions. The findings are presented in Figure 3. The significant within-subjects effect of emotion and between subjects effect of group can mostly be attributed to very high rates of accuracy for happiness and low rates for fear,
along with deficit patients being generally less accurate on affect identification than non-deficit patients, who were in turn less accurate than controls.

An ANCOVA was then conducted with IQ as a covariate. Results indicated a nonsignificant emotion X IQ interaction, \( F(10, 36) = 1.38, p = .233 \) (eta squared = .03), suggesting that IQ does not account for group differences in emotion identification.

For the second repeated measures ANOVA, diagnostic group as a between-subjects factor and facial affect (6 levels: happiness, surprise, sadness, anger, fear, neutral) as a within-subjects factor. However, rather than number correct, group differences in the speed with which facial displays of discrete basic emotion were used as the dependent variable. For these analyses, only those faces where correct responses were obtained were included. Results indicated a significant main effect of emotion, \( F(5, 36) = 17.03, p < .001 \) (eta square = .32). There was also a significant between subjects effect for group, \( F(2, 36) = 24.66, p < .001 \). Consistent with hypotheses, there was also a significant emotion X group interaction, \( F(10, 36) = 2.13, p = .024 \) (eta squared = .11), which is presented in Figure 4. The significant emotion X group interaction can largely be attributed to differences in the processing of positive vs. negative emotions, which was particularly apparent for the deficit syndrome group. While all groups evinced an increase in processing speed for happy faces (indicated by shorter reaction times), this was particularly evident in the deficit syndrome group, who exhibited much slower processing for negative faces, especially fear and sadness. To further examine this interaction, one-way ANOVAs were then
conducted to determine the nature and significance of these differences among
the groups, and to explore hypothesized differences among deficit and non-
deficit patients. Results indicated statistically significant differences for all 6
emotional conditions: Anger = \( F (2, 57) = 19.98, p < .001 \); Fear = \( F (2, 45) = 27.26, p < .001 \); Happiness = \( F (2, 59) = 29.87, p < .001 \); Neutral = \( F (2, 55) = 33.50, p < .001 \); Sadness = \( F (2, 54) = 18.47, p < .001 \); Surprise = \( F (2, 59) = 32.07, p < .001 \). One-way ANOVAs were followed up by Scheffe post hoc
comparisons. Results indicated that deficit patients labeled facial affect stimuli,
for all 6 emotional conditions, significantly more slowly than non-deficit patients
and controls. Non-deficit patients also processed all 6 facial affect conditions
significantly more slowly than controls, who were fastest at processing all
affective conditions. Means, standard deviations, and post hoc results are
presented in Table 11.

To examine the effects of IQ on facial affect labeling speed, ANCOVA was
conducted using IQ as a covariate. Results indicated a nonsignificant emotion X
group interaction, \( F (10, 36) = 1.15, p = .337 \) (eta squared = .04), indicating that
group differences in IQ do not account for the observed effects of diagnostic
group on facial affect labeling time.

*Hypothesis 3.*

A One-Way ANOVA was conducted to assess the effects of diagnosis on
smell perception. Smell perception was assessed using the University of
Pennsylvania Brief Smell Identification Test (SIT). BSIT total recognition scores
served as the dependent variable (range 0-12). ANOVA indicated statistically
significant differences in total smell identification accuracy among diagnostic
groups, $F (2, 60) = 14.34, p < .001$. Post hoc Scheffé tests indicated that deficit
patients performed significantly worse than non-deficit patients and controls;
however, differences between non-deficit patients and controls were
nonsignificant (see Figure 5). Total BSIT accuracy means, standard deviations,
and post hoc data are presented in Table 12.

Two repeated measures ANOVAs were then conducted to examine
potential differences in accuracy and valence judgment within positive and
negative odors. Individual items were divided into pleasant and unpleasant
categories using normative valence ratings published by Doty, Shaman, and
Dann (1984), which are also available within the 40-item UPSIT manual. Ratings
for each odor were made on a 5 point scale, with 5 representing extremely
unpleasant, 3 representing a neutral reference point, and 1 representing
extremely pleasant. Items included within the pleasant category had mean
valence ratings that fell toward the pleasant end of the neutral reference point.
Similarly, items were considered unpleasant if they fell toward the unpleasant
end of the neutral reference point. This division resulted in 7 positive and 5
negative items.

Using diagnostic group as a between-subjects factor, and odor valence
(pleasant, unpleasant) as a within-subjects factor, a repeated measures ANOVA
was calculated to determine whether there were group differences in the
identification of pleasant and unpleasant odors. Accuracy per emotional condition
(\% correct) served as the as the dependent variable. Mauchly's Test of Spericity
indicated that assumptions of sphericity were violated. As such, the Greenhouse-Geisser correction was applied, and significance levels reflect this correction.

Results indicated that the main effect for emotion was significant, $F(1, 60) = 5.81, p = .019$ (eta square = .10), which can be attributed to all diagnostic groups displaying greater accuracy for pleasant than unpleasant odors. There was also a significant between-subjects effect for group, $F(2, 60) = 16.03, p < .001$ (eta square = .37). This significant effect of diagnostic group reflects that controls displayed the highest performance, followed by non-deficit patients, and deficit patients (see Figure 6). Contrary to hypotheses, the emotion X group interaction was nonsignificant, $F(2, 60) = 1.26, p = .292$.

To further evaluate the hypothesis, separate one-way ANOVAs were then calculated to determine whether differences existed among groups for both pleasant and unpleasant items. Results indicated statistically significant differences in the overall magnitude of performance on pleasant item accuracy, $F(2, 60) = 8.473, p = .001$, and unpleasant item accuracy, $F(2, 60) = 13.53, p < .001$. Post hoc Scheffé analyses were conducted to determine the nature of differences among groups. Results indicated that deficit patients performed significantly more poorly than non-deficit patients and controls for both pleasant and unpleasant items. However, non-deficit patients and controls did not significantly differ on accuracy for either pleasant or unpleasant items. These results indicate that deficit patients are significantly less accurate at identifying both pleasant and unpleasant odors than non-deficit patients and controls, while non-deficit patients do not significantly differ from controls on either pleasant or
unpleasant odor identification. Means, standard deviations, and post hoc data are presented per emotional condition in Table 12.

ANCOVA, conducted with IQ as a covariate, was nonsignificant, $F(1, 60) = 1.72, p = .195$ (eta square = .03), indicating that IQ did not influence group differences in positive and negative odor identification.

Analyses were also conducted to examine the effects of diagnostic group on judging the valence of pleasant and unpleasant odors. Diagnostic group served as a between-subjects factor and odor valence (pleasant, unpleasant) as a within-subjects factor. Mean valence judgment ratings per odor condition (range 1: extremely pleasant to 7: extremely unpleasant) served as the dependent variable. Repeated measures ANOVA indicated a significant main effect for emotion, $F(1, 60) = 110.49, p < .001$ (eta square = .67) and a nonsignificant between subjects effect for group, $F(2, 60) = 1.44, p = .245$ (eta square = .05). As hypothesized, there was also a statistically significant emotion X group interaction, $F(2, 60) = 4.94, p = .011$, which is presented in Figure 7. One-way ANOVAs were used to follow-up the significant emotion x group interaction, and indicated a trend toward significant differences in valence judgments of both pleasant, $F(2, 60) = 2.58, p = .085$, and unpleasant items, $F(2, 60) = 2.96, p = .06$. These analyses suggest that deficit patients rated positive items as being significantly more neutral than controls, and that they tended to rate pleasant items as being somewhat more neutral than non-deficit patients, although this was only at a trend level of difference. Deficit patients also displayed a trend toward rating unpleasant items as being more neutral than
controls, but did not significantly differ from non-deficit patients. Additionally, non-deficit patients do not differ from controls on judging pleasant items, but rate unpleasant items as being more neutral than controls. Although only a trend level of significance was achieved, the results are generally consistent with the notion that both deficit and non-deficit patients judge unpleasant items as being more neutral than controls. However, deficit patients display a unique impairment in judging pleasant items as being more neutral than either the non-deficit patients or the controls. Valence judgment means, standard deviations, and post hoc results are presented per emotional condition in Table 12.

Considering that groups significantly differed on IQ, ANCOVA was conducted to determine whether IQ influenced valence ratings across groups. A nonsignificant emotion X IQ interaction was present, $F(1, 60) = 0.20, p = .654$ (eta square = .03), indicating that IQ did not account for differences in valence ratings found between groups.

Cognitive Processing of Emotion

Hypothesis 4 and 5 were concerned with potential differences in cognitive processing of emotional information for the deficit and non-deficit groups. Two fundamental processes were examined, memory (Hypothesis 4) and attention (Hypothesis 5).

**Hypothesis 4.**

To evaluate potential differences in learning and memory for emotional information among the groups, two separate repeated measures ANOVAs were conducted, one examining memory bias and the other examining acquisition of
emotional information (learning). The first analyses investigated whether there were group differences in emotional word recall for discrete emotional categories as measured by the EVLT, with diagnosis serving as a between-subjects factor, and emotion as a within-subjects factor (4 levels: happiness, sadness, anger, anxiety). For each of the four levels of emotion, separate memory bias scores were calculated using the following formula: EVLT Emotion Condition Trials 1-5 Total Recall / EVLT Trials 1-5 Total Recall * 100. For each emotion, this memory bias score reflects the relative proportion of words recalled out of the total number of words recalled across the five learning trials, and therefore provides an estimate of the relative bias in recall toward a given emotion. Results of the ANOVA indicated a significant main effect for emotion, $F(3, 60) = 8.73, p < .001$ (eta square = .13), nonsignificant between-subjects effect for group, $F(2, 60) = 0.780, p = .463$ (eta square = .03), and a significant emotion X group interaction, $F(6, 60) = 2.54, p = .022$ (eta square = .08), indicating that the pattern of recall bias differs across diagnostic groups (see Figure 8). As the figure demonstrates, the interaction effect results from the deficit patients' unique bias toward recalling anger, which is not seen in non-deficit patients or controls. Additionally, they do not display the memory bias for happiness seen in both non-deficit patient and control groups, who consistently have a higher proportion of recall for happiness words relative to sadness, anger, and anxiety. Thus, deficit syndrome patients do not display a memory bias for positive emotion. For comparison purposes, descriptive statistics for the raw data are also presented for total recall across trials 1-5 per emotion (i.e., raw recall means per emotional condition) in Figure 9.
This significant interaction was followed by individual one-way ANOVAs and post hoc Scheffé analyses to clarify the nature of differences. One-way ANOVA indicated statistically significant differences in memory bias for anger between the groups, $F(2, 62) = 3.328, p = .043$, and a trend toward differences in happiness, $F(2, 62) = 2.08, p = .134$, and anxiety, $F(2, 58) = 2.87, p = .065$. Post Hoc analyses indicate that deficit patients evidence a significantly greater memory bias for anger than controls ($p = .044$); however, differences between deficit and non-deficit patients and non-deficit patients and controls were nonsignificant.

To determine whether these findings were influenced by significant differences in group IQ, ANCOVA was conducted using IQ as a covariate. Results indicated a nonsignificant emotion X IQ interaction, $F(3, 60) = 1.31, p = .272$ (eta square = .02), indicating that IQ did not significantly influence differences in emotional memory bias.

The second set of analyses were then conducted to examine group differences in emotional and non-emotional word recall in order to determine whether emotional memory impairments reflect a diminished capacity for learning emotional information in patients with the deficit syndrome. In these analyses measures of emotional learning and memory (EVLT) and non-emotional learning and memory (CVLT) were included in a two test (CVLT vs. EVLT) X five trial (1 through 5), X 3 group repeated measures ANOVA. Results are presented in Figure 10. Significant main effects were found for test, $F(1, 58) = 14.77, p < .001$, and trial, $F(4, 58) = 141.91, p < .001$. These differences can be attributed
to participants evidencing greater recall on the CVLT than EVLT, and the expected learning slope occurring across the 5 trials on both tests. A significant trial X group interaction was found, $F(8, 58) = 8.57, p < .001$, which indicates that non-deficit patients and controls demonstrated a typical learning increase across each of the 5 trials, on both the EVLT and CVLT, and a subsequent failure of deficit syndrome patients to display learning effects on either of the two memory tests. A significant test X trial interaction was also found, $F(4, 58) = 3.35, p = .01$. This significant interaction can be attributed to a steeper initial learning slope (trials 1 – 3) for the CVLT with a leveling out of performance on trials 4 and 5. In contrast, the EVLT demonstrated a gradual increase in learning across the five trials. Results also indicated a nonsignificant test X group interaction, $F(2, 58) = 2.01, p = .14$, and a nonsignificant test X trial X group interaction, $F(8, 60) = 0.95, p = .48$. Although these interactions were nonsignificant, when diagnostic group is considered, results are consistent with the idea that both deficit and non-deficit patients display impaired memory for emotional and non-emotional information relative to controls, with deficit patients exhibiting even greater impairment than non-deficit patients. Additionally, non-deficit patients and controls both recalled significantly fewer emotional than neutral words, and display increased learning across the five immediate recall trials for both emotional and emotional words. These patterns were not found in deficit syndrome patients who failed to display superior recall for non-emotional words, and were unable to make gains in emotional and non-emotional word learning after the 2nd (EVLT) and third (CVLT) learning trials, respectively. These
findings suggest that while emotional information impacts memory in similar manner for controls and patients with non-deficit schizophrenia such that emotional information is more difficulty to learn, this disparity is not present for deficit patients. Rather, learning in deficit patients is not affected by the emotional content of the word, or put another way, emotional information is not salient for them and is processed like neutral information. These findings are consistent with an emotion processing abnormality in patients with deficit syndrome.

ANCOVA was also conducted, entering IQ as a covariate, to examine the effects of IQ differences on emotional and nonemotional word recall. There was a significant trial X IQ interaction, $F(4, 58) = 3.27, p = .012$, nonsignificant test X IQ interaction, $F(1, 58) = 0.00, p = .964$, and a significant test X trial X IQ interaction, $F(4, 60) = 2.73, p = .03$. These results indicate that while IQ does not influence global differences in emotional and nonemotional memory, it does have impact on general learning, and differentially impacts the rate at which emotional and non-emotional information is learned. Estimated marginal means are plotted for EVLT and CVLT data across the 5 immediate learning trials in Figure 12 to demonstrate the effects of partialing out the influence of IQ on the learning and memory measures. As the Figure illustrates, the IQ effects can largely be attributed to IQ impacting EVLT scores of deficit syndrome patients, causing them to drop in later recall trials (particularly 3 and 4), in comparison to calculations not accounting for IQ. Thus, IQ appears to be associated with the learning impairments associated with emotional information that are seen in
deficit syndrome patients. However, it does not moderate the abnormality originally noted in which it appeared that emotional information was not salient for those with deficit syndrome.

*Hypothesis 5.*

Two separate repeated measures ANOVAs were conducted to examine the effects of diagnosis on attentional processing. In the first analysis, attention bias was assessed using the E-Stroop Attention Grabbing Task. E-Stroop difference scores calculated for each of the 4 emotional conditions (happiness, sadness, anger, anxiety) served as the dependent variable (difference score = emotional condition RT - Neutral condition RT). Results indicated a significant main effect of emotion, $F(3, 56) = 2.76, p = .044$, indicating that there were significant differences in the extent to which individual emotions captured attention across participant groups. As can be seen from Figure 12, these differences can largely be attributed to anxiety words capturing attention to a greater extent than other emotions, and happiness capturing attention less than anger, anxiety, and sadness. The between-subjects effect of group was nonsignificant, $F(6, 56) = 1.02, p = .367$, indicating that the overall magnitude of E-Stroop interference does not differ among groups. Although unexpected, the emotion X group interaction was nonsignificant, $F(6, 56) = 1.34, p = .242$, indicating that the pattern of attentional capture for emotional information did not significantly differ as a function of diagnostic group (see Figure 12). ANCOVA was also conducted, using IQ as a covariate to assess the influence of group IQ differences on attention bias, and indicated a nonsignificant emotion X IQ
interaction, $F(3, 56) = 0.871, p = .458$. This nonsignificant interaction indicates that IQ did not influence group differences in attention bias for emotional information.

A one-way ANOVA was also conducted to examine the specific sub-hypothesis that happiness would fail to grab attention (i.e., a lack of attention bias) in deficit syndrome patients. Consistent with hypotheses, results indicated that deficit, non-deficit, and control participants significantly differed with regard to attention bias for happiness, $F(2, 58) = 4.23, p = .019$. Post hoc Scheffé analyses indicate that these differences can be attributed to deficit patients displaying significantly less attention bias for happiness than non-deficit patients ($p = .021$), as well as a trend toward less attention bias for happiness than controls ($p = .141$).

The second series of analyses examined the extent to which emotional information continues to disrupt on-going attentional processes, even when it is no longer present, using the E-Stroop Lingering Effect task. A lingering effect difference score (neutral word position #2 – emotional or category word position #1) served as the DV. Results indicated a significant main effect of emotion, $F(2, 58) = 20.93, p < .001$, which is indicative of differences in overall lingering effect across groups, such that negative emotion produced the greatest lingering effect, while category words and positive words failed to produce a lingering effect. A significant between-subjects effect was also found for group, $F(2, 58) = 3.23, p = .047$, which can be attributed to deficit syndrome patients displaying a greater lingering effect than non-deficit patients or controls across all word conditions.
The emotion X group interaction was nonsignificant, $F(4, 58) = 1.33, p = .248$.
This lack of significant differences is contrary to the hypothesis and indicates that
diagnostic groups all display similar lingering effect patterns, such that negative
emotions produced the greatest and positive emotions the least lingering effect
for each group. ANCOVA using IQ as a covariate produced a nonsignificant,
emotion X IQ interaction, $F(2, 58) = 1.51, p = .226$, indicating that IQ did not
influence effects reported for the lingering effect task.

Individual One-Way ANOVAs were conducted to examine the specific
sub-hypothesis that both deficit and non-deficit patients would evidence a
significantly greater lingering effect for negative emotions than controls, and to
determine whether differences are unique to negative emotions. Results
indicated a statistically significant difference among deficit, non-deficit, and
control groups with regard to the lingering effect resulting from negative words, $F$
$(2, 58) = 4.26, p = .02$, but not positive, $F(2, 58) = 1.63, p = .21$, or non-
emotional category words, $F(2, 58) = 0.84, p = .92$. Scheffé post hoc
comparisons clarified the nature of these differences by indicating that deficit
patients experience a significantly greater lingering effect than non-deficit
patients ($p = .02$), but not controls ($p = .23$); however, non-deficit patients did not
significantly differ from controls ($p = .48$), suggesting that the lingering effect
abnormality is specific to deficit syndrome patients. Figure 13 presents E-Stroop
lingering effect means and standard errors for patients and controls.
CHAPTER 5

DISCUSSION

Results are discussed in relation to three major aspects of emotional functioning: emotional experience, emotion perception, and cognitive processing of emotional information. Within these sections, emotion perception is divided into facial affect identification and olfactory perception, while the cognitive processing section is separated into attention and memory. These 5 separate sections correspond with the 5 hypotheses outlined in the literature review.

Emotional Experience

Prior research has provided evidence that patients with deficit syndrome schizophrenia have a diminished capacity to experience emotion. In comparison to non-deficit patients, those classified as having the deficit syndrome have been found to report lower levels of trait positive affect (Horan & Blanchard, 2003) and higher levels of physical anhedonia (Kirkpatrick & Buchanan, 1990; Herbener, Harrow, & Hill, 2005; Loas et al., 1996). They have also been reported to have less severe ratings on clinical scales measuring negative emotions, such as anxiety, guilt, and hostility (Kirkpatrick et al., 1993; Subotnik et al., 2000; Tek et al., 2001), depression/suicidality (Fenton et al., 1997; Kirkpatrick & Buchanan, 1990; Loas et al., 1996), and suspicion (Kirkpatrick et al., 1996). Experimental
paradigms also suggest that deficit patients experience lower levels of induced stress (Cohen & Docherty, 2004; Cohen et al., 2003; however, see Earnst & Kring, 1999). Together, these previous findings appear to suggest that deficit syndrome patients experience diminished levels of both positive and negative emotions, and are consistent with the original clinical conceptualization of the syndrome (Kirkpatrick et al., 1989).

However, findings from the current study provide mixed support for the idea that the deficit syndrome is associated with a diminished capacity to experience both positive and negative emotion, by suggesting that there is a greater diminishment for the experience of positive emotion alone. More specifically, when the frequency of self-reported emotional experience was compared across a range of positive and negative emotions, deficit syndrome patients reported experiencing significantly less frequent experience for the positive emotions of joy, interest, and surprise. In contrast, no differences were found among deficit, non-deficit, and control participants with regard to reported experience of discrete negative emotions. Additionally, when intensity of emotional experience was examined using the Positive and Negative Affect Scale (PANAS), data were also consistent with a diminished capacity to experience positive, but not negative emotion. These findings provide converging evidence which suggests that deficit syndrome patients report a diminished capacity to experience positive emotions, whereas the experience of negative emotion may be intact.

However, an important caveat of this finding is that the positive emotion impairment was specific to trait experience, as state affect was not diminished. In
fact, deficit, non-deficit, and control subjects did not significantly differ on state measures of either positive affect, as measured by the PANAS, and both patient groups reported experiencing higher levels of state negative affect than controls. Diminished capacity for trait positive, but not negative affect, is consistent with findings reported by Horan and Blanchard (2003) who also noted diminished trait positive but not negative affect in their deficit syndrome group. Additionally, similar to the current findings, Earnst and Kring, (1999) did not find differences in state positive affect between their deficit and non-deficits groups. However, results are contrary to findings of Cohen et al. (2005) who failed to find impaired experience of positive affect. Cohen et al. and Horan and Blanchard also found deficit patients to display heightened experience of negative emotion, as was found to occur in the current study. Contradictory findings regarding positive emotion may be due to differences in measurement, as previous studies used different self-report questionnaires than those administered in the current study. In this regard, findings from the current study were most consistent with those of Horan and Blanchard, who used a measure similar to the one employed that required participants to provide emotional intensity ratings across a variety of positive and negative affective states. Previous studies also did not examine differences in trait and state experience, using measures selective to one time frame or another. The current findings may therefore reflect differences in the time frame in which patients are asked to rate their subjective level of experience. Also, the current findings are consistent with a larger body of literature in patients with schizophrenia that documents association among
negative symptom and diminished positive but not negative emotions (Suslow et al., 2005). Thus, while not wholly consistent with prior studies of deficit syndrome schizophrenia, the present findings are interpreted as valid in that some of the inconsistency seems to be accounted for by methodological variation from one study to the next, and they are more generally consistent with the literature examining negative symptoms in schizophrenia.

While the cause of this impairment in positive emotional experience is not fully understood, deficit syndrome patients may report experiencing significantly less positive emotion when measured in trait, but not state form for several reasons. As previously suggested, one potential explanation for the trait vs. state discrepancy relates to differences in measurement. It is possible that interview-based clinical rating scales, self-report questionnaires, and experimental mood induction paradigms yield divergent findings due to measuring different components of emotional experience. Thus, conclusions made regarding the diminished capacity to experience positive emotion may be specific to self-report questionnaire measurement. It is possible that use of an alternative method, such as inner-experience sampling, or on-line assessment of hedonic capacity in relation to mood induction techniques (e.g., film clips, food, etc.) might produce different results. Future studies are needed that compare multiple measurement methods of emotional experience in deficit and non-deficit patient, considering that self-report questionnaires examined in the current study may require use of other cognitive factors in addition to experience in and of itself, such as appraisal or complex emotion knowledge, and that evidence for reduced positive emotional
experience may be better accounted for by these factors, and not a diminished capacity *per se*.

Another possibility is that deficit syndrome patients may have poor memory for previously experienced positive feelings or events. Recent findings by Horan et al. (2006) and Herbener et al. (2007) provide further insight into how emotional memory may play a role in diminished positive emotional experience. Horan et al. investigated the possibility that anhedonia in schizophrenia occurs due to poor encoding, retention, or retrieval of positive feelings using an experimental mood induction paradigm. Although patients reported significant levels of anhedonia, they did not have impairment in immediate or delayed recall for their feelings during mood induction. However, Herbener et al. (2007) utilized an experimental paradigm and found that patients with schizophrenia evidenced impaired memory for positive information after a 24 hour delay period, potentially reflecting an impaired ability to integrate positive experience in the memory consolidation process. These discrepant findings may reflect that patients with schizophrenia are able to adequately recall their general feelings, potentially because there is little variation in positive experience, yet fail to accurately recall positive information after a significant amount of time. Thus, there is some suggestion that anhedonia in schizophrenia may be associated with a failure to accurately recall positive information. The inability to recall positive information after substantial delay (e.g., 24 hours) may explain why patients with the deficit syndrome exhibit trait decreases in positive emotion. In other words, because they are unable to appropriately consolidate positive events in memory, deficit
patients may be less accurate at rating their emotional experience when asked to think back over the past few weeks, even though they have the capacity to experience positive emotions in the moment in a manner that is similar to that of healthy individuals. Alternatively, poor consolidation of positive information may render positive events unavailable in episodic memory, and thus cannot be recalled to create a positive perception or more general increase in positive mood, both of which are present in normal individuals.

Finally, it is also important to note that the specificity of trait impairment for positive emotion is consistent with the assertion that consummatory pleasure (i.e., pleasure derived from momentary experience; Depue & Iacono, 1989) is unimpaired in schizophrenia (Germans & Kring, 2000). Thus, although unable to express emotions, patients with schizophrenia, including those with the deficit syndrome, appear to be able to experience positive and negative emotions in the moment at a normal level.

Considering that frequent experience of positive emotion has been found to precede and cause a number of desirable functional outcomes (e.g., social, occupational, health) (Lyubomirsky, King, & Diener, 2005), it is likely that diminished positive emotional experience contributes at least in part to symptoms and functional impairments seen in deficit patients. For example, deficit patients may fail to develop interest in people and things or the drive to socialize, because they do not experience positive emotion frequently enough for these activities to become rewarding (or perhaps to be remembered as rewarding). Symptoms and functional impairments may therefore develop because of a lack of positive
emotional experience needed for behavioral activation, and for these behaviors to be reinforced. Additional studies are needed to directly determine whether the diminished capacity to experience positive emotion is a root cause of the negative symptoms seen in deficit syndrome patients, or whether positive emotion abnormalities are simply a result of severe negative symptoms. The extensive and comprehensive meta-analysis on positive affect conducted by Lyubomirsky, King, and Diener provides a basis for this work, and suggests that additional investigations of positive affect and negative symptoms would be worthwhile.

From a neurophysiological stand-point, abnormalities in experiencing positive emotion may be subsumed by dopaminergic dysfunction. Two previous studies have reported that deficit status has been linked to lower dopamine function (Ribeyre et al., 1994; Thibaut et al., 1994). Considering that higher dopamine levels are known to be key for higher levels of positive emotional experience (Ashby, Isen, & Turken, 1999), it is possible that the diminished capacity to experience positive emotion may be due to reduced dopamine function. Dampened response of the DA system in deficit patients could create deficiencies in the normal reward circuitry characterized by diminished DA release in the presence of positively reinforcing stimuli. Thus, normal associations between positive events and the internal experience of hedonic pleasure would not be formed, leading to a decreased inner experience of positive emotion, as well as a decreased drive to engage in pleasurable events. Additional studies using functional neuroimaging procedures could establish a
direct link between the experience of positive affect and dopaminergic
dysfunction in deficit and non-deficit patients.

Emotional Perception

Emotion perception was examined in relation to visual and olfactory
sensory domains. These results are discussed in facial affect identification and
olfactory perception sections, respectively.

Facial Affect Identification

Results of the facial affect identification procedure provided some support
for the hypothesis that deficit syndrome patients would display impairment in
processing positive facial expressions. In general, results indicated that deficit
syndrome patients were significantly less accurate and slower at processing
positive faces than controls. However, these impairments were not specific to
positive emotions, as deficit patients were also significantly less accurate than
controls at identifying negative emotions. More specifically, when differences
were analyzed in relation to individual emotions, results indicated that deficit
patients were significantly more impaired than non-deficit patients and controls
for surprise, fear, and neutral faces. The expected finding of decreased accuracy
on the part of the deficit syndrome groups for identification of happy faces was
not found. In examining the data for happy faces, it is apparent that ceiling
effects were present particularly for controls (% correct = 99), but also for the
non-deficit group (% correct = 89) and the deficit group (% correct = 87). So,
while the pattern of results was consistent with hypotheses, these ceiling effects
may have attenuated differences among the groups, differences which would have otherwise been present if the task difficulty level was greater. Thus, the mixed findings for positive emotions, surprise and joy, may be accounted for by this factor.

With regard to prior research, these findings are generally consistent with results reported by Bryson et al. (1998) who also found deficit syndrome patients to display accuracy impairments for surprise, and neutral. However, unlike Bryson et al., deficit syndrome patients in the current study were not significantly more impaired than non-deficit patients for displays of sadness. Our findings and those of Bryson et al. are also contrary to results reported by Horan and Blanchard (2003), who failed to find differences in facial affect identification between deficit and non-deficit patient groups. These discrepant findings may be due to differences in methodology and analytic strategy, as Horan and Blanchard did not examine differences in relation to discrete emotions which may have precluded the observation of noticeable differences by compounding variance across multiple emotional conditions. It has also been suggested that emotion recognition varies based on the manner in which the stimuli are presented (for a review see Edwards, Jackson, & Pattison, 2002). The method used here can most accurately be called a facial affect labeling task, which assesses controlled cognitive processes. Similar tasks have been shown to produce a pattern of results in patients with schizophrenia (not examining deficit and non-deficit distinctions) that suggest that negative emotions are most significantly impaired, particularly fear and sadness. The current findings suggest that negative
emotions are more significantly impaired for schizophrenia patients than positive emotions, and provide further evidence that fear and sadness may be the two facial affect displays that are most affected. Additionally, deficit syndrome patients do not appear to differ from non-deficit patients with regard to this pattern of impairment typically found with labeling tasks. However, they are impaired on displays of fear to a significantly greater extent than non-deficit patients, suggesting that the facial affect identification abnormalities typically seen in patients with schizophrenia are magnified in patients with primary negative symptoms.

Mixed support was also found for the hypothesis that deficit patients would be significantly slower than non-deficit patients and controls for positive faces. Similar to accuracy findings, deficit patients were significantly slower at processing positive faces (both happiness and surprise in the case of RT) than both non-deficit patients and controls; however, these findings were not specific to positive emotions, as deficit patients were slower than non-deficit patients, who were in turn significantly slower than controls, for all emotions examined. Thus, deficit patients are simply slower at processing faces, a finding that may reflect a generalized impairment in attention or processing speed. Although not supporting the expected distinction between reaction times for positive and negative affect, these findings provide the first data examining differences in RT for facial affect perception in patients with deficit and non-deficit schizophrenia.

Qualifications should be placed on facial affect findings reported. The task design used in the current study largely tapped into controlled cognitive
processes since facial affect stimuli remained on the computer screen for a maximum of 5 seconds or until participants provided a vocal response, whichever came first. It is likely that tasks utilizing a faster presentation, which would measure automatic processing, would result in different findings (see Edwards, Jackson, & Pattison, 2002 for a review of methodological considerations in face perception tasks). This is particularly likely considering that controlled and automatic emotion processing tasks activate different neural circuits. Additional studies could compare controlled and automatic facial affect processing tasks among deficit and non-deficit patients. Findings are also limited by the stimulus set used. Although widely used in emotion studies, the Matsumoto and Ekman (1988) stimulus set presents only intense facial displays of emotion, and does not offer variations in emotional intensity afforded by other stimulus sets. These intense facial displays undoubtedly accounted for some of the ceiling effects noted for happiness. Future studies could examine the possibility of differences among high and low intensity emotional faces using other standardized facial affect stimulus sets.

**Olfactory Perception**

Consistent with hypotheses, results indicate that deficit syndrome patients evidenced significantly poorer olfactory identification performance than non-deficit patients and controls. These findings are in-line with several studies indicating decreased olfactory identification in deficit syndrome patients (Goudsmit et al., 2003; Malaspina & Coleman, 2003; Malaspina et al., 2002; Moberg et al., 2006; Seckinger et al., 2003), and provide additional support for
the use of the Brief-Smell Identification Test (B-SIT) as a measure capable of differentiating deficit and non-deficit schizophrenia (Goudsmit et al., 2004).

Results extend previous studies of olfaction in deficit syndrome patients by separating out individual B-SIT items to examine impairments in accurately identifying and judging the valence of pleasant and unpleasant odors. Relative to non-deficit patients and controls, deficit patients were found to identify both positive and negative items with less accuracy. This finding of diminished accuracy for both odor categories was specific to deficit patients, as non-deficit patients and controls did not differ in relation to identifying either pleasant or unpleasant items. Thus, data did not support the hypothesis of a differential impairment in identifying pleasant items among deficit syndrome patients.

However, an odor specific impairment was found when individual items were examined in relation to valence judgment. When asked to rate individual B-SIT items for pleasantness on a 7-point scale, deficit syndrome patients were uniquely found to rate pleasant items as significantly more neutral than controls. No differences were found with regard to judging negative smells, as deficit, non-deficit patients and controls were all found to judge unpleasant smells as being similarly negative. This finding of a hedonic impairment for judging pleasantness is consistent with previous studies examining olfactory hedonics in patients with schizophrenia (Crespo-Facorro et al., 2001; Doop & Park, 2006; Hurdy et al., 2002), and provides the first evidence suggesting that valence judgment may be more impaired in deficit syndrome patients. It is also consistent with our finding of decreased trait positive affect, particularly as it relates to a diminished experience
of positive events being experienced as pleasurable, possibly due to a dampening of DA response and a dysfunctional reward circuit.

When evidence for impaired judgment of pleasantness is viewed in relation to previous work, results also provide insight into the neural mechanisms underlying deficit syndrome classification. Research by Crespo-Facorro et al. (2001) examined neural response to pleasant and unpleasant smells using PET, to determine whether patients with schizophrenia displayed impairment in rating intensity and valence of odors. No differences were found in judging valence of unpleasant odors; however, pleasant smells were rated by patients as being less pleasant than healthy controls. PET data indicated that the judgment of unpleasant smells was associated with less activation in limbic/paralimbic regions and increased frontal cortical activation. Hurdy et al. (2002) also found patients with schizophrenia to display reduced pleasantness ratings, in addition to impaired familiarity and edibility judgments. These impairments were thought to be subsumed by temporolimbic and orbitofrontal dysfunction. When the current findings are viewed in relation to results reported by previous studies, results are consistent with dysfunctional limbic and frontal system contribution to olfactory performance in schizophrenia, and suggest that frontal and limbic circuitry may be particularly related to the neuropathology inherent to the deficit syndrome.
Cognitive Processing of Emotion

Memory

The current study provides the first data examining emotional memory impairment in patients with deficit syndrome schizophrenia. Results provide some support for the hypothesis that deficit syndrome patients would display a memory impairment for positive information. In comparison to non-deficit patients and controls, deficit patients recalled significantly fewer happiness words; however, recall impairments were not specific to happiness, as deficit patients evidenced poorer performance for sadness and anxiety as well. Although memory impairment was not selective to positive information, deficit patients failed to display the Pollyanna effect (i.e., superior recall for happiness) displayed by both non-deficit patients and healthy controls. Rather, deficit syndrome patients showed an equal recall bias for happiness and anger words— a pattern not seen in other groups.

In addition to providing the first data on deficit syndrome patients, the current study also provides the first comparison of verbal emotional memory for multiple discrete emotions. Early studies on emotional memory in schizophrenia indicated that patients fail to exhibit the Pollyanna effect (Matlin & Stang, 1978) typically found in healthy individuals, both in relation to verbal (Koh, Grinker, Marusarz, & Forman, 1981) and visual/facial (Koh et al., 1981) information. Koh et al. (1981) also found that patients recognized a significantly greater number of negative than positive faces, potentially signifying that patients have a bias toward recognizing negative information, a finding supported by Calev and
Edelist (1996) who reported that patients recalled a greater number of negative than positive words and forgot negative words less rapidly after a 2 day delay period. Results of the current study are generally consistent with previous investigations, as deficit, but not non-deficit patients, failed to display a Pollyanna effect, as well as a bias toward recalling anger words at a high rate. Additionally, both patient groups recalled significantly less emotional information than healthy controls, a finding consistent with the majority of previous studies (see Matthews et al. 2005 for contradictory results). The finding of a lack of Pollyanna effect may be specific to deficit syndrome patients or more general to those patients who experience high levels of negative symptoms. Whatever the case, dividing patients into deficit and non-deficit groups may have allowed the current findings to emerge, something that was not accomplished in prior studies.

Our findings extend previous work by providing evidence that deficit syndrome patients display even more severe emotional memory impairments than non-deficit patients. Interestingly, deficit syndrome patients also failed to show superior recall for non-emotional words, as was found in both non-deficit patients and healthy controls. This finding may reflect that emotional memory impairments seen in deficit patients are due to generally poor memory performance, or perhaps a failure to use learning strategies when recalling verbal material. Alternatively, this difference may suggest that the emotional content of the words examined were not as salient for the deficit group, and thus do not produce the expected increase in difficulty level in these patients. In other words, deficit syndrome patients may process emotional information in a manner similar
to neutral information, which is consistent with disturbance in affective memory. Additional studies are needed to determine why deficit patients display equivalent recall for emotional and nonemotional information, and to extend findings to other aspects of memory using multiple sensory domains.

Attention

Results indicate that deficit syndrome schizophrenia is associated with a unique pattern of attentional impairments not seen in non-deficit patients. Consistent with hypotheses, positive information captured the attention of deficit syndrome patients significantly less than non-deficit patients or controls. This was indicated by deficit syndrome patients receiving a significantly lower difference score (happiness – neutral) than non-deficit patients and controls for high-intensity happiness words on the attention grabbing task. In fact, deficit patients did not only evidenced markedly less attentional capture (i.e., interference) toward happiness, but they displayed less capture for happiness words than for neutral words (i.e., a negative difference score). Importantly, this pattern was unique to the deficit syndrome, as non-deficit patients and controls displayed an attention bias for happiness (as indicated by a positive difference score).

This lack of an attention bias for happiness, which our data suggest is unique to deficit syndrome patients in this study, may be explained in several ways. First, happiness may either be inconsistent with the mood state of deficit patients at the time of testing or unrelated to their general cognitive preoccupations. Considering that our sample of deficit patients had high symptom ratings for
measures of anhedonia and avolition (e.g., curbed interests, diminished social drive), this explanation seems plausible. Correlational findings provide further support for the role of mood as there was a trend level positive correlation between state positive affect and attention bias for happiness, indicating that low state positive affect is associated with less attention bias for happiness in schizophrenia patients. This finding may suggest that deficit syndrome patients display a mood-congruent processing abnormality related to happiness, such that diminished capacity to experience positive affect is associated with less of a tendency to have attention drawn toward positive information. Thus, findings suggest that deficit patients exhibit a failure to attend to positive information at the automatic level of cognitive processing, and that this impairment is related to core symptoms of the disorder.

Results also indicated that patients with schizophrenia and controls showed an attention bias toward negatively valenced material, as has been commonly found in healthy individuals (McKenna & Sharma, 2005), other patient groups (see Williams, Matthews, & McLeod, 1996), and patients with schizophrenia (Epstein et al., 1999) or delusional disorder (Fear et al., 1996). Our findings extend previous work in the area by examining attention bias in relation to discrete emotions. When attention bias is compared across multiple emotions, results indicate that emotional information of all types grabbed the attention of non-deficit patients to a greater extent than controls. This may be due to patients with schizophrenia having less cognitive resources than healthy individuals, and particular impairment in executive functioning, which is required to successfully
perform the E-Stroop task. Non-deficit patients displayed the greatest attention bias for words representing anxiety and sadness, two emotions which are most consistent with their clinical presentation. This pattern differed in deficit syndrome patients, who showed an attention bias for anxiety, but not sadness. Considering that deficit syndrome patients in our sample were lower than non-deficit patients on symptoms of depression, this lack of attention bias for sadness may represent yet another mood congruent processing impairment. Thus, it is possible that the reduced risk for depression and suicide seen in deficit patients may be maintained by a failure to have attention captured by sad information. Another major finding was that deficit patients displayed a significantly greater lingering effect for negative information than non-deficit patients, as indicated by a positive interference score (neutral word position 2 – negative word position 1) on the E-Stroop lingering effect task. Within our schizophrenia sample, the lingering effect was specific to deficit syndrome patients, as non-deficit patients failed to display a lingering effect. This lingering effect for negative emotion seen in the deficit patients suggests that once negative information captures attention, it continues to disrupt ongoing cognition, even when negative stimuli are no longer present.

Several factors may explain this lingering effect finding. Meta-analytic and empirical findings reported by Cohen et al. (2006) suggest that deficit syndrome patients display markedly reduced cognitive function in relation to non-deficit patients and controls, and indicate that these impairments may reflect a generalized abnormality (i.e., no selective neuroanatomical substrates). It is
possible that the lingering effect seen in deficit syndrome patients results from a generally reduced cognitive capacity. They may be less able to disengage attentional focus once it has been captured by a salient negative stimulus, and have a greater propensity to perseverate on the negative information that initially grabbed their attention. Further studies are needed to determine whether deficit syndrome patients do in fact perseverate on negative information once it grabs their attention, or whether the lingering effect findings reported here may be better accounted for by other cognitive processes, such as slower processing speed.

Alternatively, the lingering effect seen in deficit patients may reflect either hypo or hyper limbic system activation. In healthy individuals, it has been documented that once negative information captures attention, it activates specific limbic system structures, particularly the amygdala. Deficit syndrome patients may have abnormalities in one of the two major amygdala activation systems that are associated with negative emotions. However, given that negative stimuli have been reported to produce both hyper and hypo-active amygdala activation in patients with schizophrenia, neuroimaging studies are needed to identify the precise nature of limbic system dysregulation, and to determine whether attentional abnormalities noted in deficit syndrome patients are associated with amygdala dysfunction.

These findings shed light onto how emotional information processing impairments contribute to negative symptoms of schizophrenia, and suggest that the failure to have attention captured by positive information may be core to the
volitional symptoms characteristic of deficit syndrome schizophrenia, whereas affective symptoms are associated with a failure to maintain a flexible attentional system capable of diverting attention away from negative information and toward goal-directed behavior.

General Limitations

The current study has several general limitations that affect the interpretation and generalizability of findings. First, although the total sample size of schizophrenia patients is adequate \((n = 42)\), the division of patients into deficit \((n = 15)\) and non-deficit \((n = 26)\) groups reduces power. Replication of findings is needed to ensure that results generalize to the population of schizophrenia patients as a whole. Second, a large number of analyses were conducted to analyze differences in emotion processing across a number of cognitive domains. The number of analyses conducted raises concerns with inflated Type I error. It is therefore possible that some differences found among patient groups fail to represent true differences on variables assessed, suggesting further need for replication of these results. Third, conclusions drawn regarding the role of dopamine in producing diminished positive emotional experience in deficit syndrome patients is speculative. Although these conjectures are based on research conducted on healthy individuals showing that higher levels of positive emotional experience are associated with higher dopamine levels, dopamine was not directly assessed in the current study and it is therefore impossible to know whether lower dopamine levels contribute to affective disturbances found.
Additionally, given that the majority of patients were stably medicated with antipsychotics, it is impossible to determine how medication effects influenced dopamine levels within our sample of deficit patients. Future studies could address this issue by studying medication naïve first episode psychotic patients with and without deficit syndrome schizophrenia.

Summary and Conclusions

The purpose of the present study was to evaluate the hypothesis that deficit syndrome schizophrenia is associated with a unique pattern of affective disturbance. The majority of previous work examining emotional abnormalities in deficit syndrome patients has focused on understanding affective experience. Findings from several studies appear consistent with the notion that deficit syndrome patients experience diminished levels of both positive and negative emotions (i.e., there is not a differential affective experience impairment). Our findings provide mixed support for this idea. Similar to previous studies, our deficit patients reported higher levels of anhedonia as measured through clinical interview, received less severe clinical ratings of anxiety, guilt, and hostility, and reported less depression, suicidality, and suspicion than non-deficit patients. However, we did not find evidence for generalized diminished emotional experience, as was originally conceptualized by Kirkpatrick et al. (1989). Rather, findings point to a selective impairment in experiencing positive, but not negative emotion, which is specific to trait measures of emotional experience. Considering that frequent experience of positive affect has been found to precede and cause
a number of desirable functional outcomes (Lyubomirsky et al., 2005), which are
deficient in deficit syndrome patients, it is possible that diminished positive
emotional experience is at the core of primary negative symptoms in
schizophrenia. Additionally, considering that deficit syndrome patients have been
reported to exhibit decreased dopamine levels, and higher dopamine levels have
been linked to both reward and the experience of positive emotion (Ashby, Isen,
& Turken, 1999), it is possible that dopaminergic system dysfunction may
underlie both clinical negative symptoms and the diminished capacity to
experience positive affect.

Relatively fewer studies have examined emotional information processing in
patients with deficit syndrome schizophrenia. Although previous findings have
been mixed (Horan & Blanchard, 2003), there is some evidence that deficit
syndrome patients display poorer facial affect identification performance than
non-deficit patients, particularly for the emotions of surprise, fear, disgust,
sadness, and neutral (Bryson et al., 1998). Our findings provide support for the
view that deficit patients display abnormalities in emotion perception, as deficit
patients evidenced decreased accuracy for several facial affect displays,
including fear, surprise, and neutral. Deficit patients also processed emotional
faces more slowly than non-deficit patients; however, it is likely that these delays
are due to a generalized processing speed impairment, rather than an emotion
specific abnormality.

Emotion perception abnormalities were also found in relation to olfactory
identification. Similar to previous studies indicating that smell identification
deficits may represent one of the most stable neurocognitive impairments in 
deficit syndrome schizophrenia, findings indicate that deficit patients were 
significantly poorer than non-deficit patients at identifying common odors. When 
these olfactory performance impairments were analyzed in relation to pleasant 
and unpleasant affective conditions, deficit syndrome patients displayed a unique 
tendency to rate pleasant smells as significantly more neutral than controls. 
Differences were not found in relation to judging the valence of negative smells, 
suggesting that these perceptual abnormalities may be unique to positive 
emotion.

A similar pattern of impairment for positive information was found in relation to 
attention and memory. In comparison to non-deficit patients and controls, deficit 
patients displayed a unique failure to have attention captured by positive 
information. Correlational analyses indicated that less attention bias for 
happiness was associated with greater state ratings of diminished positive affect, 
potentially implicating a mood-congruent processing abnormality. Deficit patients 
were also found to display a significantly greater lingering effect for negative 
information than non-deficit patients or controls, suggesting that once negative 
information captures their attention, it continues to disrupt on-going cognitive 
processes, even when the negative stimulus is no longer present. Together, 
these attention findings reflect that deficit syndrome patients display impairment 
at the automatic level of emotion processing, such that they are less drawn 
toward positive information and find it more difficult to disengage from negative 
information once it captures their attention. Such difficulties may reflect a failure
to maintain a flexible attentional system that is capable of shifting between rewarding and threatening information in response to environmental contingencies, which is essential for adaptive functioning.

Deficit syndrome patients also displayed significant memory impairment for emotional information. Relative to non-deficit patients and controls, deficit patients displayed markedly reduced emotional memory and learning. However, these differences may reflect a generalized neurocognitive impairment, as deficit patients were also significantly more impaired on recall for non-emotional information and failed to exhibit beneficial recall for non-emotional information as found in non-deficit patients and controls. As hypothesized, deficit patients also failed to display the Pollyanna effect of superior recall for positive information that has been consistently found in healthy individuals (Matlin & Stang, 1978). Similar to attentional findings, this positive emotion memory impairment may reflect that deficit syndrome patients evidence a mood-congruent memory impairment, considering that they reported higher levels of anhedonia and trait positive affect.

When findings from the multiple emotion domains examined are viewed together, results provide some indication that deficit syndrome patients evidence more severe affective disturbance in relation to positive emotion. In comparison to non-deficit patients and controls, deficit patients reported less frequent and intense experience of positive emotion, recalled significantly fewer positive words, and displayed an impaired ability to accurately identify and judge the valence of pleasant odors. Additionally, deficit patients demonstrated a unique failure to have their attention captured by positive information, as well as less
accurate and efficient labeling of positive faces (surprise) than non-deficit patients or controls. Thus, results provide support for the notion that the deficit syndrome is uniquely associated with abnormalities in the experience and processing of positive emotion.

However, affective abnormalities were not entirely specific to positive emotion, and it should be noted that deficit syndrome patients displayed cognitive and perceptual disturbances for negative emotions as well. For example, deficit patients were significantly more impaired than non-deficit patients at identifying fear faces, and displayed a lingering effect in attention for negative words that was not seen in non-deficit patients. They also exhibited a bias toward recalling anger words at a high rate, which was not seen in non-deficit patients, and impairment on accurately identifying, negative, as well as positive, odors. Thus, although deficit syndrome patients display a more consistent pattern of impairment toward positive emotion, which is unique to their clinical presentation, they also evidence impairment in relation to the processing of some negative information.

In conclusion, the present findings provide support for the suggestion that deficit syndrome schizophrenia is a disorder characterized by impairments in emotional experience, perception, and information processing. Furthermore, this abnormality is not global in nature, but is characterized most notably by deficits that are specific to positive emotions. Such findings are consistent with characteristic functional deficits in these patients, as well as neurophysiological findings implicating dysfunction within dopaminergic and reward-system circuits.
Additional studies are needed to determine the extent to which emotional abnormalities reported in the current study are due to generalized cognitive impairment and to identify the specific neural circuits that contribute to this dysfunction. Using a matched task for our measure of emotional memory, the CVLT, results provide some suggestion that general emotional memory impairments displayed by deficit syndrome patients may be due to global recall difficulties. Paradigms using matched emotional and non-emotional tasks could determine the extent to which emotional abnormalities are caused by general neurocognitive impairment. Our findings also point to some potential neuroanatomical substrates for the emotion dysfunction seen in deficit patients when findings are viewed in relation to previous work on schizophrenia, most notably dysfunctional limbic and frontal circuitry. Future neuroimaging studies could further explore the potential contributions of these circuits to deficit syndrome symptomatology.
APPENDIX A

TABLES
Table 1.

*Patient and Control Demographic Characteristics.*

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<th>Controls (n = 22)</th>
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<td>% Asian</td>
<td>00.0%</td>
<td>07.7%</td>
<td>04.5%</td>
</tr>
<tr>
<td>% Biracial</td>
<td>13.3%</td>
<td>03.8%</td>
<td>13.6%</td>
</tr>
<tr>
<td>% Caucasian</td>
<td>26.7%</td>
<td>53.8%</td>
<td>68.2%</td>
</tr>
<tr>
<td>% Hispanic/Latino</td>
<td>00.0%</td>
<td>07.7%</td>
<td>09.1%</td>
</tr>
</tbody>
</table>
Table 2.

*Patient and Control Social Demographic Characteristics.*

<table>
<thead>
<tr>
<th></th>
<th>Deficit (n = 15)</th>
<th>Non-Deficit (n = 26)</th>
<th>Control (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Homelessness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Never</td>
<td>66.70%</td>
<td>57.70%</td>
<td>100.0%</td>
</tr>
<tr>
<td>% Lifetime</td>
<td>33.30%</td>
<td>42.30%</td>
<td>00.00%</td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Divorced</td>
<td>00.00%</td>
<td>11.50%</td>
<td>09.10%</td>
</tr>
<tr>
<td>% Married</td>
<td>00.00%</td>
<td>00.00%</td>
<td>77.30%</td>
</tr>
<tr>
<td>% Never</td>
<td>93.30%</td>
<td>80.80%</td>
<td>09.10%</td>
</tr>
<tr>
<td><strong>Married</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Separated</td>
<td>00.00%</td>
<td>03.80%</td>
<td>04.50%</td>
</tr>
<tr>
<td>% Widowed</td>
<td>06.70%</td>
<td>00.00%</td>
<td>00.00%</td>
</tr>
</tbody>
</table>
Table 3.

*Patient and Control Smoking Characteristics.*

<table>
<thead>
<tr>
<th></th>
<th>Deficit (n = 15)</th>
<th>Non-Deficit (n = 26)</th>
<th>Control (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Who Currently Smoke</td>
<td>73.30%</td>
<td>46.20%</td>
<td>40.90%</td>
</tr>
<tr>
<td># Cigarettes Smoked per Day</td>
<td>16.82 (11.46)</td>
<td>16.55 (5.13)</td>
<td>10.11 (4.76)</td>
</tr>
</tbody>
</table>
Table 4.

Descriptive Statistics for Patient Medication use, Clinical Variables, and Extrapyramidal Symptoms.

<table>
<thead>
<tr>
<th></th>
<th>Deficit</th>
<th>Non-Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine Equivalent Dosage (^a)</td>
<td>861.4</td>
<td>705.2</td>
</tr>
<tr>
<td>Depakote Dosage (^b)</td>
<td>450.0</td>
<td>480.0</td>
</tr>
<tr>
<td>Mood Stabilizer Dosage (^c)</td>
<td>74.3</td>
<td>83.3</td>
</tr>
<tr>
<td>% Prescribed Medication at Testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clozaril</td>
<td>33.3%</td>
<td>15.4%</td>
</tr>
<tr>
<td>Halodol</td>
<td>13.3%</td>
<td>11.5%</td>
</tr>
<tr>
<td>Risperdal</td>
<td>46.7%</td>
<td>34.6%</td>
</tr>
<tr>
<td>Zyprexa</td>
<td>40.0%</td>
<td>38.5%</td>
</tr>
<tr>
<td>Seroquel</td>
<td>20.0%</td>
<td>19.2%</td>
</tr>
<tr>
<td>Geodon</td>
<td>13.3%</td>
<td>26.9%</td>
</tr>
<tr>
<td>Abilify</td>
<td>33.3%</td>
<td>19.2%</td>
</tr>
<tr>
<td>Depakote</td>
<td>46.7%</td>
<td>42.3%</td>
</tr>
<tr>
<td>Mood Stabilizer</td>
<td>46.7%</td>
<td>46.2%</td>
</tr>
<tr>
<td>Anti-Parkinsonian</td>
<td>06.7%</td>
<td>03.8%</td>
</tr>
<tr>
<td>EPS Medication</td>
<td>33.3%</td>
<td>19.2%</td>
</tr>
<tr>
<td>Extrapyramidal Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rockland Rating Scale- Total (^d)</td>
<td>18.1</td>
<td>21.1</td>
</tr>
<tr>
<td>AIMS - Total (^e)</td>
<td>7.5</td>
<td>6.5</td>
</tr>
<tr>
<td>EPS Scale- Total (^f)</td>
<td>11.5</td>
<td>9.0</td>
</tr>
<tr>
<td>Clinical Variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotic Onset (^g)</td>
<td>17.3</td>
<td>15.1</td>
</tr>
<tr>
<td>Illness Length (^h)</td>
<td>28.5</td>
<td>24.5</td>
</tr>
</tbody>
</table>

\(^a\) F (1.39) = 0.63, p = .43  
\(^b\) F (1.39) = 0.03, p = .87  
\(^c\) F (1.39) = 0.04, p = .85  
\(^d\) F (1.39) = 1.89, p = .18  
\(^e\) F (1.39) = 0.20, p = .66  
\(^f\) F (1.39) = 1.69, p = .20  
\(^g\) F (1.39) = 0.59, p = .45  
\(^h\) F (1.39) = 0.73, p = .40
Table 5.

*Brief Psychiatric Rating Scale Clinical Symptom Characteristics.*

<table>
<thead>
<tr>
<th>Individual Items</th>
<th>Deficit (n = 15)</th>
<th>Non-Deficit (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Somatic Concern</td>
<td>02.5 (01.1)</td>
<td>02.4 (01.6)</td>
</tr>
<tr>
<td>2. Anxiety</td>
<td>01.7 (01.0)</td>
<td>02.7 (01.4)</td>
</tr>
<tr>
<td>3. Emotional Withdrawal</td>
<td>04.0 (01.9)</td>
<td>02.5 (01.6)</td>
</tr>
<tr>
<td>4. Conceptual Disorganization</td>
<td>01.9 (01.4)</td>
<td>02.5 (01.9)</td>
</tr>
<tr>
<td>5. Guilt Feelings</td>
<td>01.1 (00.5)</td>
<td>02.2 (01.3)</td>
</tr>
<tr>
<td>6. Tension</td>
<td>02.1 (01.5)</td>
<td>01.9 (01.3)</td>
</tr>
<tr>
<td>7. Mannerisms and Posturing</td>
<td>02.4 (01.8)</td>
<td>02.0 (01.2)</td>
</tr>
<tr>
<td>8. Grandiosity</td>
<td>01.5 (01.1)</td>
<td>02.2 (01.6)</td>
</tr>
<tr>
<td>9. Depressive Mood</td>
<td>01.1 (00.3)</td>
<td>01.8 (01.1)</td>
</tr>
<tr>
<td>10. Hostility</td>
<td>01.3 (00.6)</td>
<td>01.9 (01.2)</td>
</tr>
<tr>
<td>11. Suspiciousness</td>
<td>01.8 (01.2)</td>
<td>03.2 (01.8)</td>
</tr>
<tr>
<td>12. Hallucinatory Behavior</td>
<td>02.8 (01.7)</td>
<td>03.5 (01.8)</td>
</tr>
<tr>
<td>13. Motor Retardation</td>
<td>02.8 (01.6)</td>
<td>01.9 (01.4)</td>
</tr>
<tr>
<td>14. Uncooperativeness</td>
<td>02.3 (01.6)</td>
<td>01.4 (01.0)</td>
</tr>
<tr>
<td>15. Unusual Thought Content</td>
<td>03.5 (01.3)</td>
<td>03.4 (01.7)</td>
</tr>
<tr>
<td>16. Blunted Affect</td>
<td>05.1 (01.6)</td>
<td>03.0 (01.9)</td>
</tr>
<tr>
<td>17. Excitement</td>
<td>01.1 (00.5)</td>
<td>01.4 (00.8)</td>
</tr>
<tr>
<td>18. Disorientation</td>
<td>01.7 (01.0)</td>
<td>01.4 (00.8)</td>
</tr>
<tr>
<td>BPRS Positive Syndrome</td>
<td>07.6 (02.5)</td>
<td>09.5 (03.7)</td>
</tr>
<tr>
<td>BPRS Negative Syndrome</td>
<td>12.0 (04.1)</td>
<td>07.3 (03.6)</td>
</tr>
<tr>
<td>BPRS Disorganized Syndrome</td>
<td>07.3 (02.5)</td>
<td>07.5 (03.3)</td>
</tr>
<tr>
<td>BPRS Total</td>
<td>40.9 (06.3)</td>
<td>41.0 (08.4)</td>
</tr>
</tbody>
</table>
Table 6.

*Patient Clinical Characteristics for Depression*

<table>
<thead>
<tr>
<th>Individual Items</th>
<th>Deficit (n = 15)</th>
<th>NonDeficit (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Depression</td>
<td>0.13 (0.35)</td>
<td>0.27 (0.45)</td>
</tr>
<tr>
<td>2. Hopelessness</td>
<td>0.13 (0.35)</td>
<td>0.27 (0.45)</td>
</tr>
<tr>
<td>3. Self Deprivation</td>
<td>0.20 (0.78)</td>
<td>0.35 (0.69)</td>
</tr>
<tr>
<td>4. Guilty Ideas of Reference</td>
<td>0.13 (0.35)</td>
<td>0.27 (0.60)</td>
</tr>
<tr>
<td>5. Pathological Guilt</td>
<td>0.13 (0.35)</td>
<td>0.38 (0.64)</td>
</tr>
<tr>
<td>6. Morning Depression</td>
<td>0.00 (0.00)</td>
<td>0.23 (0.59)</td>
</tr>
<tr>
<td>7. Early Wakening</td>
<td>0.13 (0.52)</td>
<td>0.58 (0.99)</td>
</tr>
<tr>
<td>8. Suicide</td>
<td>0.00 (0.00)</td>
<td>0.27 (0.32)</td>
</tr>
<tr>
<td>9. Observed Depression</td>
<td>0.07 (0.26)</td>
<td>0.12 (0.33)</td>
</tr>
<tr>
<td>Calgary Depression Scale Totalª</td>
<td>0.93 (1.34)</td>
<td>2.73 (3.45)</td>
</tr>
</tbody>
</table>

ªF(1, 39) = 3.70, p = .062
Table 7.

Scale for the Assessment of Positive Symptoms Clinical Symptom Characteristics.

<table>
<thead>
<tr>
<th>Individual Items</th>
<th>Deficit (n = 15)</th>
<th>Non-Deficit (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Auditory Hallucinations</td>
<td>1.47 (1.69)</td>
<td>2.35 (1.67)</td>
</tr>
<tr>
<td>2. Voices Commenting</td>
<td>0.47 (1.25)</td>
<td>0.31 (0.93)</td>
</tr>
<tr>
<td>3. Voices Conversing</td>
<td>0.20 (0.78)</td>
<td>0.69 (1.29)</td>
</tr>
<tr>
<td>4. Somatic or Tactile Hallucinations</td>
<td>0.53 (1.19)</td>
<td>0.50 (0.95)</td>
</tr>
<tr>
<td>5. Olfactory Hallucinations</td>
<td>0.00 (0.00)</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>6. Visual Hallucinations</td>
<td>0.47 (0.99)</td>
<td>0.35 (0.80)</td>
</tr>
<tr>
<td>7. Global Rating of Hallucinations</td>
<td>1.80 (1.61)</td>
<td>2.31 (1.64)</td>
</tr>
<tr>
<td>8. Persecutory Delusions</td>
<td>1.27 (1.39)</td>
<td>2.27 (1.51)</td>
</tr>
<tr>
<td>9. Delusions of Jealousy</td>
<td>0.13 (0.52)</td>
<td>0.19 (0.63)</td>
</tr>
<tr>
<td>10. Delusions of Guilt or Sin</td>
<td>0.13 (0.52)</td>
<td>0.81 (1.06)</td>
</tr>
<tr>
<td>11. Grandiose Delusions</td>
<td>0.47 (0.99)</td>
<td>1.12 (1.48)</td>
</tr>
<tr>
<td>12. Religious Delusions</td>
<td>0.40 (1.12)</td>
<td>0.81 (1.42)</td>
</tr>
<tr>
<td>13. Somatic Delusions</td>
<td>0.73 (1.28)</td>
<td>1.00 (1.50)</td>
</tr>
<tr>
<td>14. Delusions of Reference</td>
<td>0.93 (1.34)</td>
<td>1.46 (1.50)</td>
</tr>
<tr>
<td>15. Delusions of Being Controlled</td>
<td>0.13 (0.52)</td>
<td>0.31 (0.88)</td>
</tr>
<tr>
<td>16. Delusions of Mind Reading</td>
<td>0.53 (1.13)</td>
<td>0.50 (1.30)</td>
</tr>
<tr>
<td>17. Thought Broadcasting</td>
<td>0.47 (1.00)</td>
<td>0.62 (1.20)</td>
</tr>
<tr>
<td>18. Thought Insertion</td>
<td>0.07 (0.26)</td>
<td>0.31 (0.88)</td>
</tr>
<tr>
<td>19. Thought Withdrawal</td>
<td>0.20 (0.56)</td>
<td>0.12 (0.59)</td>
</tr>
<tr>
<td>20. Global Rating of Delusions</td>
<td>2.60 (0.99)</td>
<td>2.88 (1.14)</td>
</tr>
<tr>
<td>21. Clothing and Appearance</td>
<td>1.67 (1.59)</td>
<td>0.62 (1.06)</td>
</tr>
<tr>
<td>22. Social and Sexual Behavior</td>
<td>0.27 (0.70)</td>
<td>0.62 (1.02)</td>
</tr>
<tr>
<td>23. Aggressive and Agitated Behavior</td>
<td>0.27 (0.70)</td>
<td>0.92 (1.26)</td>
</tr>
<tr>
<td>24. Repetitive or Stereotyped Behavior</td>
<td>0.40 (0.91)</td>
<td>0.35 (0.89)</td>
</tr>
<tr>
<td>25. Global rating of Bizarre Behavior</td>
<td>1.40 (1.18)</td>
<td>1.42 (1.23)</td>
</tr>
<tr>
<td>26. Derailment</td>
<td>0.87 (1.36)</td>
<td>1.23 (1.80)</td>
</tr>
<tr>
<td>27. Tangentiality</td>
<td>0.93 (1.22)</td>
<td>1.54 (1.92)</td>
</tr>
<tr>
<td>28. Incoherence</td>
<td>0.40 (0.91)</td>
<td>0.23 (0.86)</td>
</tr>
<tr>
<td>29. Illogicality</td>
<td>0.13 (0.52)</td>
<td>1.12 (1.51)</td>
</tr>
<tr>
<td>30. Circumstantiality</td>
<td>0.53 (0.92)</td>
<td>1.12 (1.68)</td>
</tr>
<tr>
<td>31. Pressure of Speech</td>
<td>0.00 (0.00)</td>
<td>0.23 (0.82)</td>
</tr>
<tr>
<td>32. Distractible Speech</td>
<td>0.87 (1.46)</td>
<td>0.85 (1.52)</td>
</tr>
<tr>
<td>33. Clanging</td>
<td>0.00 (0.00)</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>34. Global Rating of Formal Thought Disorder</td>
<td>1.20 (1.08)</td>
<td>1.88 (1.75)</td>
</tr>
<tr>
<td>SAPS Total</td>
<td>21.93</td>
<td>31.00 (14.04)</td>
</tr>
<tr>
<td></td>
<td>(11.34)</td>
<td></td>
</tr>
</tbody>
</table>
Table 8.

Scale for the Assessment of Negative Symptoms Clinical Symptom Characteristics.

<table>
<thead>
<tr>
<th>Individual Items</th>
<th>Deficit (n = 15)</th>
<th>Non-Deficit (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Unchanging Facial Expression</td>
<td>3.40 (1.18)</td>
<td>2.00 (1.63)</td>
</tr>
<tr>
<td>2. Decreased Spontaneous Movements</td>
<td>2.40 (1.68)</td>
<td>1.12 (1.51)</td>
</tr>
<tr>
<td>3. Paucity of Expressive Gestures</td>
<td>3.13 (1.25)</td>
<td>1.85 (1.59)</td>
</tr>
<tr>
<td>4. Poor Eye Contact</td>
<td>2.53 (1.19)</td>
<td>1.19 (1.32)</td>
</tr>
<tr>
<td>5. Affective Nonresponsivity</td>
<td>2.67 (1.88)</td>
<td>1.00 (1.47)</td>
</tr>
<tr>
<td>6. Inappropriate Affect</td>
<td>0.53 (1.41)</td>
<td>0.62 (1.17)</td>
</tr>
<tr>
<td>7. Lack of Vocal Inflections</td>
<td>2.73 (1.39)</td>
<td>1.65 (1.62)</td>
</tr>
<tr>
<td>8. Subjective Complaints of Emotional</td>
<td>2.53 (2.00)</td>
<td>0.58 (1.30)</td>
</tr>
<tr>
<td>9. Global Rating of Affective Flattening</td>
<td>3.67 (0.72)</td>
<td>2.19 (1.60)</td>
</tr>
<tr>
<td>10. Poverty of Speech</td>
<td>1.73 (1.58)</td>
<td>0.54 (0.95)</td>
</tr>
<tr>
<td>11. Poverty of Content of Speech</td>
<td>1.67 (1.35)</td>
<td>1.23 (1.45)</td>
</tr>
<tr>
<td>12. Blocking</td>
<td>1.40 (1.68)</td>
<td>0.62 (1.30)</td>
</tr>
<tr>
<td>13. Increased Latency of Response</td>
<td>1.93 (1.44)</td>
<td>1.15 (1.45)</td>
</tr>
<tr>
<td>14. Subjective Rating of Alogia</td>
<td>1.57 (1.68)</td>
<td>0.58 (1.14)</td>
</tr>
<tr>
<td>15. Global Rating of Alogia</td>
<td>2.67 (1.18)</td>
<td>1.58 (1.30)</td>
</tr>
<tr>
<td>16. Grooming and Hygiene</td>
<td>2.73 (1.34)</td>
<td>1.23 (1.31)</td>
</tr>
<tr>
<td>17. Impersistence at School and Work</td>
<td>2.93 (1.67)</td>
<td>1.23 (1.63)</td>
</tr>
<tr>
<td>18. Physical Anergia</td>
<td>2.93 (1.67)</td>
<td>0.85 (1.15)</td>
</tr>
<tr>
<td>19. Subjective Complaints of Avolition</td>
<td>2.07 (1.71)</td>
<td>0.46 (0.86)</td>
</tr>
<tr>
<td>20. Global Rating of Avolition</td>
<td>3.40 (0.74)</td>
<td>1.31 (1.26)</td>
</tr>
<tr>
<td>21. Recreational Interests and Activities</td>
<td>2.93 (1.49)</td>
<td>1.04 (1.14)</td>
</tr>
<tr>
<td>22. Sexual Interests and Activity</td>
<td>1.40 (1.77)</td>
<td>1.04 (1.76)</td>
</tr>
<tr>
<td>23. Ability to Feel Intimacy and Closeness</td>
<td>2.87 (1.60)</td>
<td>1.31 (1.52)</td>
</tr>
<tr>
<td>24. Relationships with Friends and Peers</td>
<td>3.00 (1.77)</td>
<td>1.50 (1.61)</td>
</tr>
<tr>
<td>25. Subjective Awareness of Anhedonia</td>
<td>2.60 (1.45)</td>
<td>0.77 (1.18)</td>
</tr>
<tr>
<td>26. Global Rating of Anhedonia/Asociality</td>
<td>3.33 (0.90)</td>
<td>1.42 (1.53)</td>
</tr>
<tr>
<td>27. Social Inattentiveness</td>
<td>2.53 (1.55)</td>
<td>1.15 (1.19)</td>
</tr>
<tr>
<td>28. Inattentiveness During Mental Testing</td>
<td>3.87 (1.06)</td>
<td>1.65 (1.50)</td>
</tr>
<tr>
<td>29. Subjective Complaints of Inattentiveness</td>
<td>1.53 (1.92)</td>
<td>1.54 (1.48)</td>
</tr>
<tr>
<td>30. Global Rating of Inattention</td>
<td>3.53 (0.92)</td>
<td>2.00 (1.06)</td>
</tr>
<tr>
<td>SANS Total</td>
<td>76.33</td>
<td>36.38</td>
</tr>
</tbody>
</table>

(23.26) (19.84)
Table 9.

Characteristics and Distribution of Schedule for the Deficit Syndrome Symptoms

Based Upon Symptom Severity

<table>
<thead>
<tr>
<th>Symptom</th>
<th>M</th>
<th>SD</th>
<th>% Moderate or &gt; Severity&lt;sup&gt;a&lt;/sup&gt;</th>
<th>% with primary Symptoms&lt;sup&gt;b&lt;/sup&gt;</th>
<th>% with Enduring Symptoms&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restricted Affect</td>
<td>2.53</td>
<td>0.99</td>
<td>93.3%</td>
<td>78.6%</td>
<td>66.7%</td>
</tr>
<tr>
<td>Diminished Emotional Range</td>
<td>1.87</td>
<td>1.06</td>
<td>66.7%</td>
<td>66.7%</td>
<td>66.7%</td>
</tr>
<tr>
<td>Poverty of Speech</td>
<td>1.67</td>
<td>1.29</td>
<td>53.3%</td>
<td>53.3%</td>
<td>53.3%</td>
</tr>
<tr>
<td>Curbing of Interests</td>
<td>2.47</td>
<td>0.92</td>
<td>93.3%</td>
<td>93.3%</td>
<td>93.3%</td>
</tr>
<tr>
<td>Diminished Sense of Purpose</td>
<td>2.67</td>
<td>1.11</td>
<td>86.7%</td>
<td>93.3%</td>
<td>93.3%</td>
</tr>
<tr>
<td>Diminished Social Drive</td>
<td>2.67</td>
<td>0.90</td>
<td>93.3%</td>
<td>86.7%</td>
<td>93.3%</td>
</tr>
</tbody>
</table>

Note. n = 15 deficit syndrome patients

<sup>a</sup> Percentage of participants in which symptom severity is moderate or greater. Moderate or higher severity is required on at least two symptom domains.

<sup>b</sup> Percentage of participants in which symptom was not caused by neuroleptic akinesia, depression, anxiety, paranoia, or other psychotic symptoms and had at least moderate severity. At least two symptom domains must meet primary criteria to be considered deficit.

<sup>c</sup> Percentage of participants in which symptom was present during the proceeding 12 months, including during periods of clinical stability. At least two symptom domains must meet these stability criteria to be considered deficit.
Table 10.

Intensity and Frequency of Emotional Experience in patient and control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Deficit (1)</th>
<th>Non-Deficit (2)</th>
<th>Control (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>PANAS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trait PA</td>
<td>26.07</td>
<td>10.33</td>
<td>33.58</td>
</tr>
<tr>
<td>Trait NA</td>
<td>17.60</td>
<td>09.00</td>
<td>16.42</td>
</tr>
<tr>
<td>State PA</td>
<td>28.87</td>
<td>11.29</td>
<td>32.69</td>
</tr>
<tr>
<td>State NA</td>
<td>16.27</td>
<td>08.13</td>
<td>14.92</td>
</tr>
<tr>
<td>DES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interest</td>
<td>02.69</td>
<td>01.23</td>
<td>03.38</td>
</tr>
<tr>
<td>Joy</td>
<td>02.71</td>
<td>01.32</td>
<td>03.65</td>
</tr>
<tr>
<td>Surprise</td>
<td>02.81</td>
<td>00.82</td>
<td>02.73</td>
</tr>
<tr>
<td>Sadness</td>
<td>02.11</td>
<td>01.15</td>
<td>02.26</td>
</tr>
<tr>
<td>Anger</td>
<td>02.18</td>
<td>01.15</td>
<td>02.12</td>
</tr>
<tr>
<td>Disgust</td>
<td>01.98</td>
<td>01.08</td>
<td>01.94</td>
</tr>
<tr>
<td>Contempt</td>
<td>01.91</td>
<td>00.84</td>
<td>02.00</td>
</tr>
<tr>
<td>Hostility</td>
<td>02.44</td>
<td>01.06</td>
<td>02.65</td>
</tr>
<tr>
<td>Fear</td>
<td>02.38</td>
<td>01.20</td>
<td>02.40</td>
</tr>
<tr>
<td>Shame</td>
<td>01.96</td>
<td>00.85</td>
<td>02.46</td>
</tr>
<tr>
<td>Shyness</td>
<td>01.96</td>
<td>00.92</td>
<td>02.14</td>
</tr>
<tr>
<td>Guilt</td>
<td>02.02</td>
<td>01.12</td>
<td>02.28</td>
</tr>
</tbody>
</table>

Note. The numbers in parentheses in column heads refer to the numbers used for illustrating significant differences in the last column titled "Post Hoc." PANAS = Positive and Negative Affect Scale; PA = Positive Affect; NA = Negative Affect; PANAS scores represent totals (range = 0-50); DES = Differential Emotions Scale; DES means represent averages (range = 0-5), rather than totals.
Table 11.
Facial affect labeling RT in patient and control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Deficit (1)</th>
<th>Non-Deficit (2)</th>
<th>Control (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Anger</td>
<td>1976</td>
<td>490</td>
<td>1491</td>
</tr>
<tr>
<td>Fear</td>
<td>2517</td>
<td>483</td>
<td>1827</td>
</tr>
<tr>
<td>Happiness</td>
<td>1478</td>
<td>311</td>
<td>1243</td>
</tr>
<tr>
<td>Neutral</td>
<td>2303</td>
<td>602</td>
<td>1544</td>
</tr>
<tr>
<td>Sadness</td>
<td>2289</td>
<td>696</td>
<td>1689</td>
</tr>
<tr>
<td>Surprise</td>
<td>2051</td>
<td>538</td>
<td>1616</td>
</tr>
</tbody>
</table>

*Note.* The numbers in parentheses in column heads refer to the numbers used for illustrating significant differences in the last column titled “Post Hoc.”
Table 12.

Differences in olfactory perception among patient and control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Deficit (1)</th>
<th>Non-Deficit (2)</th>
<th>Control(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Accuracy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Item</td>
<td>65.56</td>
<td>12.75</td>
<td>80.57</td>
</tr>
<tr>
<td>% Correct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Item</td>
<td>53.33</td>
<td>23.50</td>
<td>78.40</td>
</tr>
<tr>
<td>Item% Correct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Correct</td>
<td>07.20</td>
<td>01.78</td>
<td>09.56</td>
</tr>
</tbody>
</table>

Valence

<table>
<thead>
<tr>
<th></th>
<th>Deficit (1)</th>
<th>Non-Deficit (2)</th>
<th>Control(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Positive Item</td>
<td>03.09</td>
<td>01.12</td>
<td>02.45</td>
</tr>
<tr>
<td>Total Valence</td>
<td>03.64</td>
<td>00.92</td>
<td>03.22</td>
</tr>
</tbody>
</table>

Note. The numbers in parentheses in column heads refer to the numbers used for illustrating significant differences in the last column titled "Post Hoc."
APPENDIX B

FIGURES
Figure 1.

Differences in intensity of state and trait emotional experience among patient and control subjects.
Figure 2.

Differences in frequency of emotional experience among patients and control subjects
Figure 3.

*Differences in facial affect labeling accuracy among patient and control subjects.*
Figure 4.

Differences in facial affect labeling RT among patient and control subjects.
Figure 5.

*Differences in total olfactory identification among patient and control subjects.*
Figure 6.

*Differences in olfactory identification within pleasant and unpleasant conditions among patient and control subjects.*
Figure 7.

*Differences in pleasant and unpleasant olfactory valence judgments among patient and control subjects.*
Figure 8.

*Differences in emotional memory bias for discrete emotional categories between patient and control subjects*
Figure 9.

Differences in emotional word recall for discrete emotional categories between patient and control subjects.
Figure 10.

*Differences in emotional and non-emotional word learning among patient and control subjects.*
Figure 11.

Differences in emotional and non-emotional word learning using estimated marginal means adjusted for IQ among patient and control subjects.
Figure 12.

Differences in attention bias for individual emotions among patient and control subjects.
Figure 13.

Differences in E-Stroop Emotional Lingering Effects for positive and negative words in patient and control groups.
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