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Emotional processing in schizophrenia

Christian G. Kohler and Elizabeth A. Martin
University of Pennsylvania, Philadelphia, PA, USA

Introduction. Persons with schizophrenia have impaired emotional processing, involving experience, expression, and recognition of emotions.
Methods. This article reviews the historical descriptions and more recent work on emotion processing in schizophrenia.
Results. Although abilities of emotional processing relate directly to interpersonal communication and psychosocial functioning, methodological issues exist in the current body of studies and resultant knowledge, which limit translation to novel treatment options.
Conclusions. Further improvement in emotion processing in persons with stable schizophrenia are unlikely to result from conventional pharmacotherapy of psychosis. New treatment modalities and behavioural interventions offer possible improvements in quality of life and psychosocial functioning.

Schizophrenia (SZP) has traditionally been viewed as a psychiatric illness with prominent clinical features of psychosis or positive symptoms, negative symptoms, and cognitive dysfunction. Deficits in cognition, considered a stable hallmark of SZP, have long been described and in the early part of this century were used to differentiate SZP from affective illnesses. Investigations have shown quite marked neuropsychological functioning deficits in SZP, of a magnitude similar to performance of patients with brain injury (for a review, see Heaton & Crowley, 1981), and these impairments are present in patients treated with medications (Braff et al., 1991), as well as in neuroleptic naive patients with first-episode SZP (Censits, Ragland, Gur, & Gur, 1997; Saykin et al., 1994).

Emotional processing can be parsed along dimensions of emotion experience, expression, and recognition. Early descriptions of the phenomenology of SZP included disturbances in emotional processing, such as abnormal expression and abnormal experience as symptoms inherent, but not always characteristic to the illness. Kraepelin's (1896) Binary Model proposed an exclusivity of SZP and

Correspondence should be addressed to Dr Christian G. Kohler, Neuropsychiatry Section, 10 Gates Building, Hospital of the University of Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104, USA.

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affective phenomena and delineated a clear distinction between dementia praecox and manic-depressive illness. However, by 1919, Kraepelin (1919/1971) reported affective disturbances that were present at the onset of dementia praecox ''... usually anxious, despondent, they weep and lament, would like to die...''. and became less prominent during later stages of the illness with more prominent somatic preoccupation and self-neglect. Eugen Bleuler (1911), whose views have been influential on our understanding of SZP, divided the phenomenology of SZP into primary and secondary symptoms. Primary symptoms—disturbances in affect, association, ambivalence and, autism—were seen as partial, but necessary for the diagnosis. Secondary symptoms, which included not only delusions and hallucinations but also depressed mood, could be absent or could change without any alteration in the underlying process.

Similarly, impaired interpersonal skills have been described as hallmarks of the illness, and both expression and recognition of facial emotions are viewed as major components of interpersonal communication. With respect to expression, although affective flattening is an established symptom of SZP, little is known about its characteristics and course and, except for clinical ratings, limited quantitative data are available. An increasing body of literature has examined emotion recognition deficits in SZP. Once acute psychotic symptoms are stabilised, the ability to express and recognise emotions substantially relates to interpersonal and social functioning.

Over the past 30 years, these dimensions of emotional processing and their respective dysfunctions have been subjected to increasing clinical attention and research. As noted above, SZP presents as a heterogeneous illness with wide-ranging symptoms encompassing positive and negative symptoms, dysfunction in cognition and emotion processing, and psychosocial impairment. Approaches to treatment have moved beyond treatment of acute psychosis and, in people who are in the chronic or residual phases of the illness, have increasingly focused on improvement in negative symptoms, cognition, emotional processing, and psychosocial remediation. This article describes the recent literature on experience, recognition, and expression in SZP, and delineates possible future directions to investigate and remediate these areas of dysfunction, which in turn may ameliorate psychosocial functioning and quality of life in persons with SZP.

NEUROBIOLOGY OF EMOTIONAL PROCESSING

Different concepts exist regarding the constituents of emotions, and most theories include the combination of a physical and cognitive, or attributional, component. The Jamesian Model (James, 1884/1922) postulates that physical changes as the result of an external experience are sufficient to qualify for an emotional experience. Cannon (1927/1987) argued that the central component of emotional experience is not the physical sensation, but a brain-based feeling,

which he claimed to be located in the thalamus. Schachter and Singer (1962) expanded the Jamesian Model in that emotional experience is based on the cognitive attribution to bodily changes or arousal.

According to Tomkins (1962), emotions are motor and glandular responses originating in the face and other bodily regions, which are triggered by subcortical affect programmes and provide sensory feedback to cortical brain regions. It is unclear whether this model proposes the brain-based experience to represent the trigger or the consequence of the emotion. However, important for further research on emotions, it emphasises the face as a principal constituent in the processing of emotions. Similarly, Damasio (1994) postulated that the content of emotional experience consists of bodily changes, juxtaposed to the precipitant or cause of the emotional experience and the associated change in one's mode of thinking.

Processing of emotions is supported by distributed neural systems, and as lesion studies show, preferentially of the right hemisphere. There are two main theories regarding the hemispheric specialisation in processing emotions (Borod et al., 1998). The hemispheric hypothesis posits that the right hemisphere is specialised to process all emotions, whereas the valence hypothesis regards the right hemisphere as superior for processing negative and the left hemisphere superior for positive emotions. Common negative emotions are sadness, fear, and anger, common positive emotions are happiness and, possibly, surprise. In human and primate studies, the amygdala, which receives input from sensory cortical areas and via subcortical input from the visual pathways of the midbrain and thalamus, is thought to be preferentially involved in the processing of fear and other emotions. A recent model based on animal and human studies (Phillips, Drevets, Rauch, & Lane, 2003) proposes that emotion perceptions may be dependent on two neural systems. The ventral limbic system, including amygdala, insula, ventral striatum, and ventral regions of the anterior cingulate gyrus and prefrontal cortex, is vital for identification of the emotional significance of a stimulus and the subsequent production of the affective state. The dorsal system includes the hippocampus and dorsal regions of the anterior cingulated gyrus and prefrontal cortex, and is important in executive functioning required for the regulation of affective states. In schizophrenia, which affects most functions of the limbic system, all dimensions of emotional processing, including experience, expression, and recognition are commonly affected. Impairment in these areas may be most relevant in persons who have achieved remission of stability with respect to acute psychosis.

There is limited information concerning brain regions and their dysfunction associated with euphoric and manic mood states. In idiopathic and secondary mania, functional imaging studies have revealed asymmetric temporal lobe activity (Gyulai et al., 1997; Migliorelli et al., 1993; Starkstein et al., 1990), which may remit with stabilisation of mood (Gyulai et al., 1997). Functional imaging studies in idiopathic depression (Baxter et al., 1985; Dolan & Friston,

1989; Drevets, 1999; Mayberg, Lewis, Regenold, & Wagner, 1994) and depression associated with schizophrenia (Kohler, Swanson, Gur, Harper Mozley, & Gur, 1998), stroke (Robinson, Kubos, Starr, Rao, & Price, 1984), epilepsy (Bromfield et al., 1992), and neurodegenerative disorders (Mayberg et al., 1990; Mayberg, Starkstein, Peyser, Brandt, & Dannals, 1992) have implicated left inferior frontal and cingulate brain dysfunction.

EMOTION EXPERIENCE

Emotional Intensity

In a healthy individual, issues of emotional experience and intensity remain poorly understood. Difficulties lie in measuring one's emotional experience, gauging its quality, and quantifying its intensity. Measurements of emotional experience via emotion induction have included presentation of emotional faces and pictures, exposure to emotive music, and the recall of one's own emotional experiences.

Historically, SZP was thought to involve decreased range of emotional experience. According to Sass and Parnas (2003), SZP is defined as an ipseity disturbance and anhedonia, defined as diminished ability to experience pleasure, represents both a crucial element of such a lack of self-experience and a characteristic, common negative symptom of SZP. Lack of emotional experience is typically inferred from a deficiency to identify one's own emotional experience and/or impairment in facial expression of emotions, also referred to as flattening of affect.

It has been described that persons with SZP may be unreliable reporters of their own emotional experience (Jaeger, Bitter, Czobor, & Volavka, 1990). However, others have described that SZP subjects report similar emotional experience in comparison with depression (Berenbaum, 1992; Sison, Alpert, Fudge, & Stern, 1996) and healthy controls (Kring & Neale, 1996). Kring and Neale (1996) found that in response to emotionally stimulating film clips, patients with SZP are less facially expressive than healthy controls but report similar emotional experiences and greater autonomic responses.

Employing an emotional startle paradigm, Volz, Hamm, Kirsch, and Rey (2003) reported that in response to unpleasant pictures, people with SZP exhibited lessened affective modulation upon early but not late tone presentations. This suggests delayed responses to affective stimuli in schizophrenia. Lastly, blunted facial expression of emotions in SZP may not correlate with emotional experience (Earnst et al., 1996; Kring, Kerr, Smith, & Neale, 1993; Myin-Germeys, Delespaul, & de Vries, 2000; Sison et al., 1996), which in some of these studies has been described as similar to emotional experience in healthy controls.

Applying a mood induction procedure with happy and sad faces to 40 SZP individuals and gender-matched healthy controls, Schneider, Gur, Gur, and

Shtasel (1995) found that patients exhibited impaired ability to respond to mood induction, particularly for happiness irrespective of medication status and gender. Performance on mood induction was positively correlated with hallucinations, and negatively correlated with the negative symptom complex of anhedonia. Taylor, Liberzon, Decker, and Koeppe (2002) examined emotional experience and processing in SZP and healthy subjects using positron emission tomography and found that while patients reported similar subjective experience to aversive stimuli, the groups differed in brain activation consistent with impaired processing of emotional stimuli in SZP.

Future directions and investigations into emotional experience in SZP may include presentation of emotional stimuli in conjunction with self-rating measures during neuroimaging procedures, which will elucidate the neural substrate of feelings and the putative alteration of its function in SZP.

Depression

Relationship to treatment. Following the introduction of antipsychotic medications in treating SZP, clinical reports appeared that described a dramatic increase in the incidence of depression and suicide (Beisser, 1961; Hussar, 1962). Experience with the antihypertensive agent reserpine, also used to treat SZP and known to cause depression in some patients, created the concern that depression might result from pharmacotherapy in SZP. This led to the introduction of the term ''pharmacogenic depression'' (Galdi, 1983; Helmchen & Hippius, 1967). A further suggestion was that treatment with typical antipsychotics may cause depressive symptoms due to extrapyramidal motor disturbances or ''akinetic depression'' (Craig, Richardson, Pass, & Bregman, 1985; Johnson, 1981; Rifkin, Quitkin, & Klein, 1975; Van Putten & May, 1978). Subsequent studies refuted the association between antipsychotics and depression in general and describe that depression occurs most commonly during the onset of psychosis (Bowers & Astrachan, 1967; House, Bostock, & Cooper, 1987; Johnson, 1981; Knights & Hirsch, 1981; Koreen et al., 1993; Mayer-Gross, Slater, & Roth, 1955; McGlashan & Carpenter, 1976; Roth, 1970; Steinberg, Green, & Durell, 1967; Stern, Pillsbury, & Sonnenberg, 1972). Reduction of acute symptoms and clinical stabilisation are associated with decline of depressive symptoms (House et al., 1987; Knights & Hirsch, 1981; Shanfield, Tucker, Marrow, & Detre, 1970), that may re-emerge during each relapse (Shanfield et al., 1970; Johnson, 1981; Koreen et al., 1993). Depression appears less common with increasing chronicity of illness, perhaps because its symptoms are gradually replaced by negative symptoms (House et al., 1987).

Relationship to onset of illness. Koreen et al. (1993) followed 70 men and women with first-episode SZP for up to 5 years. Before initiation of treatment, three fourths of the subjects experienced significant symptoms of depression, as

measured by the Hamilton Rating Scale for Depression, and approximately one third experienced syndromatic criteria of major depression. Symptoms of depression remitted with stabilisation of psychosis and without specific antidepressant treatment. In an attempt to examine the differentiation between SZP and major depression, Wassink, Flaum, Nopoulos, and Andreasen (1999) reported on 75 persons with recent-onset SZP who were followed for 5 years and about one third met the algorithmic criteria for a major depressive episode, while no symptoms were present in about one tenth of persons. The findings highlight that depressive symptoms are common early in the course of SZP with potential implications for diagnostic and treatment practices. In addition, these studies offer support of depression in schizophrenia being associated with positive symptoms.

Suicidality. An increased risk of suicide attempts by people with SZP has been identified (Harkavy-Friedman, Nelson, Venarde, & Mann, 2004). Between 8% and 13% of people with SZP commit suicide (Meltzer & Okayli, 1995; Miles, 1977; Nyman & Jonsson, 1986), and suicide presents as a common cause of premature death in SZP (Palmer, Pankratz, & Bostwick, 2005). The majority of suicide completers were depressed in the few months directly preceding their deaths (Cohen, Leonard, Farberow, & Shneidman, 1964; Planansky & Johnston, 1971) underscoring the need for treatment of depression in SZP.

Another and less dramatic, but pernicious consequence, is the pervasive negative effect of depression on enjoyment of life and psychosocial functioning (Hofer et al., 2004), which is already quite impaired in persons with SZP who do not experience depression.

Relationship to negative and positive symptoms. A major difficulty in evaluating depression in SZP stems from the potential overlap of negative symptoms with depression. While several studies found little or no association between depressive and negative features (Goldman, Tandon, Liberzon, & Greden, 1992; Kibel, Laffont, & Liddle, 1993; Kuck, Zisook, Moranville, Heaton, & Braff, 1992), other studies (Craig et al., 1985; Kulhara et al., 1989; Prosser et al., 1987) found an association between negative and vegetative features of depression. Vegetative symptoms of depression consist of somatic and nonspecific behavioural disturbances, such as anergia, anhedonia, insomnia, and lack of appetite, and can be found in many psychiatric disorders. Kitamura and Suga (1991) reported an association between depressive ratings and negative ratings, specifically for avolition-apathy and anhedonia-asociality, and Ring et al. (1991) noted a correlation between negative and depressive symptoms but only in male patients. No study found a link between negative symptoms and depressive cognitions, which are more specific to depression and consist of sadness, anxiety, guilt, tension, and somatic concern.

In an effort to separate the effects of depression from symptoms primarily attributable to SZP, such as negative and positive symptoms, we compared a group of SZP subjects with depression to a group without depression (Kohler, Gur, Swanson, Petty, & Gur, 1998). There were 63 patients (35 men, 28 women) in the high (HD) and 81 patients (52 men, 29 women) in the low depression (LD) group. The groups were compared on demographic, clinical, and eight neuropsychological domains. They differed in age at onset of illness (later in HD group), delusional thinking (more severe in HD group), and performance in a single neuropsychological domain, attention (more impaired in HD group). The presence of specific attentional impairment associated with depressive symptoms in SZP is consistent with the hypothesis of frontal lobe dysfunction in depression, as these regions have been implicated in maintaining a euthymic mood state and in attentional processes. In people from this group who underwent magnetic resonance imaging (MRI) and PET, we found larger temporal lobes and relatively decreased left anterior cingulate metabolism in HD. Although the former finding was not expected, it may indicate that intact temporal lobes are necessary for emotional experience. The finding on decreased left medial frontal metabolism is consistent with other studies in idiopathic depression and depression in brain-related disorders as outlined above.

Studies on the treatment of depression in SZP have suffered methodological limitations concerning criteria and scales for assessing depression. Most studies have centred on the treatment of depressive symptoms in chronic institutionalised patients and only a few have examined the role of antidepressant treatment in patients suffering from an acute episode. Studies, which evaluated the presence of depressed mood in patients with acute psychosis, reported improvement in both psychotic and depressive symptoms in patients with SZP who were treated with mostly typical (Johnson, 1981; Knights & Hirsch, 1981; Koreen et al., 1993; McGlashan & Carpenter, 1976) and atypical antipsychotics (Arvaniitis & Miller, 1997; Keck et al., 1998; Meltzer, Lee, & Ranjan, 1994; Tollefson, Sanger, Lu, & Thieme, 1998).

Two comprehensive reviews (Plasky, 1991; Siris, 1991) of the literature on depressive symptoms in SZP preceding the widespread use of atypical antipsychotics evaluated over 30 publications and found only three papers that clearly support the benefit of antidepressant treatment. Many studies were difficult to interpret due to flawed designs, such as inadequate assessment of depression and small number of patients.

According to a more recent review of studies on the treatment of depression in SZP by Levinson, Umapathy, and Musthaq (1999), the beneficial effect of antidepressants is limited to patients who were treated as outpatients or exhibited more stable psychotic symptoms. Antidepressants show no benefit in patients without clear depressive symptoms, with florid psychotic symptoms, and of questionable or limited benefit in chronic inpatients and patients with

unstable psychotic symptoms. Coinciding with the advent of atypical antipsychotics, there is a lack of treatment studies on depression in SZP using newer generation selective serotonin and noradrenalin-serotonin reuptake inhibitor medications.

Over the last 10 years atypical neuroleptics, which affect serotonergic pathways, have found widespread usage in the treatment of SZP. Of this group of medications, clozapine and olanzapine have been investigated for specific antidepressant effect in SZP and mood disorders (Meltzer et al., 1994; Weisler, Ahearn, Davidson, & Wallace, 1997; Zarate, Tohen, & Baldessarini, 1995). In a large cohort of patients with acute exacerbation of chronic SZP, average dosed olanzapine treatment was found to improve anxious-depressed symptoms, when compared to placebo and haloperidol. This effect was found to be independent of improvement in positive, negative and extrapyramidal symptoms (Tollefson et al., 1998; Tran et al., 1997). To a lesser extent, there is similar evidence for improvement of mood symptoms in SZP as the result of treatment with quetiapine (Arvanitis & Miller, 1997) and ziprasidone (Keck et al., 1998) and no clear evidence for risperidone (Ceskova & Svestka, 1993; Müller et al., 1998; Tran et al., 1997). In addition, atypical antipsychotics in general (Barak, Mirecki, Knobler, Natan, & Aizenberg, 2004), and clozapine in particular (Meltzer & Okayli, 1995), have been found to reduce suicidality and completed suicide.

While depression is a well-described comorbidity of SZP, future goals remain in improved treatment of depression and prevention of suicidality. Depression has been associated with worse quality of life in SZP (Hofer et al., 2004), and its improvement will lead to better psychosocial functioning. Apart from medication treatment for depression, there has been increased interest in applying psychotherapy beyond supportive measures, most recently employing cognitive-behavioural therapy (CBT). This therapy is based on the premise that thoughts exert influence on a person's emotions and behaviour. Identifying and correcting cognitive distortions can lead to improvement in a person's emotional state and behaviour. Unlike supportive therapy, which is not time-limited, CBT represents a structured and time-limited approach, typically consisting of 20 sessions over 6–9 months. Patients learn to view delusions and hallucinations as symptoms, that are part of their psychiatric disorder, rather than as frightening and believable entities. Over the past 10 years a number of studies (Beck & Rector, 2000; Drury, Birchwood, Cochrane, & Macmillan, 1996; Kingdon & Tuckington, 1991; Kuipers et al., 1997; Lewis et al., 2002; Pinto, La Pia, Mennella, Giorgio, & DeSimone, 1999; Sensky et al., 2000; Tarrier et al., 2000) on the benefit of cognitive therapy in schizophrenia. Although most studies focused on positive and negative symptom improvement, a single study (Sensky et al., 2000) showed significant and lasting reduction of depressive symptoms over a period of nine months.

Mania

Unlike depression in SZP, mania has been much less commonly described. Bleuler (1911) postulated that any affective symptoms may occur in the setting of SZP, provided that criteria for certain fundamental schizophrenic symptoms were met: splitting of cognition from emotion and behaviour, formal thought disorder, flat or blunted affect, autism, and ambivalence.

Manic symptoms in the setting of SZP may represent a short-lived schizomanic state or warrant the diagnosis of schizoaffective disorder. Patients with SZP sometimes display manic symptoms (Tsuang & Loyd, 1988) and become agitated, irritable, impulsive, and insomniac as part of an acute psychotic exacerbation or in response to hallucinations and delusions. Usually, these symptoms are temporary and the behavioural picture lacks more typical manic symptoms (e.g., pressured speech, grandiosity, and elated mood). Kasanin (1933/1994) coined the term ''schizoaffective psychosis'' to describe a group of patients with sudden onset in youth of prominent affective and schizophrenic symptoms, who experienced an external stressor and had good premorbid adjustment. The initial description did not specify the relationship of schizoaffective disorder to affective disorders or SZP.

The primary difficulty in diagnosing schizoaffective disorder is the differentiation from SZP with an atypical affective disorder or from a mood disorder with incongruent psychotic features. Thus, the existence of schizoaffective disorder as a separate clinical entity has repeatedly been questioned. In addition, bipolar disorder (BPD) and SZP may share genetic factors as evidenced by epidemiologic characteristics, family studies, and overlap in confirmed linkages of BPD and/or SZP (Craddock, O'Donovan, & Owen, 2005; Murray et al., 2004). Treatment of manic symptoms in SZP beyond antipsychotics includes mood stabilisers, such as valproate, which are frequently used as secondary medications in SZP (Casey et al., 2003). However, their utility remains controversial (Basan, Kissling, & Leucht, 2004).

FACIAL EXPRESSION OF EMOTIONS

Healthy individuals

The ability to produce and recognise facial expressions of emotion represents an important component of interpersonal communication in humans and primates (Darwin, 1872). Six basic emotions—happiness, sadness, anger, fear, disgust, and surprise—and their corresponding facial expressions are recognised across different cultures (Eibl-Eibesfeldt, 1970; Ekman, Friesen, & Ellsworth, 1972; Huber, 1931; Izard, 1994). Although universal emotions are recognised across different cultural and ethnic groups, social emotions, such as guilt, shame, arrogance, admiration, and flirtatiousness, are particular to culture specific interactions.

Schizophrenia

Diminished affect, the facial expression of emotion, has been described since Bleuler (1911) as a core symptom in SZP. Impairment commonly consists of flat or inappropriate affect, may precede the onset of psychosis by many years (Walker, Grimes, Davis, & Smith, 1993) and can be worsened by administration of neuroleptics with strong nigrostriatal dopaminergic blockade (Krakowski, Czobor, & Volavka, 1997; Rifkin et al., 1975; Van Putten & May, 1978).

Measurement

The main difficulty in studying flat affect is the lack of reliable, objective, and efficient methods for quantifying facial expressions. Whereas there are widely used and validated instruments that measure and parse aspects of cognitive dysfunction and its neurobiology, overall assessment of negative symptoms have been limited to observer-based rating scales, such as the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984) and the Positive and Negative Symptom Scale (PANSS; Kay, Fiszbein, & Opler, 1987). Initial studies on facial affect in SZP beyond measurement with rating scales found both intact posed (Gottheil, Thornton, & Exline, 1976) and decreased spontaneous expressions (Gottheil, Paredes, Exline, & Winkelmayer, 1970).

The Facial Active Coding System (FACS), created by Ekman and Friesen (1978), remains the ''gold standard'' for qualitative analysis of facial emotional expressions. Unfortunately, FACS presents as a subjective rating measure that requires extensive training and still falls short of yielding measures thata are sensitive to subtle effects. The FACS has been simplified and adapted for clinical research, yielding EMFACS (Friesen, 1986) and FACES (Kring et al., 1993). While EMFACS identifies the presence or absence of muscle movements associated with the predicted expression of the particular emotion, FACES rates overall dynamic facial changes, according to number of positive and negative expressions, their intensity, and duration. Limitations of these methods include that EMFACS catalogues only those facial movements which Ekman and Friesen determined as part of posed facial expressions, while FACES does not rate individual muscle movements or action units. Using EMFACS (Berenbaum, 1992; Berenbaum & Oltmanns, 1992), persons with depression, but not SZP, exhibited fewer happy expressions during happy induction and more anger expressions during disgust induction. However, this difference may not be disorder-specific, as SZP compared to controls, but not psychosomatic probands (Steimer-Krause, Krause, & Wagner, 1990), displayed decreased variability of spontaneous expressions. While viewing brief emotion-inducing film clips, SZP probands exhibited less expressivity, as measured by FACES, but similar experience (Kring et al., 1993). Two recent studies examined posed (Trémeau et al., 2005)

and spontaneous (Gaebel & Wölwer, 2004; Trémeau et al., 2005) expressions of emotions in persons with SZP during acute and post-acute stages of illness, and depression. Both patient groups exhibited less spontaneous expressions, irrespective of typical or atypical antipsychotic medication effect (Gaebel & Wölwer, 2004; Trémeau et al., 2005) and did not differ according to illness acuity in SZP. Studies that measured facial EMG in response to emotional films (Earnst et al., 1996; Mattes, Schneider, Heimann, & Birbaumer, 1995) and pictures (Kring, Kerr, & Earnst, 1999) reported similar (Earnst et al., 1996; Mattes et al., 1995) and maybe inappropriate expressions (Kring et al., 1999) in SZP. Limitations in these studies consisted of placing foreign objects (i.e. electrodes), over only two selected facial muscles and possibly over-identifying expressions, including micro-expressions which cannot be discerned in real-life settings. The potential influence of extrapyramidal side-effects on emotion expression remains unclear. While antipsychotics, in particular typicals, have been associated with akinesia, recent studies (Earnst et al. 1996; Kring et al., 1999) examined patients both on and off antipsychotics and revealed no clear effect on expressivity and emotional experience.

These studies provide evidence for the presence of affective flattening in SZP and its independence from emotion experience and medication effects. However, they fail to elucidate and more thoroughly characterise facial expression of emotions in SZP, which in the clinical realm presents as an inherent and stable symptom of SZP, associated with poor prognosis (Edwards, McGorry, Waddell, & Harrigan, 1999; Ho, Nopoulos, Flaum, Arndt, & Andreasen, 1998; Shatsel, Gur, Gallacher, Heimberg, & Gur, 1992). None of above-described studies employed classic or traditional FACS ratings, which may account for the lack of findings specific to SZP. Conversely to FACES or EMFACS, which provide holistic assessments and take into consideration previous predictions on facial expressions, FACS allows for ratings of individual muscle movements across all regions of the face.

Future studies that examine emotion expression in SZP, and possible improvement, will need to employ better quantification of emotion expression. Our group has developed a set of algorithms for highly automated analysis of facial expressions (Verma et al., 2005), which can quantify differences in expression. We anticipate that reliable quantification of affective flattening and emotion expression in persons with stable SZP in conjunction with novel medications or remediation treatment, may provide improvement in theses characteristic and debilitating symptoms of SZP.

FACIAL EMOTION RECOGNITION

Over the last 15 years, a large body of literature has examined emotion recognition, as measured by the ability to identify the emotional quality of facial expression, in brain-related disorders and healthy people and documented

impairment in right brain-injured people (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod, Martin, Alpert, Brozgold, & Welkowitz, 1993), SZP (for reviews, see Kohler et al., 2003; Mandal, Pandey, & Prasad, 1998; Morrison, Bellack, & Mueser, 1988) depression (Feinberg, Rifkin, Schaffer, & Walker, 1986; Gur et al., 1992; Mikhailova, Vladimirova, Iznack, Tsusulkovskaya, & Sushko, 1996), bipolar disorder (Addington & Addington, 1998; Lembke & Ketter, 2002), Alzheimer disease (for a review, see Kohler et al., 2005), and Huntington's chorea (Jacobs, Shuren, & Heilman, 1995).

Few studies have examined brain functioning in schizophrenia during emotion recognition. An earlier study in which persons judged the emotional intensity of faces showed increased amygdala activation in patients as the possible result of impaired sensory gating and impaired inhibition of amygdalar response (Kosaka et al., 2002). Two subsequent studies (Gur et al., 2002; Hempel, Hempel, Schonknecht, Stippich, & Schroder, 2003) found decreased activation during emotion recognition, in concordance with previous findings of amygdala activation deficits in schizophrenia (Phillips et al., 1999; Schneider et al., 1998).

Methodological limitations

Whereas cognitive deficits are well characterised in SZP, the findings of emotion recognition impairment in SZP are limited by the use of differing emotional stimuli and different stages of illness. More recent emotion recognition studies in SZP have employed standardised face stimuli and better characterised samples and have reported on associations of emotion processing in SZP, duration of illness (Mueser et al., 1996; Silver, Shlomo, Turner, & Gur, 2002), and symptomatology (Bryson, Bell, & Lysaker, 1997; Kohler et al., 2003; Mandal et al., 1998; Penn et al., 2000; Schneider et al., 1995). For example, in institutionalised patients, duration and severity of illness, and social competence (Mueser et al., 1997; Silver et al., 2002) were found to correlate with emotion recognition. In addition, research using standardised face stimuli to examine cognition found a relationship between cognitive abilities and affect perception in SZP in general (Bozikas, Kosmidis, Anexoulaki, Giannakou, & Karavatos, 2004), and more specifically, a link between deficits in emotional recognition and cognitive abilities (Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000; Sachs, Steger-Wuchse, Kryspin-Exner, Gur, & Katschnig, 2004).

Another issue raised is whether a differential deficit (Chapman & Chapman, 1978) can be demonstrated against the more general impairment in facial processing (Archer, Hay, & Young, 1994; Edwards, Pattison, Jackson, & Wales, 2001; Johnston, Katsikitis, & Carr, 2001; Kerr & Neale, 1993; Kohler et al., 2000; Novic, Luchins, & Perline, 1984; Penn et al., 2000; Salem, Kring, & Kerr, 1996). Some of the initial studies reported a differential deficit, but the majority of recent investigations (Kerr & Neale, 1993; Penn et al., 2000; Salem et al.,

1996) failed to support that notion. Regardless of whether differential or part of general impairment, emotion recognition abilities will exert effects on psychosocial functioning independent of the presence and severity of positive and negative symptoms and cognitive difficulties. Impaired emotion recognition may also be related to the tendency of persons with SZP to visually scan features of the face that are not important in the expression of a particular emotion, as has been shown with computerised scanpath procedures (Loughland, Williams, & Gordon, 2002).

Comparison with affective disorders

Findings of emotion recognition impairment in SZP are limited by the relative lack of comparison with other patient groups with psychosis, such as major depression or bipolar disorder. Studies on SZP using emotion recognition tasks found that patients performed more poorly than depressed patient groups and controls (Feinberg et al., 1986; Gessler, Cutting, Frith, & Weinman, 1989; Schneider, Koch, Mattes, & Heimann, 1992; Walker, McGuire, & Bettes, 1984; Zuroff & Colussy, 1986). Improvement of emotion recognition has been reported in persons with major depression (Mikhailova et al., 1996) and BPD (Addington & Addington, 1998) after treatment response, however, the deficit may be more stable in SZP (Gaebel & Wölwer, 1992). More recently, first-episode patients with SZP were found to perform worse than affective psychosis patients and controls, particularly in recognition of fear and sadness. Similarly, a single study (Addington & Addington, 1998) that compared emotion recognition in young persons with stable SZP and BPD found impaired affect identification and discrimination in SZP outpatients.

Treatment

Recently, there has been increased emphasis on identifying targets of treatment beyond positive symptoms and improvement in psychosocial functioning. The MATRICS programme (Green & Nuechterlein, 2004; Marder, Fenton, & Youens, 2004) has underscored that cognitive dysfunction, including social cognition as one of seven domains, accounts for considerable residual psychosocial impairment in outpatient SZP populations. Social cognition—including recognition of emotional behaviour and its context—has been described as independent of clinical symptoms in persons with acute SZP (Penn et al., 2002), and its improvement is related to social abilities to a greater extent than nonsocial cognition or neurocognition (Penn, Corrigan, Bentall, Racenstein, & Newman, 1997). In a recent large scale effort on cognitive remediation in persons with stable schizophrenia (Hogarty et al., 2004), cognitive enhancement therapy but not supportive therapy produced improvement in social cognition over a period of 2 years. While emotion recognition impairments have implications for persons with mental illness regardless of stage of illness, the

ability to recognise nonverbal communication, specifically facial expressions of emotions, may be most problematic once psychotic symptoms have stabilised and treatment goals have been focused on improving psychosocial functioning (i.e., interpersonal relationships, work, and education). In support of possible malleability of emotion recognition abilities and underscoring the potential link to psychosocial functioning in SZP, recent attempts have shown an effect with even brief remediation (Penn et al., 2000; Silver, Goodman, Knoll, & Isakov, 2004) and for prolonged cognitive remediation on emotion perception (van der Gaag, Kern, van den Bosch, & Liberman, 2002; Frommann, Streit, & Wölwer, 2003), but not neurocognition (van der Gaag et al., 2002). While these attempts aimed at mostly chronic and institutionalised patients, a 12-week training programme (Frommann et al., 2003) showed emotion recognition improvement in the majority of 16 post-acute SZP patients. Persons with SZP who are hospitalised or have been ill for many years may benefit from improved emotion recognition, yet young persons who are clinically stable will derive the greatest benefit from improved emotional processing and the effect on psychosocial functioning. Such rehabilitation measures may take into account recent findings showing an selective effect of eye regions on brain-based processing of emotions (Whalen et al., 2004), in particular fear, and successful remediation of fear recognition impairment by training a person with amygdala damage to focus on the eyes (Adolphs, Tranel, & Buchanan, 2005) Remediation may also be guided by training people to focus on facial features that are emotionally salient, using behavioural techniques or scanpath methods.

CONCLUSIONS AND FUTURE DIRECTIONS

We have outlined the dimensions of emotion processing—experience, expression and recognition—with respect to historical perspectives and the recent increase in efforts to understand their dysfunction in SZP. In all three areas there are methodological issues, which limit our understanding of their respective impact and our ability to pursue novel potential modalities of treatment. However, and as recent studies show, further improvement in emotion processing in persons with stable SZP are unlikely to result from conventional pharmacotherapy of psychosis. In the illness of schizophrenia, which includes heterogeneous symptoms of psychosis, negative symptoms, dysfunction in cognition and emotional processing, and resultant marked psychosocial impairment, it is imperative to pursue novel treatment modalities to alleviate symptoms of this multifaceted and vexing illness. Such treatment modalities may include medications that exert differential effects on the dopaminergic system, similar to aripiprazole, or selectively affect nicotinic receptors, which may improve abilities of emotion expression and recognition. Similarly, more

novel and selective behavioural interventions that target areas of emotional processing offer possible improvements in quality of life and psychosocial functioning.

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