

Emotion Deficits in People with Schizophrenia

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Abstract

Translational research on emotion in schizophrenia has revealed deficits in emotion perception and expression, as well as intact areas, including emotional experience and brain activation in the presence of emotionally evocative material. Yet, a closer look at emotional experience reveals that all is not well in the experience domain. People with schizophrenia have difficulty anticipating emotional events and maintaining or savoring their emotional experiences, as evidenced in behavioral, psychophysiological, and brain imaging studies. Furthermore, people with schizophrenia have difficulty integrating emotion perception with context and reporting on feelings that are differently valenced than presented emotional stimuli. Differences in brain activation are typically observed in areas tightly coupled with cognitive control, such as the dorsolateral prefrontal cortex, and thus the latest research on emotion in schizophrenia explicitly integrates emotion and cognition. Translational research holds promise to identify when in the course of the disorder emotion deficits emerge and to develop more effective interventions for schizophrenia.

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INTRODUCTION

Common notions about schizophrenia suggest that it is a disorder that disrupts thinking and perception. People with schizophrenia may have delusional beliefs (e.g., I am being followed by federal agents), have hallucinations (e.g., I hear voices warning me to avoid certain public places), or exhibit extremely disorganized speech. Schizophrenia can also disrupt emotion, however, and emotion deficits in schizophrenia can influence a wide swath of other life domains, including social relationships, communication, and motivation. In this review, we discuss the types of emotion deficits that have been observed in schizophrenia, when in the course of the disorder these deficits emerge, and how emotion deficits are connected with other cognitive and behavioral deficits in schizophrenia. However, schizophrenia is not just about emotion deficits. As we discuss, people with schizophrenia experience strong feelings in the same ways as do people without schizophrenia. The integration of affective and cognitive neuroscience research in schizophrenia has opened a new window of understanding about how and when emotion does (and does not) go awry in schizophrenia. Before we get to these exciting developments, we begin by constraining definitions of schizophrenia and emotion.

WHAT IS SCHIZOPHRENIA?

Before discussing what schizophrenia is, it is useful to first consider what it is not. First, schizophrenia is not a new illness. In fact, some of the earliest written accounts of mental disorders include descriptions of what we would today call schizophrenia. It wasn't until the early 1900s, however, that our current conceptualization of schizophrenia began to take shape. In 1908, the Swiss psychiatrist Eugen Bleuler proposed the term schizophrenia, which he formed by combining the Greek words *schizein* (to split) and *phren* (mind), capturing what he viewed as the essential nature of the condition, which was the “breaking of associative threads.” Unfortunately, the literal meaning of the word schizophrenia—splitting of the mind—causes people to confuse schizophrenia with dissociative identity disorder (DID), which was formerly called multiple personality disorder, or split personality, which it is decidedly not. Indeed, the symptoms of schizophrenia are quite distinct from the symptoms of DID. In DID, a person experiences at least two distinct ways of thinking, feeling, and being in the world—having at least two distinct personalities. Yet, none of the personalities exhibit the symptoms of schizophrenia. Confusion between the two disorders is further

perpetuated by incorrect use of the word schizophrenic as an adjective in the popular vernacular. For example, a headline in the *New York Times* sports section from March 2010 read: “Wake Forest outlasts Schizophrenic Texas.” The article went on to discuss the ambiguous prospects for Wake Forest against Texas in their first-round NCAA tournament game, wondering if Wake Forest would face the winning, streaking Texas or the Texas that had lost nine games in a row (Evans 2010). In this article, the adjective schizophrenic was used to characterize two sides to the Texas team, akin to a Dr. Jekyll and Mr. Hyde, or, in clinical terms, two different personalities. Aside from the obvious fact that this is an article about basketball and not mental illness, there is nothing about this headline or article that even metaphorically suggests the symptoms of schizophrenia. Thus, not only is the use of this adjective inappropriate in that it is insulting to those who have this disorder, it is also incorrect because, if anything, it refers more to DID than it does to schizophrenia.

What are the actual symptoms of schizophrenia? To meet the diagnosis for schizophrenia in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-IV-TR (Am. Psychiatr. Assoc. 2000), a person must demonstrate at least two symptoms from among five domains (hallucinations, delusions, disorganized speech, disorganized behavior or catatonia, negative symptoms) for at least one month. In addition, these symptoms must persist, either in their active phase or in attenuated form for six months. The DSM-5 (scheduled for release in May 2013; Am. Psychiatr. Assoc. 2010) will contain minor changes to the diagnosis, including the requirement that one of the two symptoms be hallucinations, delusions, or disorganized speech, and a clearer specification of negative symptoms. Negative symptoms are labeled as such because they involve absences of or deficits in something that is typically present among healthy people. For example, people with the symptom of blunted affect do not show many outward expressions of emotion. Although consensus in the field suggests five negative symptoms, including blunted affect, alogia, asociality, anhedonia, and avolition (Kirkpatrick et al. 2006), several studies have demonstrated that these five symptoms can be more parsimoniously explained by two factors: diminished expression (blunted affect) and diminished motivation and pleasure (avolition, anhedonia, asociality) (Blanchard & Cohen 2006, Horan et al. 2011, Kring et al. 2012). It is worth noting here that both of these factors involve deficits in emotion. That is, diminished expression refers to a deficit in the outward expression of emotion via the face and voice, and diminished motivation and pleasure refers to a deficit in pleasure and goal-directed behavior across social, work, and recreational life domains (Horan et al. 2011). The DSM-5 will characterize the negative symptoms in this two-factor manner, referring to them as restricted affect and avolition/asociality.

The lifetime prevalence of schizophrenia is nearly one percent, and it affects men slightly more often than women (Kirkbride et al. 2006, Walker et al. 2004). The typical age of schizophrenia onset is between 18 and 25, usually somewhat earlier in men than in women. Perhaps related to their later age of onset, women with schizophrenia tend to have an overall better prognosis than men, including better functioning prior to the onset of the first episode, fewer hospitalizations, fewer negative symptoms, better response to treatment, and better social and occupational functioning (Canuso & Pandina 2007, Leung & Chue 2000).

WHAT IS EMOTION?

Emotions cannot be identified by any one characteristic. Instead, multiple components comprise emotions, including expression, experience, and physiology. The assumption embedded within many theoretical views of emotion is that the different components of a specific emotion (e.g., fear, anger) cohere or occur together and that each emotion has a distinct pattern of coherence (e.g., Levenson 2011), just as the different musical instruments cohere in a symphony albeit distinctly, depending upon the particular piece.

Anhedonia:
diminished interest
and pleasure from
things that at one time
brought pleasure

A funny thing happened on the way to emotion component coherence, however. It turns out that coherence is more the exception than the norm (e.g., Barrett 2006, Bradley & Lang 2000, Mauss et al. 2005), and the evidence in favor of distinctiveness is mixed, though hotly debated (e.g., Barrett 2011b, 2012; Levenson 2011; Shariff & Tracy 2011a,b). There are a number of methodological reasons why particular emotion components may not cohere in a given study, including sample characteristics, emotion elicitation methods, emotion component measurements, or data analytic techniques. But setting these explanations aside for the moment, recent theoretical models embrace the lack of coherence as a tenet of the theory. For example, Barrett (e.g., 2006, 2009, 2011a) has argued that emotion consists of two key features: (a) a core affect system, which is reflected as neurophysiological states that are omnipresent indicators of a person's relationship to his or her environment at any given time and that are experienced as feelings of pleasure or displeasure, and to a lesser extent arousal or activation; and (b) a conceptual knowledge system about emotion that reflects the stored experiences and learning about what constitutes emotion. People categorize their core affect as fear, happiness, anger, etc. at any given moment to both make sense of what they are feeling and to compel them to appropriate action or communicate to others. This theoretical model of emotion, known as the conceptual act model, predicts variability in emotion component coherence given that people will vary in their core affect and in their conceptual knowledge about emotion. This model has important implications for understanding emotion deficits in schizophrenia, a topic we revisit in the next sections.

Emotions also serve many important functions. Most theorists agree that emotions have developed through the course of human evolutionary history to prepare organisms to act in response to a number of environmental events and challenges. That is, emotions help us to recognize problems, open us to new experiences, propel us to action, strengthen social relationships, and help us to make decisions. Under most circumstances, emotions serve a number of important intra- and interpersonal functions (e.g., Keltner & Kring 1998, Lang et al. 1990, Levenson 2011, Shariff & Tracy 2011a). Unfortunately, emotion deficits in schizophrenia often interfere with the achievement of these emotion-related functions. For example, the absence of facial expressions in a person with schizophrenia may provoke a negative response from others (Krause et al. 1992), thus negatively impacting social relationships and interactions (e.g., Hooley et al. 1987).

EMOTION DEFICITS IN SCHIZOPHRENIA: A TALE OF TRANSLATIONAL RESEARCH

... it has appeared both from crude observation and from detailed study of the facial expression that the alleged indifference, apathy, and emotional disharmony of the schizophrenic is more a matter of impression than correct evaluation of the inner experience of such a patient. It has followed that the study of such inner affective experiences by positive objective means seems urgently indicated if the nature of the schizophrenic processes is to be elucidated. (Harry Stack Sullivan 1927)

Observations of emotion deficits in schizophrenia are not new. Indeed, Sullivan, Bleuler, and Kraepelin all wrote eloquently about different problems in emotion as part of their careful clinical case histories and books. The apparent disconnect between the outward expression component of emotion and the experience or feeling component that Sullivan referred to in the quote above is also consistent with observations from family members. For example, Bouricius (1989) presented samples of her son's diary writings that articulated the experience of clear and complex emotions despite a limited repertoire of outward expression. Quite reasonably, she wondered: "Is it possible that many persons who suffer from schizophrenia and exhibit negative symptoms are actually experiencing strong emotions that they are unable or afraid to express?" Unfortunately,

psychological research addressing this question lagged behind the astute observations of the early theorists, clinicians, and family members.

In our view, what has propelled research on emotion in schizophrenia toward the answers to this question has been the development and translation of methods and theories in basic affective science to the study of schizophrenia. The study of emotion was neglected for years, kept tucked away in the back of the research closet until the right set of ideas helped bring it out to the forefront. The pioneering work of researchers such as Paul Ekman and Carroll Izard in the 1960s paved the way for emotion research to become well accepted and integrated into psychology and neuroscience and translated to the study of schizophrenia. Indeed, the explosion of brain imaging techniques has further advanced our understanding of how the brain responds when someone is experiencing emotion (Barrett et al. 2007), and taken together, we now know a great deal about emotional behavior and perception as well as the neural underpinnings in healthy people (e.g., Lindquist et al. 2012). This type of research has greatly expanded our understanding of the nature of emotion deficits in schizophrenia.

Before turning to a review of the current literature covering emotion deficits in schizophrenia, it is useful to first clarify some terminology. First, many studies in the literature purport to study emotion processing; however, this is a term that is in desperate need of meaning. It is used to describe all sorts of studies and tasks, from the study of facial expressions following the presentation of emotionally evocative film clips, to the recognition of different emotions, to the reading of emotional words. Not only are these tasks different, but they recruit different brain regions in the service of performing them (Wager et al. 2008). The term emotion processing is just as vague as information processing, an older term in cognitive science that has been replaced with more precise terms and constructs (e.g., cognitive control, episodic memory) thanks to advancements in cognitive neuroscience. Similarly, as affective science has advanced, it is necessary to be more precise in describing the terms and constructs related to emotion processing. As the current review points out, certain areas of emotion perception and response are well preserved in schizophrenia while others are disrupted, and the use of vague terms such as emotion processing only serves to blur the landscape of emotion deficits in schizophrenia.

We have argued elsewhere that it is helpful to make distinctions between the types of studies and types of dependent variables or measures of emotion that are commonly employed to study emotion in schizophrenia (Kring & Moran 2008). Five types of studies are most often utilized to examine emotion in schizophrenia: (a) studies that elicit emotion responses from participants (e.g., presenting emotionally evocative pictures and assessing reports of emotional experience); (b) studies that assess perception (recognition) of affective stimuli (e.g., presenting pictures of faces and assessing accuracy of emotion expression identification); (c) naturalistic studies that assess emotion in participants' natural environments (e.g., experience sampling studies); (d) studies that assess trait or individual differences in emotion (e.g., collecting self-report measures of positive and negative affect); and (e) studies that assess the impact of emotion on some other process (e.g., assessing how the emotional valence of words impacts the ability to recall the words). Although many studies combine more than one of these methods, we believe it is helpful to keep in mind these distinctions in order to more readily focus on what aspect(s) of emotion is being manipulated and assessed (Kring & Moran 2008).

These types of distinctions are also important for developing a more specific understanding of the neural systems that support emotion perception and response. Recent meta-analyses in healthy people and people with schizophrenia point to dissociations in relative brain region activations, depending upon the emotion task that is used (Lindquist et al. 2012, Taylor et al. 2002, Wager et al. 2008). Specifically, studies of emotion perception in healthy people demonstrate relatively more robust activation in areas such as amygdalae, parahippocampal cortex, pregenual cingulate,

Emotion perception: identifying emotion in another person or stimulus; most typically studied using pictures of faces displaying different emotion expressions

Emotion expression: the outward display of emotional feelings across different channels, including the face, voice, and body

and dorsal portions of the inferior frontal gyrus. By contrast, elicitation studies that assess participants' reports of emotional experience demonstrate relatively greater activation in other areas, such as prefrontal cortex (ventromedial, orbitofrontal, dorsolateral), anterior insula, medial temporal lobe, ventral inferior frontal gyrus, and temporal pole (Wager et al. 2008). Furthermore, tasks that involve less cognitive demand are more likely to activate amygdalae, but studies that involve more cognitive demand, including judgments related to the self, are more likely to activate ventromedial prefrontal cortex (e.g., Ochsner 2008). In short, although different tasks do not recruit entirely different brain regions, the relative emphasis of recruitment differs depending upon the task.

Emotion Perception in Schizophrenia

Understanding emotional facial expressions is important in daily life. Consider the following scenario: You find yourself meeting someone for the first time, and you want to ask for help. How do you know if they are trustworthy and likely to help? You may scan their face for hints of a smile or the absence of a scowl, thus suggesting to you that the person may be friendly. To navigate the social world, we must be able to perceive emotion in others, and we must do so by also integrating the surrounding context and situation.

Emotion perception, particularly facial expression perception, has been studied in schizophrenia for over 40 years. Explicit facial expression perception tasks have typically taken one of two forms. In identification tasks, the researcher presents pictures of faces displaying different emotions (e.g., happiness, fear, surprise, disgust) and asks participants to identify the emotion being displayed, sometimes with a label provided, sometimes without. In discrimination tasks, the researcher presents two pictures of emotional faces side by side and asks participants if the people in the pictures are showing the same or different emotions. Early studies were hampered by small sample sizes and methodological inconsistencies that made it difficult to determine whether people with schizophrenia had trouble perceiving emotion in others (Edwards et al. 2002). Recent meta-analyses, however, have provided a much clearer picture, indicating that people with schizophrenia have deficits in facial emotion identification and discrimination tasks (Kohler et al. 2010) as well as with facial perception tasks more generally (Chan et al. 2010).

These findings thus beg the question: Do people with schizophrenia have problems with perceiving emotion or with perceiving faces, one of the most complex visual items we must interpret in daily life? Overall, the evidence suggests that people with schizophrenia do not have a specific deficit in perceiving emotion on the face but rather a more generalized deficit in facial processing (Chan et al. 2010, Kerr & Neale 1993). What remains to be resolved, however, is whether problems in face perception reflect deficits in low-level sensory processes underpinning more basic perception or higher-level processes requiring integration of perception with surrounding contextual information in order to make sense of what is being observed. Unfortunately, only a handful of studies have identified face or emotion perception deficits using paradigms that keep sensory/perceptual aspects similar across different contextual demands (e.g., Leitman et al. 2011), thus making it more feasible to tease apart lower level deficits from higher level deficits that require integration of context with perceptual processing to make a judgment about emotion. Only recently have investigators begun to examine emotion perception in other channels besides the face. For example, in a particularly clever study, Van den Stock et al. (2011) found that people with schizophrenia had trouble identifying emotion in the body (faces were blurred) compared to people without schizophrenia.

Despite this evidence of widespread deficits in emotion perception, there are nevertheless areas of emotion perception that appear to be preserved in schizophrenia. For example, people with

schizophrenia do not exhibit a deficit in implicit emotion perception tasks, such as priming (e.g., Hoschel & Irle 2001) or incidental labeling tasks (Van't Wout et al. 2007), or if asked to make a simple target versus nontarget distinction between two emotional faces (Gur et al. 2007). In these types of tasks, emotion information signaled via the face is influencing subsequent behavior among people with schizophrenia in the same way that it does for people without schizophrenia. Why might this be? We propose that people with schizophrenia will be more likely to exhibit emotion perception deficits on tasks that require integration of context with emotion perception, given their broader deficit in the integration of prefrontally mediated cognitive control mechanisms [e.g., dorsolateral prefrontal cortex (DLPFC), ventromedial PFC] with brain regions associated with emotion (e.g., amygdala) (for review, see Lesh et al. 2011).

What do we mean by context? Most broadly, context can refer to information that is not present in the stimulus but can be used to determine the most task-appropriate response to the current stimulus (e.g., the social scene that the smiling face is embedded in, or the participant's current emotional state). We have found it useful to think about contextual influences on emotion perception in schizophrenia using the rubric of Barrett et al. (2011), who distinguish stimulus-based contexts (e.g., social situation, visual scenes, voice or posture of the person depicting emotion on the face) and perceiver-based contexts (e.g., perceivers' knowledge of emotion, such as emotion labels, and the perceiver's emotional state). An example of stimulus-based context might be perceiving the face of an excited baby as distressed if embedded in a scene of being left alone but perceiving the face as happy if in a scene of a peekaboo game. The less obvious (but still crucially important) type of context is perceiver-based context, which includes the knowledge that we gain about facial expressions and the words to label them over years of experience. This type of context allows us, for example, to compare a to-be-appraised face with our memories of other emotion-expressing faces that we have encountered.

Indeed, one of the most exciting current ideas in affective science research is this notion of perceiver-based context. Emotion labels provide internal contextual information insofar as these words themselves can influence the emotional state of the perceiver (Barrett et al. 2007). One interesting hypothesis that flows from the studies of emotion labels as context is the idea that emotion words not only create a context by activating stored conceptual knowledge about what that emotion label means (i.e., our years of experience with and memories of emotion faces and words), but that these words shape what we actually see (Barrett et al. 2007). For example, an ambiguous facial expression paired with the word fear is more likely to be remembered as fearful, but the same ambiguous face paired with the word happy is more likely to be remembered as happy (Halberstadt & Niedenthal 2001). Similarly, scenes that are paired with emotion faces are more readily recalled when participants were given emotion words to identify the emotion on the face (Barrett & Kensinger 2010). The requirement of assigning emotion labels to faces is standard in many emotion perception tasks, and people with schizophrenia do not perform well on these types of tasks. This suggests the intriguing notion that the deficit might not be in perceiving emotion *per se*, but in the integration of context (prior knowledge about emotion faces and labels) with the faces presented in the task. Indeed, one of the key domains for future research in schizophrenia is in the area of contextual integration and perception (Kring & Campellone 2012).

Emotion Responding in Schizophrenia

Translating affective science theory and methods to schizophrenia began in earnest in the late 1980s/early 1990s. These studies examined emotional responding in schizophrenia by assessing one or more components of emotion response as defined by affective science researchers (i.e., expression, experience, physiology). Beginning in the late 1990s and continuing today, researchers

began examining neural correlates of emotional responding using functional magnetic resonance imaging (fMRI). Over the past 30 years of research, we have learned a great deal about where people with schizophrenia do and do not have deficits in the various components of an emotional response.

Emotional expression. One of the most well-replicated findings in the literature on emotional responding in schizophrenia is that people with schizophrenia are much less expressive (both facially and vocally) than people without schizophrenia in response to a variety of emotional situations and stimuli. Studies have used many different methods to elicit emotion, including film clips, pictures, music, foods, odors, and social interactions, as well as many methods to measure facial expression, including observational coding systems, acoustical analyses of speech, and electromyography (EMG). Across all types of methods, the consistency of the findings is striking. Compared to people without schizophrenia, people with schizophrenia display fewer positive and negative facial expressions in response to emotionally evocative film clips, foods, and social interactions (for a review, see Kring & Moran 2008). One study did not find group differences in embarrassment following presentation of a loud, unexpected noise while someone was watching, but the overall levels of embarrassment in both groups were extremely low (Bailey et al. 2009). Importantly, diminished expression is observed among people with schizophrenia both on (e.g., Aghevli et al. 2003, Berenbaum & Oltmanns 1992) and off (Kring & Earnst 1999, Kring & Neale 1996) medication.

Although people with schizophrenia are less outwardly expressive than people without schizophrenia, their facial muscles are still responding in a way that is consistent with the valence (positive, negative) of the stimuli, at least in studies of spontaneous expression. A few studies have assessed facial muscle movement using EMG, and these studies find that people with schizophrenia contract their zygomatic muscle (cheek muscle that is contracted when smiling) more when viewing positive stimuli compared to when viewing negative stimuli. Similarly, people with schizophrenia contract their corrugator muscle (brow muscle that is contracted when frowning) more when viewing negative stimuli than when viewing positive stimuli (Kring & Earnst 2003; Kring et al. 1999; Wolf et al. 2004, 2006). Interestingly, the timing of these muscle contractions may be different for people with and without schizophrenia. Varcin and colleagues (2010) found that people with schizophrenia exhibited less zygomatic and corrugator activity than the control group in responses to pictures of faces, but only during the first 500 ms of picture presentation. Thus, people with schizophrenia are exhibiting emotionally congruent, albeit very subtle, nonobservable expressions when presented with emotionally evocative stimuli, though the timing of these expressions may be different from that of people without schizophrenia.

Why are these expressions so subtle? As noted previously, it does not seem to be a medication side effect, as this same pattern of expression is observed in the same group of people, both on and off medication (Kring & Earnst 1999). Nevertheless, many medications used to treat schizophrenia can unfortunately dampen all sorts of muscle activity, including facial muscle activity (sometimes referred to as akinesia), and thus medications cannot be ruled out entirely here. Might people with schizophrenia be lacking in expressiveness because of a broader social skills deficit? Indeed, expressive behavior is part of any successful repertoire of social skills, and people with schizophrenia have a number of deficits in this area (e.g., Harvey et al. 2007, Penn et al. 2002). However, diminished expression is observed among people with schizophrenia in both minimally and maximally social situations (e.g., Aghevli et al. 2003, Salem & Kring 1999), and emotion expression is not strongly correlated with social skills, suggesting that a social skills deficit cannot entirely account for why people with schizophrenia exhibit so few outward emotion expressions.

Another avenue for exploring emotion expression has been to assess whether people with schizophrenia can produce or pose different emotion expressions following instructions to do

so. In these studies, participants are either given emotion words or pictures of people exhibiting different emotion expressions (e.g., happy, sad, angry, disgusted, surprised) and asked to generate the emotion expression themselves. Findings from these studies are mixed, with some indicating that people with schizophrenia do not pose any emotion expressions as accurately or intensely as do people without schizophrenia (e.g., Gaebel & Wölwer 1992, Trémeau et al. 2005) and others indicating that people with schizophrenia do not pose some expressions as intensely and accurately (surprise, sadness) but are just as accurate and intense in others (happiness, disgust, fear, anger) (Putnam & Kring 2007). Once again, medication may account for some of the inconsistencies as well as the way in which the cues and instructions for posing were presented (pictures, words, or both).

In summary, people with schizophrenia are markedly less observably expressive than people without schizophrenia, even though they spontaneously contract their facial muscles consistent with the valence of what they are responding to. One of the most interesting aspects of emotional responding in schizophrenia is that this lack of outward expression belies the internal experience of emotion as well as physiological and brain activation responses, the areas we turn to next.

Emotional experience. Despite the overwhelming evidence that people with schizophrenia are less expressive than people without schizophrenia, there is also fairly consistent evidence that people with schizophrenia report experiencing similar and in some cases greater amounts of emotion than those without schizophrenia (Cohen & Minor 2010, Kring & Moran 2008). There is greater variability in the findings on emotional experience than the findings on emotion expression, but this variability likely reflects the intermixing of deficits in cognitive processes needed to support emotion rather than deficits in emotion responding per se.

To assess how people feel in response to evocative stimuli or in the course of daily life most often requires collecting self-report questionnaires or asking for verbal reports of emotional experience. Some may question whether individuals with schizophrenia can accurately and reliably report on their feelings given the cognitive and language disturbances that often accompany the disorder. However, convergent findings point to the ability of people with schizophrenia to provide reliable and valid reports of their emotional experience as evidenced by high internal consistency and test-retest reliability of emotion experience reports, even when assessments occur across changes in symptoms and medication status (Kring & Earnst 1999). Furthermore, multidimensional scaling techniques have revealed that people with schizophrenia represent emotion in the same two-dimensional structure (valence and arousal) as do people without schizophrenia (Kring et al. 2003), thus bolstering confidence in self-reports of emotional experience in people with schizophrenia.

The most common method used to study emotion experience as it naturally unfolds in daily life has been the experience sampling method (ESM). In a typical study, participants report on thoughts, feelings, and behaviors several times a day for a week or longer. Although the demands of these studies can be great, there is convincing evidence that people with schizophrenia can rise to the challenge and complete the ratings several times a day for a week or more (Granholm et al. 2007, Oorschot et al. 2009). Although the primary dependent variables obtained in an ESM study are self-report, because these data are collected “in the moment,” they do not suffer from biases associated with retrospective reports (Bolger et al. 2003). In analyses explicitly designed to assess emotional experience in the course of daily life, two studies have found that people with schizophrenia report comparable amounts of positive and negative emotion compared to people without schizophrenia (Gard & Kring, 2009, Oorschot et al. 2012), and one study found that people with schizophrenia reported experiencing more negative emotion and less positive emotion than did people without schizophrenia (Myin-Germeys et al. 2000) is particularly noteworthy because the study included a large sample (149 people with schizophrenia or schizoaffective disorder

Emotion experience:
the feeling component
of an emotion response

and 143 healthy controls) and parsed emotion experience in response to identified pleasant and unpleasant events rather than simply lumping all events and experiences together. Although people with schizophrenia reported engaging in fewer pleasant events in daily life compared to the control group, they reported experiencing comparable amounts of pleasant emotion when they actually participated in pleasant events, including events involving other people.

Another recent naturalistic study compared reports of emotion experience obtained during the course of a one-week experience sampling study with retrospective reports of the prior week's emotion experience (Ben-Zeev et al. 2012). That is, people with and without schizophrenia first participated in an ESM study where they reported on their feelings six times day for one week. On the eighth day, they were asked to think back on the week and report on the feelings they experienced in summary fashion. Although both groups reported greater emotion experience in their retrospective reports than in their daily, in-the-moment reports, the mean retrospective rating was not greater than the highest rating (peak experience) during the ESM week, suggesting that there was not a global overestimation effect occurring. Most interestingly, the people without schizophrenia did more overestimating than the people with schizophrenia did. Stated differently, the retrospective reports of people with schizophrenia were closer to their daily reports than were the controls' reports. Thus, not only are people with schizophrenia reporting comparable amounts of emotion relative to people without schizophrenia, but they also appear to do so accurately and consistently in response to evocative stimuli, in the course of daily life, and when reflecting back on the prior week. This finding ought to bolster confidence in interpreting responses to clinical interviews, where people with schizophrenia are often asked to report on their symptoms and emotions in the prior week. It is important to note that there was a large sex difference in the samples—there were more women than men in the control group and more men than women in the schizophrenia group. Prior research in affective science has indicated that women report greater emotional experience on retrospective measures than men but do not differ in momentary reports of emotional experience (e.g., Barrett et al. 1998). Thus, the greater discrepancy between retrospective and daily emotion experience ratings for controls may reflect a gender difference as much as or more than a difference between people with and without schizophrenia. Nevertheless, that the schizophrenia group was less prone to overestimation is still an important finding that could productively be followed up with a larger sample of women with schizophrenia.

Studies that have assessed emotional experience in response to evocative stimuli typically find that people with schizophrenia report feelings that are (*a*) comparable in intensity and frequency to people without schizophrenia and (*b*) consistent with the valence of the stimuli. That is, they report experiencing more negative emotion in response to negative stimuli than positive stimuli and vice versa in the same frequency and intensity as do people without schizophrenia. Indeed, across a variety of stimuli (pictures, films, odors), the majority of studies find that people with schizophrenia do not differ from people without schizophrenia in their reports of experienced emotion.

An important conceptual and methodological distinction to emphasize is the difference between rating feelings in response to a stimulus and rating the stimulus itself. For example, a study may present emotionally evocative pictures and instruct participants to rate how they feel when watching these pictures, or the study may instruct participants to rate the stimulus properties. The rating scale provided for such judgments can be identical or not—participants can rate how unpleasant they feel when viewing a burn victim and they can rate the unpleasantness of the picture itself. This is not only important conceptually, but evidence from affective neuroscience indicates that different brain regions are recruited for these different types of ratings. For example, reporting feelings is associated with activation in dorsal/rostral areas of medial prefrontal cortex (mPFC), whereas rating the affective properties of a stimulus is associated with activation in more ventral portions of mPFC (Ochsner et al. 2004, Olsson & Ochsner 2008, Wager et al. 2008).

Interestingly, rating the feelings of others also activates similar regions in mPFC even as there are distinctions in regions of mPFC activation associated with judgments about the self and others' feelings (Denny et al. 2012).

Despite the number of studies reporting similarities in emotion experience between people with and without schizophrenia, an intriguing group difference has been repeatedly reported. Specifically, a number of studies have found that people with schizophrenia report experiencing emotions that were not necessarily what the stimuli were designed to elicit, and this seems to be particularly true for neutral and positive stimuli. Indeed, people with schizophrenia report experiencing more negative emotion to these stimuli than do people without schizophrenia (e.g., Cohen et al. 2009, Trémeau et al. 2009, Ursu et al. 2011; for meta-analysis, see Cohen & Minor 2010).

What might account for the mismatch between the valence of reported feelings and stimuli? A few ideas have been suggested, yet no clear answers have yet emerged. First, it may be the case that only some people with schizophrenia exhibit this mismatch pattern. Using cluster analysis, Strauss & Herbener (2011) identified a subgroup of people who reported experiencing more negative emotion to both positive and negative pictures than either people without schizophrenia or another group of people with schizophrenia. This group had more negative symptoms and poorer overall functioning, suggesting that the people more likely to evidence a mismatch between the valence of feelings and stimuli may be those who have a more severe form of the illness. Second, reporting on emotions that are incongruent with a stimulus (e.g., how unpleasant do you feel after viewing a smiling baby?) requires a good deal of cognitive control. For one, the stimulus is typically no longer in view, and thus participants must maintain their experience even in its absence. Additionally, reporting on stimulus-incongruent feelings requires drawing upon representations of emotion that are less readily available in that moment in order to overcome prepotent responding, which in this case would be the valence that matches the stimulus. Broadly speaking, cognitive control is supported by the DLPFC (e.g., Miller & Cohen 2001), and in one recent study, DLPFC activation was correlated with incongruent ratings for people without schizophrenia but not for those with schizophrenia, suggesting that the DLPFC supports these more inaccessible experience ratings (Ursu et al. 2011). Third, some investigators have suggested that the mismatch between the valence of reported feelings and stimuli reflects greater ambivalence on the part of people with schizophrenia. In this case, ambivalence is defined as unpleasantness felt in response to pleasant stimuli and pleasantness felt in response to unpleasant stimuli (Trémeau et al. 2009) or as the consequence of being unable to inhibit ever-present negative affect, even in the presence of something positive or neutral (Cohen et al. 2010, Horan et al. 2006a). Indeed, people with schizophrenia generally report experiencing more negative affect and less positive affect on trait measures than people without schizophrenia report (for review, see Horan et al. 2008), and the greater effort required for people with schizophrenia to complete some laboratory tasks may engender greater negative affect (e.g., frustration, anxiety, confusion). Finally, Strauss & Gold (2012) have suggested that the mismatch in valence ratings and stimuli may reflect a difficulty in accessibility of emotion that is compounded by cognitive deficits, including episodic memory. More specifically, when asked to report on their noncurrent feelings (i.e., reporting on unpleasant feelings when currently feeling pleasant), people with schizophrenia must draw upon past situations and beliefs—including beliefs about the self—that may be dysfunctional and thus unable to support the accessibility of these feelings.

These are all intriguing hypotheses that lend themselves to testing. For example, if an ever-present negative affective state is responsible for greater reports of negative emotion in response to positive stimuli, it would seem possible to dampen the baseline negative affect and thus diminish the mismatch between valence of feelings and stimuli. Similarly, if cognitive control or accessibility supports the reporting of these mismatch feelings, it should be possible to generate situations

where cognitive control or accessibility demands are altered, and these manipulations should then impact the degree to which people with schizophrenia evidence a mismatch between the valence of their feelings and the valence of the stimuli.

Emotion and psychophysiology. To our knowledge, only one study has assessed expression, experience, and autonomic physiology in an elicitation study. Kring & Neale (1996) found that people with schizophrenia exhibited greater skin conductance reactivity than people without schizophrenia in response to emotionally evocative film clips, even though they displayed very few observable facial expressions. Additional studies have assessed skin conductance responses to emotional pictures (but not other components of emotion response), and the findings are mixed, with two studies finding greater skin conductance reactivity among people with schizophrenia than those without (Williams et al. 2004, 2007), two studies finding no group differences (Hempel et al. 2005, Volz et al. 2003), and two studies finding less reactivity among people with schizophrenia (Schlenker et al. 1995, Taylor et al. 2002). In addition to methodological differences, it seems that individual differences in skin conductance reactivity are likely relevant for understanding the disparate findings.

Emotional responses have also been examined using the affective startle modulation paradigm. In this paradigm, startling noises or puffs of air are intermittently introduced while participants are viewing emotionally evocative stimuli, most often pictures. The noise or puff of air elicits a reflexive startle response, and one measurable component of this response is the magnitude of an eyeblink (i.e., the contraction of the orbicularis oculi muscle). The magnitude of the startle responses is greatest while viewing negatively valenced material and smallest while viewing positively valenced material (Lang et al. 1990). All five studies that have assessed affective modulation of the startle response have found that people with schizophrenia exhibit the same pattern of startle modulation as those without schizophrenia (Curtis et al. 1999, Kring et al. 2011, Schlenker et al. 1995, Volz et al. 2003, Yee et al. 2010). Thus, people with schizophrenia exhibit stronger eyeblink responses when viewing negative pictures compared to positive pictures.

In summary, studies of emotion physiology in schizophrenia yield results that are consistent with studies of emotional experience. That is, people with schizophrenia show comparable responses to people without schizophrenia, at least in the startle responses, though there may be particular groups of people with the illness who show either under- or overactivation of skin conductance responding.

Emotion and brain activation. Neuroimaging studies using positron emission tomography (PET) or fMRI during presentation of emotionally evocative stimuli have been a more recent addition to studies of emotion deficits in schizophrenia, with most of these conducted in the past 10 years. As is the case with other studies of emotion in schizophrenia, translational research on emotion and brain activation has helped to guide interpretation of results from studies in schizophrenia. Indeed, studies of facial expression perception find relatively more robust brain activation in areas such as amygdalae, parahippocampal cortex, pregenual cingulate, and dorsal portions of the inferior frontal gyrus. In contrast, studies that present evocative stimuli and ask participants to report on their feelings find relatively greater activation in other areas, such as prefrontal cortex (ventromedial, orbitofrontal, dorsolateral), anterior insula, medial temporal lobe, ventral inferior frontal gyrus, and temporal pole (Wager et al. 2008).

In schizophrenia, there are mixed findings, with some studies reporting no differences in brain activation in areas associated with emotion (e.g., amygdala, medial and dorsal PFC) between people with and without schizophrenia during presentation of emotionally evocative stimuli (e.g., Ursu et al. 2011), and others reporting diminished activation in people with schizophrenia

compared to people without schizophrenia (e.g., Gur et al. 2007). Interestingly, studies that involve tasks designed to elicit emotional experience and then ask participants to rate their experience are less likely to find differences in brain activation between groups (though this depends in part upon the type of contrast that is selected) (e.g., Dowd & Barch, 2010, Ursu et al. 2011), whereas studies that involve emotion perception tasks and ask participants to label or discriminate facial emotion are more likely to observe group differences (e.g., Gur et al. 2007). This again highlights the important methodological distinction between rating feelings and rating stimuli.

Three meta-analyses have been published in the past two years (Anticevic et al. 2012, Li et al. 2010, Taylor et al. 2012), and these have been able to include a relatively small number of studies (between 17 and 35). Li and colleagues (2010) examined 17 studies of emotion perception, 13 of which reported greater relative activation in people without schizophrenia compared to people with schizophrenia. Studies included were those that presented faces as stimuli and required either the identification/discrimination of the face or passive viewing followed by stimulus rating. For this meta-analysis, the authors used the activation likelihood estimation (ALE) method (Laird et al. 2005), which identified clusters of peak activations in particular brain regions in individual studies. Three clusters were identified as having greater relative activation for people without schizophrenia, including bilateral amygdalae, bilateral parahippocampal gyrus/amygdala, right superior frontal gyrus, and right middle occipital gyrus. However, the ALE method does not allow for a determination of the magnitude of activation differences.

The meta-analyses of Taylor et al. (2012) and Anticevic et al. (2012) included a broader range of studies, and despite using different meta-analytic techniques, reported consistent findings. Both of these meta-analyses focused on elicitation studies of negative emotion, studies that included contrasts between conditions (emotion condition–neutral condition as well emotion condition–baseline), included between-group comparisons, and used standardized coordinates. Anticevic et al. focused primarily on the amygdala, whereas Taylor et al. examined whole-brain activation. Taylor et al. (2012) reported that people without schizophrenia exhibited greater amygdala activation than people with schizophrenia, but only in studies of emotion perception (consistent with Li et al. 2010) and not in studies of emotion experience (i.e., studies where participants were asked to report on their feelings in response to the stimuli).

With respect to amygdala activation, an interesting and consistent result was found across the two meta-analyses. People without schizophrenia showed greater amygdala activation than did people with schizophrenia, but only for contrasts that subtracted activation during a neutral condition from activation during a negative condition (Anticevic et al. 2012, Taylor et al. 2012). Rather than contrasting activation during a neutral condition from activation in a valenced condition, Anticevic et al. analyzed direct emotion contrasts (i.e., positive condition–baseline) and found no between-group differences in amygdala activation. Results from both of these meta-analyses suggest that underactivation of amygdala in people with schizophrenia in response to negative stimuli (pictures, faces, smells) may reflect overactivation to the neutral stimuli rather than underactivation to negative stimuli. These findings are consistent with the emotional experience findings, where people with schizophrenia often report experiencing more negative emotion in response to neutral stimuli (Cohen & Minor 2010).

What about other brain regions? The meta-analysis of Taylor et al. (2012) revealed other regions of activation that distinguished people with and without schizophrenia, including less activation in people with schizophrenia in the anterior cingulate cortex (ACC), a region activated more strongly in emotion perception tasks, and dorsal medial and dorsolateral PFC, regions activated during emotion and cognitive tasks. Less activation was also observed in more posterior brain regions (occipital pole, fusiform gyrus) among people with schizophrenia, but this may reflect

neural correlates of processing complex facial stimuli more than emotion per se. Taylor et al. also examined the potential moderating effects of task type on observed brain activation differences. As such, they compared implicit tasks (e.g., rate the gender of face) with explicit tasks (e.g., rate your emotional experience) and discovered that the greater activation among people without schizophrenia in amygdala and medial PFC was found primarily in implicit tasks. This raises the question as to whether these nonemotion-focused aspects of the task may have contributed to the different patterns of activation.

In summary, although differences in brain activation have been observed in regions associated with emotion, such as amygdala, these may reflect responses to neutral rather than emotional stimuli. Taken together, fMRI and PET studies of amygdala activation reveal a pattern not dissimilar from studies of emotional experience; namely, that people with and without schizophrenia show comparable brain activation, at least in response to negative emotional stimuli (relative to baseline) that they are asked to report their feelings in response to. Despite these similarities, there also appear to be differences in the activation of regions that are also associated with cognitive control, including ACC and DLPFC. These findings point to the obvious but important notion that cognition and emotion cannot be readily separated (in either behavior or brain) in most laboratory tasks as well as in daily life. Indeed, cognitive and affective neuroscience research with healthy people has demonstrated that the brain is not simply divided into regions specific to our psychological concepts, such as cognition and emotion. Instead, overlapping brain regions support thinking and feeling in interesting and complex ways (e.g., Barrett 2009). Understanding cognition and emotion connections is also at the forefront of research on schizophrenia (Taylor & Liberzon 2007).

AFFECTIVE SCIENCE, MEET COGNITIVE NEUROSCIENCE

The evidence reviewed thus far suggests that people with schizophrenia have a deficit in the expression of emotion but not necessarily in the experience of emotion, at least when the experienced emotion is congruent with the evocative stimulus. This evidence is inconsistent with research indicating that nearly three-quarters of people with schizophrenia have the clinical symptom of anhedonia (Horan 2006b). How can we make sense of this discrepancy? On the one hand, people with schizophrenia report experiencing as much positive emotion as people without, while on the other hand, people with schizophrenia also report and are rated as having anhedonia. What gives?

Drawing upon research on the reward system in humans and animals, we (Kring & Caponigro 2010) have argued for the importance of characterizing the temporal course of emotion to distinguish anticipatory from in-the-moment, or consummatory, responses (see **Figure 1**). When people with schizophrenia are presented with pleasurable stimuli either in a lab or in daily life, they can and do derive pleasure from these experiences. However, people with schizophrenia are less likely to anticipate that future events will be pleasurable and are less likely to experience pleasure in anticipation of things to come, and thus may be less likely to seek out pleasurable experiences (Gard et al. 2007). Other behavioral, psychophysiological, and fMRI studies also find difficulties among people with schizophrenia in what we refer to as anticipatory pleasure (e.g., Juckel et al. 2006, Trémeau et al. 2010, Wynn et al. 2010).

The ability to anticipate whether something in the future will be pleasurable requires a cornucopia of cognitive skills, such as imagination, reflection, drawing upon past experiences, and maintaining an image or emotional state. Thus, the latest wave of research on emotion in schizophrenia explicitly integrates emotion and cognition. For example, consider the problem of what to have for lunch. You consider a healthy salad, which may then lead you to call forth a past experience of a delicious salad you've had from the nearby sandwich shop (activating a representation and

Temporal experience of emotion

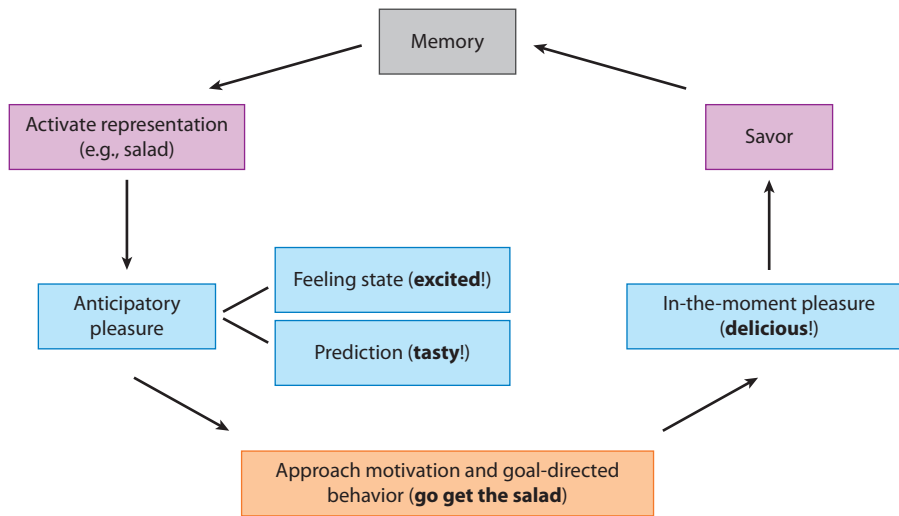


Figure 1

Model of the temporal course of emotion.

holding this in working memory). This prompts you to predict that the salad will be tasty and enjoyable, and indeed you experience pleasure now, knowing you will soon be eating the salad (anticipatory pleasure). These processes will support your motivational system such that you will walk to the shop and get the salad (approach motivation and behavior), and once you eat it, you will experience in-the-moment, or consummatory, pleasure. You will savor (maintain) the pleasure from the salad, and this experience will be encoded into memory. Thus, the next time you need to make a lunch choice, this memory may be called upon to start the temporal process again.

Cognitive neuroscience findings in healthy people suggest that our ability to anticipate the future relies upon our ability to remember the past (e.g., Schacter et al. 2007), with a core network of brain regions, including areas of the medial prefrontal and medial temporal cortex, supporting both abilities. Thus, when we imagine what it will be like to see the latest Batman movie, we likely draw upon our past experiences with Batman or superhero movies to help imagine this future experience. Maintaining and savoring emotional experiences as they occur likely facilitates the development of memories for these experiences, and evidence from psychophysiological and fMRI studies find that people with schizophrenia appear to have difficulty holding on to these experiences. That is, while viewing emotionally evocative pictures, people with schizophrenia show brain activation (Ursu et al. 2011), startle response (Kring et al. 2011), and event-related potentials (Horan et al. 2010) comparable to people without schizophrenia; however, differences begin to emerge during the end of the picture presentation and after the picture is removed from view. In an fMRI study, people with and without schizophrenia exhibited comparable activation of visual, amygdala, and prefrontal cortical regions, consistent with intact in-the-moment emotional experience. However, during the 12.5-s delay, there were marked differences between groups across prefrontal and limbic regions, and reduced activity in the DLPFC was positively correlated with clinical ratings of anhedonia.

In a startle paradigm, people with and without schizophrenia showed comparable eyeblink responses during picture viewing, yet just 2.5 s after the picture was removed from view, the group

Anticipation of emotion:

refers to the prediction of how one might feel in a future situation as well as the feeling right now that comes with thinking about the future

without schizophrenia continued to show the same pattern of startle response as during picture viewing (i.e., larger blinks to negative pictures, smaller blinks to positive pictures), but the group with schizophrenia no longer showed this differentiated pattern of blink responses. A behavioral study also found that people with schizophrenia were not able to maintain their emotion experience across a 3-s delay compared to people without schizophrenia (Gard et al. 2011), and this deficit remained even after controlling for deficits in a control task (maintaining judgments of stimulus brightness). By narrowing the time window of maintenance even further using a measure that has exquisite temporal resolution on the order of milliseconds, it was found that people with and without schizophrenia did not differ in so-called early event-related potentials (i.e., potentials that occur within 200 ms of picture onset, including P1) and middle (i.e., potentials occurring with 200–300 ms of picture onset, including P2, P3) event-related potentials. However, people with schizophrenia did not exhibit late (i.e., greater than 300 ms post onset) event-related potential responses that were distinguishable by valence, whereas people without schizophrenia did. This study suggests a different way of thinking about what in-the-moment responding actually means and that the response may not last very long among people with schizophrenia.

Interestingly, in the three aforementioned studies that identified an emotion maintenance deficit, people with schizophrenia did not differ in their reports of positive emotion to pleasant pictures or their reports of negative emotion to unpleasant pictures, no matter how long the delay between picture viewing and reporting (ranging from milliseconds to 12.5 s). In other words, despite differences in brain activation and psychophysiology, people with schizophrenia are still providing reports of experience that are indistinguishable from people without schizophrenia. How are they able to do this? It may be the case that rating questions that are congruent with the valence of the picture (e.g., how pleasant do you feel after looking at a picture of puppies?) do not require as much cognitive support or control, and thus reporting on these feelings may not rely upon the same neural underpinnings as other types of ratings. However, rating questions that are incongruent with the valence of the picture (e.g., how unpleasant do you feel after looking at a picture of puppies?) requires additional cognitive control to both inhibit a prepotent response (puppies are positive!) and marshal the task-appropriate response (how unpleasant do you feel?).

Taken together, these results are consistent with the notion that in-the-moment emotional responding (behaviorally, in the brain, physiologically) is relatively intact whereas anticipation of emotion and active maintenance of emotional information (i.e., cognitive manipulation of emotional responses) are disrupted in schizophrenia.

HOW EARLY DO EMOTION DEFICITS EMERGE IN (PRE)SCHIZOPHRENIA?

Are the emotion deficits in schizophrenia a consequence of the illness or are they antecedent to it? This important question has been a difficult one to answer. The typical age of onset for schizophrenia is between 18 and 25, and studying antecedent deficits to schizophrenia requires studying young people prior to the onset of schizophrenia. This can be done retrospectively by beginning with adults who have already been diagnosed with schizophrenia and then looking back to identify possible emotion deficits. In one of the most creative retrospective investigations of emotion deficits in schizophrenia, Walker and colleagues identified a group of adults with schizophrenia who had home movies taken at birthdays, family parties, holiday gatherings, etc. as children (Walker et al. 1993). They coded the home movies, looking for positive and negative facial expressions at younger ages, prior to the diagnosis. Walker et al. found that girls who later developed schizophrenia displayed fewer expressions of joy from infancy to adolescence compared to their healthy siblings, but both boys and girls who later developed schizophrenia exhibited

more negative expressions in adolescence compared to their siblings. Studying childhood records of adults who developed schizophrenia, Knight & Roff (1983, 1985) found evidence that affective disturbances appeared in childhood and persisted into adulthood. Although the findings from these studies are suggestive, it remains unclear whether this pattern of emotional behavior was related to the onset of schizophrenia and if the pattern was specific to schizophrenia since these studies were retrospective and had fairly limited samples. That is, children who were later seen in clinics or who have home movies made are not necessarily representative of all people with schizophrenia.

A stronger design for identifying emotion deficit antecedents is a prospective design where children can be assessed multiple times prior to and following the diagnosis of schizophrenia. How can such high-risk youth be identified? Two broad strategies can be found in the literature. In the 1970s, familial high-risk studies were developed by initially identifying one or two biological parents with schizophrenia and then following their children longitudinally in order to identify how many of these children develop schizophrenia and what types of childhood emotional deficits precede or even predict the disorder's onset. These studies did not identify emotion deficits that preceded the disorder, but this is likely because they were not specifically looking for them. As discussed previously, it was not until the late 1980s that schizophrenia researchers embraced the study of emotion. In addition, one of the difficulties with the familial high-risk studies is that extremely large sample sizes are needed given that only 1 in 10 children who have a biological parent with schizophrenia go on to develop the disorder themselves (McGuffin et al. 2004). In addition, it is not particularly easy to locate a large sample of women or men with schizophrenia who have had their own children.

Because of these difficulties, the clinical high-risk study has been used in more recent investigations of possible emotion deficit precursors. A clinical high-risk study is a design that identifies people with early, attenuated signs of schizophrenia, most often milder forms of hallucinations, delusions, or disorganization that nonetheless cause impairment. One such study, the North American Prodrome Longitudinal Study (NAPLS), is being carried out at eight different centers in the United States and Canada (Addington et al. 2007). A great majority of the participants in this study were found to have negative symptoms, including symptoms involving motivation, pleasure, and emotion expression, at their baseline assessment, well before the development of schizophrenia or psychosis (Piskulic et al. 2012). In addition, the more severe the negative symptoms were at baseline, the greater the likelihood of developing full-blown psychosis at the one-year follow-up.

The evidence is mixed as to whether emotion perception deficits are observed prior to the onset of schizophrenia. Studies of emotion experience and expression have not yet been conducted with clinical high-risk participants. A few familial high-risk studies have found that people at familial risk for schizophrenia do not differ in reports of emotional experience in either laboratory tasks or experience sampling studies compared to people not at risk for schizophrenia (Phillips & Seidman 2008). Similarly, a couple of familial high-risk studies have found that relatives of people with schizophrenia do not have a deficit in facial emotion perception (Phillips & Seidman 2008). However, findings from the NAPLS indicated the clinical high-risk group exhibited a deficit in facial emotion identification and discrimination compared to healthy controls (Addington et al. 2008).

With respect to brain activation, an intriguing preliminary finding from the NAPLS has been reported (Gee et al. 2012). In this study, participants who were and were not at clinical high risk viewed emotional faces and made one of four judgments (in blocks), including an emotion label, a gender label, an emotion match, and a gender match. Although there were no behavioral differences between groups, the group at clinical high risk exhibited less functional connectivity between the amygdala and medial PFC than the control group for emotion labeling blocks relative to emotion matching blocks. This finding suggests that at the level of the brain, connectivity between regions that support emotion and cognition may be deficient prior to the onset of the illness.

Of course, it will be important to replicate this finding and follow-up with these participants as they do (or do not) convert to psychosis. Nevertheless, the idea that issues with the neural circuitry needed to support emotion and cognition may precede the onset of schizophrenia is intriguing.

SUMMARY AND CONCLUSIONS: WHERE DO WE GO FROM HERE?

Although early theorists such as Bleuler, Kraepelin, and Sullivan observed deficits in emotion among people with schizophrenia over a century ago, the introduction of translational research paradigms to the study of emotion deficits in schizophrenia in the past 40 years has propelled our understanding of the nature of these deficits by leaps and bounds. We have reviewed the evidence for deficits in emotion perception, expression, and experience, as well as evidence for deficits in psychophysiological and brain activation responses to emotionally evocative (and neutral) material. Overall, the evidence points to some areas of emotion that are deficient and other areas that are surprisingly intact.

With respect to emotion perception, people with schizophrenia have a deficit in the identification and discrimination of emotion depicted in the face as well as in facial perception more generally. Importantly, these findings have been well replicated across different groups of participants and tasks. Intriguingly, people with schizophrenia do not show a marked impairment on implicit emotion perception tasks or tasks that require a simple target, nontarget decision. Recent affective science research points to the importance of context in emotion perception, and context can include even the presence of emotion words that are used in identification tasks (e.g., choose the word that best describes the emotion depicted on the face). Future research in this area would benefit from systematically manipulating the contextual demands of emotion perception tasks to unpack the ways in which the integration of context (e.g., prior knowledge about emotion faces and labels) with the faces presented in the task combine to influence performance. We know next to nothing about when deficits in emotion and facial perception may begin, and thus studying emotion perception among people at clinical high risk for schizophrenia is another important future direction for research. Finally, a handful of studies have tested different types of psychosocial interventions that include modules designed to bolster emotion perception skills (e.g., Horan et al. 2009, Roberts & Penn 2009). These studies have promising initial results and thus could be more formally evaluated among people with schizophrenia as well as those at risk for schizophrenia.

Another well-replicated emotion deficit in schizophrenia is in the emotion expression domain. People with schizophrenia do not show very many outward expressions of emotion, yet they do contract the facial muscles associated with emotion expressions (e.g., cheek muscle with smiling) in response to evocative material. The reasons why these muscle contractions are not strong enough to move the skin and connective tissue on the face and thus become observable remain a bit of a puzzle. Although movement in fMRI studies is often considered a nuisance variable, it would be informative to design an imaging study to map out the motor connectivity of facial expression during emotion elicitation tasks among people with and without schizophrenia. Additional research among individuals at clinical high risk for schizophrenia is also needed to better ascertain the developmental course of diminished expression in schizophrenia.

One area of emotion response that is relatively intact is the domain of emotion experience. That is, people with schizophrenia report feeling as positive in the presence of pleasant stimuli, situations, events, and they report feeling as negative in the presence of unpleasant materials, as do people without schizophrenia. Of course, people may question whether self-reports of emotion can be “trusted” among people with schizophrenia, but more than two decades of research on emotion experience has not revealed any evidence to suggest that people with schizophrenia are less capable

of reporting on their feelings than people without schizophrenia. Furthermore, evidence from other emotion response components, including psychophysiology and brain activation, reinforces the emotion experience findings.

Despite converging evidence that in-the-moment emotional experience is intact in schizophrenia, there are other signs of problems in the emotion experience domain. People with schizophrenia often report experiencing more emotion than people without schizophrenia, particularly with regard to feelings that are incongruent with the stimulus material. This is especially true for neutral stimuli, where people with schizophrenia have been found to report experiencing more negative (and sometimes positive) emotion in response to putatively emotionless material than do people without schizophrenia. Additional evidence points to reports of greater negative emotion to pleasant stimuli among people with schizophrenia as well. There are at least four intriguing ideas about why people with schizophrenia may exhibit these somewhat odd reports of emotional experience, and they are excellent avenues for future research. First, these types of incongruent or mismatched reports of emotional experience may be observed only among a subset of people with schizophrenia, and it would be useful to know more about whether such a subgroup can be meaningfully identified. Second, these types of mismatched reports may not reflect a deficit in emotion per se, but rather a deficit in cognitive control in that reporting on incongruent feelings requires drawing upon representations of emotion that are less readily available in that moment. Third, ambivalence may account for these mismatched ratings, whether because of ever-present negative affect that cannot be dampened in the presence of neutral or pleasant stimuli or a more general lack of differentiation of feeling states. Finally, people with schizophrenia may have less accessibility to beliefs about emotion and the self, which may interfere with their ability to make these incongruent ratings. Fortunately, all of these ideas lend themselves to testing, and thus we can gain purchase on understanding this phenomenon in future research. It is highly likely that more than one of these ideas will be able to further our understanding of these types of incongruent experience reports in schizophrenia.

Findings from psychophysiology and brain imaging studies are, for the most part, consistent with the findings on emotion experience. That is, in the presence of emotionally evocative material, people with schizophrenia tend to exhibit comparable psychophysiological responses and brain activation, particularly in amygdala. Where differences in brain activation are observed, they tend to be in areas that are also tightly coupled with cognitive control, such as the DLPFC and anterior cingulate cortex. Similar to findings on emotional experience, brain imaging studies also suggest a different pattern of brain activation in response to neutral stimuli. Specifically, results from two recent meta-analyses indicated that people with schizophrenia showed less amygdala activation compared to people without schizophrenia, but only when the dependent variable was a contrast that subtracted activation during a neutral condition from activation during a negative condition (Anticevic et al. 2012, Taylor et al. 2012). It is impossible to discern whether these findings reflect underactivation of amygdala in response to negative stimuli or overactivation to the neutral stimuli from these studies, but this could be disentangled in future research.

Even though people with schizophrenia appear to be responding comparably to people without schizophrenia in the presence of emotional material, whether in terms of experience or brain activation, there is evidence to suggest that something is amiss both before (i.e., anticipation) and after (i.e., maintenance) emotional material is presented. Indeed, people with schizophrenia have difficulty in anticipating and predicting whether future events will be pleasurable, and they also report less pleasure in anticipation of things to come. What we do not know, however, is whether this anticipation deficit extends to negative and even neutral events. One study found that people with schizophrenia exhibited fewer anticipatory event-related potentials (contingent negative variation, stimulus preceding negativity) than people without schizophrenia across positive, negative, and

neutral stimuli, suggesting that the anticipatory deficit may not be specific to pleasant events or even emotional events. Indeed, the focus of much current research on emotion deficits in schizophrenia is to discern the linkages and overlap with emotion and cognition, both behaviorally and neutrally. Deficits in anticipatory pleasure likely interfere with goal-directed behavior (Gard et al. 2007), though this linkage needs to be more systematically tested in future research. Another area in need of additional study is emotion maintenance. Psychophysiological and fMRI studies indicate that people with schizophrenia do not savor or maintain an emotional experience for long and that this is likely linked to their ability to remember this experience (though this remains to be fully tested).

Taking the final step of translation to treatment development is an area of research that is desperately needed. The negative symptoms of schizophrenia are among the most difficult to treat, and these are the symptoms that involve the emotion deficits we have reviewed here. The difficult reality is that we do not have treatments that work very well. Indeed, the American Psychiatric Association's (2004) *Practice Guideline for the Treatment of Patients with Schizophrenia* notes, "There are no treatments with proven efficacy for primary negative symptoms" (p. 15). In our view, changes may be most likely when basic symptom and translational research processes are targeted for intervention. For example, a couple of uncontrolled studies and one randomized controlled intervention study suggest that emotion maintenance can be improved (e.g., Grant et al. 2012, Johnson et al. 2009, Meyer et al. 2012). Thus, perhaps the most important future direction that flows from this review is the additional and intensive focus on treatment development that uses the deficits identified here as the starting point for building a better intervention for people with schizophrenia.

SUMMARY POINTS

1. People with schizophrenia do not appear to have a specific deficit in identifying facial emotion, but they do appear to have general deficits in facial processing and in the integration of context with faces.
2. Although observable outward emotional expression is diminished in schizophrenia, studies indicate that people with schizophrenia do display similar—albeit very subtle—facial muscle movements that are congruent with the valence of what they are responding to.
3. People with schizophrenia reliably report on their own feelings both in the moment and in retrospect.
4. People with schizophrenia report experiencing the same types, intensities, and frequencies of emotion as do people without schizophrenia, and their reports are consistent with the valence of the stimuli or situation. Although people with schizophrenia report comparable amounts of positive and negative emotions in response to evocative stimuli as do people without schizophrenia, they also experience neutral and positive stimuli as being more negative.
5. PET and fMRI studies indicate that people with schizophrenia by and large have comparable brain activation in response to emotional stimuli compared to people without schizophrenia. However, differences in brain activation observed in regions associated with emotion and in regions that are also associated with cognitive control may reflect responses to neutral rather than emotional stimuli.
6. People with schizophrenia have difficulty in cognitive control associated with emotion, including anticipation and maintenance of feelings.

FUTURE ISSUES

1. We need to extend what we know about the temporal course of emotion in order to distinguish whether people with schizophrenia have a general deficit in anticipation or a more specific deficit in the anticipation of emotion.
2. We need to investigate when emotion deficits emerge in (pre)schizophrenia by studying emotion perception, emotion expression, and emotion experience in clinical high-risk samples.
3. We need to integrate and translate these research findings on emotion deficits in schizophrenia to more targeted and effective treatments, both pharmacological and psychosocial.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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