The state-trait disjunction of anhedonia in schizophrenia: Potential affective, cognitive and social-based mechanisms

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Abstract

Dysfunctions in affective experience reflect a pernicious feature of schizophrenia-spectrum pathology that are largely intractable under current treatment regimens. Of note, individuals with schizophrenia show robust and marked deficits in the experience of pleasure when assessed using "trait" measures of affect (i.e., trait questionnaires and clinical interview), but fail to demonstrate this anhedonia "in the moment" using controlled laboratory mood-induction procedures. The reasons for this disjunction are unclear, but the last decade has seen a number of recent studies tackling this issue. We conduct a comprehensive review of this literature here. Five different explanations for this state-trait disjunction are identified: 1) anticipatory hedonic experience deficit, 2) affective regulation deficit, 3) encoding-retrieval deficit, 4) representational deficit and 5) social-specific deficit theories. Our present article reviews each of these theories, placing them in context of the larger affective and cognitive neuroscience literatures when appropriate. Additionally, practical recommendations for future studies examining affective dysfunction within the schizophrenia-spectrum are discussed.

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1. Introduction

Dysfunctions in affective experience are a critical aspect of schizophrenia-spectrum pathology as they often precede illness onset (Kwapil, 1998; Walker & Lewine, 1990) and are associated with a range of social and occupational impairments (Blanchard, Mueser, & Bellack, 1998; Cohen et al., 2005). Of note, anhedonia, defined as an inability to experience pleasant emotions, is postulated to be a core symptom reflecting a latent vulnerability marker of the disorder (Docherty & Sponheim, 2008; Meehl, 1962). Nonetheless, empirical study of anhedonia in schizophrenia has provided inconsistent support for this notion. On the one hand, patients generally report attenuated experience of pleasant emotions on self-report trait-based instruments of hedonic experience (Berenbaum & Fujita, 1994; Horan, Blanchard, Clark, & Green, 2008). Moreover, trained experts rate individuals with schizophrenia as having abnormally high levels of anhedonia using interview-rated instruments, typically spanning week-long epochs (e.g., Blanchard & Cohen, 2006; Sayers, Curran, & Mueser, 1996). However, examinations of state emotions under controlled laboratory conditions have revealed that individuals with schizophrenia report experiencing normal levels of pleasant emotions while processing a broad range of stimuli (Cohen & Minor, 2010; Kring & Moran, 2008). Similarly, when individuals with schizophrenia are asked about their state emotions while navigating...
through their natural environments as part of Experience Sampling Methodologies, they report relatively normal pleasant emotional reactions (Myin-Germeys, Delespaul, & deVries, 2000; Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001). Thus, there appears to be a disjunction between trait and state report of emotional experience in schizophrenia. The mechanism underlying this disjunction is unknown. We believe that understanding this issue may offer important insights into the nature of schizophrenia pathology. The present paper examines five separate theories that could explain the state-trait disjunction. While these theories are not necessarily mutually exclusive, they do differ in terms of the larger affective, cognitive and social literatures that they draw from. These five theories are: 1) anticipatory hedonic experience theory, 2) affective regulation deficit theory, 3) encoding-retrieval deficit theory, 4) representational deficit theory, and 5) social-specific deficit theory. The literature from a broad range of schizophrenia-spectrum studies is examined here and grounded in the larger affective and cognitive neuroscience literatures when appropriate.

2. Specific affective deficit: Anticipatory hedonic experience

One theory offered to explain the discrepancy between state and trait emotion is that schizophrenic patients possess a relatively specific deficit in affective processes that compromises their ability to accurately forecast affective experiences. In contrast, patients’ ability to experience affect “in the moment” is intact. As discussed below, there is solid support for the notion that “anticipatory” hedonic systems, involving the ability to predict the future experience of pleasure, are distinct from “consummatory” hedonic systems, involving experience of stimuli “on-line” (Gard, Kring, Card, Horan, & Green, 2007; Horan et al., 2008). It follows that when individuals with schizophrenia are asked to evaluate their reactions to potential events as part of a questionnaire or a clinical interview – presumably activating “anticipatory” hedonic neurocircuitry, they consistently report experiencing diminished hedonic reactions. However, when consummatory systems are activated by “in the moment” experience of stimuli, hedonic experience is normal.

There is considerable support from neuroscience more generally that anticipatory and consummatory pleasure systems are neurobiologically distinct. Distinct neural pathways have been identified related to the experience of “wanting” (analogous to anticipatory pleasure) vs. “liking” (analogous to consummatory pleasure) (Berridge & Robinson, 1998, 2003; Gard et al., 2007; Knutson, Adams, Fong, & Hommer, 2001). For example, drug and alcohol dependence in humans has been shown to persist even after the separate experience of pleasure during use is diminished (Berridge, 2007; Berridge & Robinson, 1995; Hobbs, Remington, & Glaudier, 2005; Lambert, McLeod, & Schen, 2006). Further support is seen at a neurobiological level, as dopamine has shown to be strongly linked to anticipatory rather than consummatory pleasure (Berridge, 2007; Berridge & Robinson, 1998). In contrast, the experience of consummatory pleasure has been more strongly linked to the serotonin and opioid systems (Berridge & Robinson, 1998;2003; Schultz, 2002; Wise, 2002). Given that dopaminergic abnormalities are considered central to core schizophrenia pathology (Kapur & Mamo, 2003; Stanislaw & Carson, 2004), arguably more so than either opioid or serotonin abnormalities (Abi-Dargham, 2007; Laurelle et al., 2000; Peckys & Hurd, 2001), it stands to reason that anticipatory pleasure would be affected in patients.

A challenge for this line of research concerns the lack of validated assessments for distinguishing between anticipatory and consummatory pleasure that can readily be applied to schizophrenia populations. As noted above, anticipatory pleasure abilities have been assessed from responses to self-report questionnaire and interview items assessing a broad range of potential experiences (e.g., “How do you generally feel?”) whereas consummatory pleasure abilities have been assessed using very specific affect probes (e.g., “How do you feel at this exact moment?”) following laboratory mood-induction procedures. In order to directly compare anticipatory and consummatory pleasure, similar assessment domains and methods should be employed. Two recent lines of research have addressed this issue and offer preliminary support for the notion that anticipatory but not consummatory systems are affected in schizophrenia. Gard, Gard, Kring, and John (2006) developed the Temporal Experience of Pleasure Scale (TEPS), a brief self-report questionnaire designed to separately assess anticipatory and consummatory pleasure experience. The TEPS consists of ten anticipatory (“I get so excited the night before a major holiday I can hardly sleep.”) and eight consummatory (“I enjoy taking a deep breath of fresh air when I walk outside.”) items, with good internal reliability for the total scale, acceptable reliability for the subscales, good test–retest reliability and both convergent and divergent validity. Gard et al. (2007) reported that patients with schizophrenia versus non-psychiatric controls reported abnormally low anticipatory, but not consummatory, pleasure. In terms of divergent validity, the anticipatory, but not the consummatory, subscale scores were significantly correlated with scores from another widely used and validated self report measure of social anhedonia (i.e., the Revised Social Anhedonia Scale; Eckblad et al., 1982), and scores from an interviewer-rated instrument (i.e., the Schedule for the Assessment of Negative Symptoms; Andreasen, 1983).

Perhaps the most compelling evidence supporting specific anticipatory affective deficit hypothesis comes from an Experience Sampling Methodology study examining differences in self-reported anticipatory and consummatory pleasure in individuals with schizophrenia (Gard et al., 2007). During the study, participants were paged seven times throughout the day for one week and asked to record what they were doing and how much they were enjoying it (i.e., consummatory pleasure). They were also asked to record what they were looking forward to doing and how much pleasure they anticipated experiencing during the activity. Overall, patients reported anticipating less pleasure from future activities than controls at a trend level (p = 0.06), but there was no difference in consummatory ratings. This finding is impressive in light of the small sample size employed as there were only fifteen patients and twelve controls. An interesting pattern emerged when future activities were categorized as “goal-directed” (e.g., making dinner and doing an errand) or “non-goal directed” (e.g., eating dinner and watching TV) activities. Consummatory pleasure was not significantly different for either category. Individuals with schizophrenia, however, looked forward to and engaged in goal-directed activities significantly less often and anticipated receiving significantly less pleasure from goal-directed activities compared to controls; group differences in anticipatory pleasure for non-goal directed activities were nonsignificant. In summary, individuals with schizophrenia consistently predicted experiencing abnormally low levels of pleasant emotions for goal-directed activities, but reported normal experiences on “in the moment” assessments.

While the specific anticipatory affective deficit hypothesis is particularly promising, there are several issues to contend with. First, some have raised suspicions that deficits in anticipatory pleasure are iatrogenic consequences of antipsychotic medication rather than idiopathic features of the disorder. Gard et al. (2007) argue that this is not the case, as the decreased activity in brain regions associated with reward anticipation (e.g., ventral striatum) are also evident in unmedicated patients presented with reward-indicating cues (Juckel et al., 2006). Second, anticipatory and consummatory pleasure assessments are particularly difficult to match, especially for a schizophrenia population who may suffer from a host of cognitive difficulties. This is a particular concern for questionnaire-based assessments which may employ complicated phrasings or may require high levels of abstraction to “affectively forecast” pleasure states in the future. All items on the TEPS (anticipatory and...
consummatory) require some level of abstraction as participants must consider their hedonic experiences in the absence of immediately affectively-relevant stimuli, and both subscales may tap into similar processes that may reflective at some level of anticipatory mechanisms. Third, it is important to note that hedonic experience is a broad construct encompassing a wide range of social, physical and intellectual domains. The TEPS items contain exclusively physical stimuli and there is research to suggest that hedonic dysfunction in the social domain is particularly relevant to schizophrenia spectrum disorders (see section six).

3. Affective regulation deficit

Another possibility is that the state–trait disjunction reflects a global affective regulation deficit. Affective regulation deficits reflect a variety of deliberate and automatic strategies for downregulating unpleasant emotional states and enhancing pleasant ones (Gross, 1998; Westen, Muderrisoglu, Fowler, Shelder, & Koren, 1997). Affective regulation is vital to psychological well-being. For example, difficulty in affect regulation is a core feature of various disorders and harmful behavior, such as Borderline Personality Disorder (Conklin, Bradley, & Westen, 2006), suicidal behavior (Yen, Zlotnick, & Pearlstein, 1997), posttraumatic stress (Van der Kolk et al., 1996), and vulnerability to marijuana use problems (Simons & Carey, 2002). We posit here that individuals with schizophrenia may show a specific deficit in the ability to attenuate unpleasant emotional states, and this, in turn, contaminates the experience of pleasant states.

There is good reason to think that an affective regulation deficit explains the state–trait disjunction in schizophrenia. The neural structures thought to be involved in affective regulation processes include the prefrontal and anterior cingulate cortex (ACC) regions (Kross, Davidson, Weber, & Ochsner, 2009; Ochsner, Bunge, Gross, & Gabrieli, 2002). One function these structures appear to serve involves down-regulating negative affectivity arising from limbic region structures, notably the amygdala (Davidson, 2003; Ochsner et al., 2002). Direct connections are well documented between these three structures, and a number of imaging studies have also demonstrated that neural activity in prefrontal and ACC regions mediate amygdala activity (Kross et al., 2009). Dysfunction in each of these brain regions is prominent in individuals with schizophrenia, notably aberrant ACC (Heckers et al., 2004), prefrontal (Barch, Sheline, Csernansky, & Snyder, 2003) and increased “resting” amygdala (Taylor, Phan, Britton, & Liberzon, 2005) activity relative to non-psychiatric controls. Further, conflict monitoring and working memory, abilities dependent on intact ACC and prefrontal cortical functions (Ko et al., 2009), are impaired in schizophrenia patients compared to controls (Kim et al., 2009). Affective regulation deficit theory posits that decreased frontal resources contributes to difficulty regulating limbic over-activity in schizophrenia (Cohen & Minor, 2010; Kerns & Becker, 2008).

If an affective regulation theory were a valid explanation for the “state–trait” disjunction in schizophrenia, we would expect that individuals with schizophrenia would not only report abnormally high levels of negative emotion on trait questionnaires, but also on assessments of state emotion during laboratory mood induction. Negative emotion, unchecked by ACC and prefrontal regulatory mechanisms, would be prominent in reports of trait affectivity and when processing stimuli “in the moment.” This is exactly what is seen. A recent meta-analysis of studies examining emotional reactivity of schizophrenia patients during laboratory assessments (Cohen & Minor, 2010) found that although patients did not differ from control subjects in subjective pleasant affective reactions to evocative stimuli (as discussed above), they reported abnormally strong co-occurring unpleasant affective reactions. This effect was seen in response to a wide range of positive and neutral stimuli and was striking in magnitude (mean weighted Cohen’s d = 0.72 and 0.64 for pleasant and neutral stimuli respectively) and in consistency across studies – each of the eleven studies included in this analysis reported differences between controls and patients at the medium effect size level or greater (range of Cohen’s d values = 0.51–1.31). These results support the notion that increased negative affectivity, as opposed to decreased positive affectivity (i.e., anhedonia) is the chief domain in which affective experience is disrupted in schizophrenia.

Another study of state emotion warrants mention here, as it employed a different approach to understanding emotional reactions than explicit self-report. Cohen, St-Hilaire, Aakre, and Docherty (2009b) examined lexical expression of individuals during a laboratory narrative exercise. Specifically, participants were asked to discuss positive, negative, and neutral memories from their lives. The speech was transcribed and analyzed using Pennebaker’s Lexical and Inquiry Word Count system, which has been used to understand emotion across a range of psychological issues (e.g., deceptive language, Bond & Lee, 2005; suicidality, Handelman & Lester, 2007; depression, Vanheule, Desmet, & Meganck, 2000; trauma adjustment, Beaudreau, 2007), and has been validated as a method for measuring verbal expression of emotion (Bantam & Owen, 2009; Kahn, Tobin, Massey, & Anderson, 2007). Patients with schizophrenia who were rated as being clinically anhedonic based on available information (self-report and medical records), patients with schizophrenia but without anhedonia, and controls were compared on the percentage of total words that had a pleasant (e.g., love and cheer) and unpleasant (e.g., kill and bad) valence. Patients with anhedonia showed a dramatic increase in words expressing unpleasant emotion when instructed to discuss pleasurable memories from their lives compared to the other groups, but no corresponding decrease in positive emotions. These results further the notion that anhedonia, at least as it is measured using interview-rated instruments, reflects poor regulation of negative affect rather than a deficit in positive affect.

More support for the affective regulation deficit theory comes from a study by Trémeau et al. (2009) examining subjective ratings to a variety of positive and negative stimuli presented in the laboratory. Individuals with schizophrenia reported experiencing abnormally high levels of conflicting emotions – more negative emotions to positive stimuli and more positive emotions to negative stimuli. Moreover, self-reported trait anhedonia was significantly associated with state negative ratings in the presence of pleasant stimuli, and this relationship was more robust than the relationship between self-reported trait anhedonia and state pleasant ratings. It is also noteworthy that abnormally high negative emotion evoked from positive stimuli was associated with a range of clinical variables, such as increased global illness severity and poorer overall functional life skills.

4. Encoding-retrieval deficit

The encoding-retrieval deficit is the third possible explanation for state–trait disjunction in schizophrenia. It may be the case that affective experience is not directly affected in individuals with schizophrenia, but that encoding or recall dysfunction leads to inaccurate representations of these experiences when questionnaires or clinical interviews are administered. For example, an individual confronted with the statement “I like playing with and petting soft little kittens or puppies” (from the Revised Physical Anhedonia Scale: Chapman & Chapman, 1978), may have difficulty recalling petting a kitten or puppy before, how often they have played with a kitten or puppy, and what the context surrounding these events was. This hypothesis is attractive in light of findings that memory deficits are pronounced in individuals with schizophrenia (Bilder et al., 2000; Danion, Rizzo, & Brunet, 1999; Gold, Randolph, Carpenter, Goldberg, & Weinberger, 1992; Huron et al., 1995), particularly for memories pertaining to autobiographical events (Neumann, Blairy, Leompte, &...
Philippot, 2007). Furthermore, two comprehensive reviews of neurocognitive deficits in schizophrenia have concluded that verbal memory deficits reflect the most disrupted of neurocognitive functions in schizophrenia (Green, 1996; Heinrichs & Zakzanis, 1998).

In considering this theory, one may ask why encoding or recall deficits would result in patients with schizophrenia consistently showing, in the absence of a stable memory representation, a “negativistic bias.” If they truly did not remember past experiences, one might conclude that their responses should reflect a more random response style. There are two potential answers to this concern. First, it could be the case that patients’ responses are more random than controls but their summary scores on tests of hedonic experience are significantly lower than controls who are more accurate when predicting their hedonic experiences. For example, on a “true/false” format questionnaire assessing hedonic experience (e.g., Revised Social Anhedonia Scale), individuals with random response style would presumably score close to 50%, which is much higher than control participants, who endorse approximately 15–20% of items assessing schizotypal traits as true (Chapman, Chapman, & Raulin, 1976). However, evidence against a random response style in patients concerns high test-retest reliability and internal consistency on self-report trait questionnaires in patients with schizophrenia (e.g., Blanchard et al., 1998; Herbener & Harrow, 2002). The second possibility is that patients show a more global negative bias that colors their interpretation of item content. In the absence of compelling memories to guide one’s response to a question assessing hedonic experience, patients’ natural tendency is to respond with a negativistic bias. Although to our knowledge this hypothesis remains untested, individuals with schizophrenia have shown a host of cognitive biases when interpreting stimuli. In addition to the negative bias reviewed in Section 2 of this paper, there is evidence that some individuals with schizophrenia (Cohen, Nienow, Dinzoe, & Docherty, 2009; Kohler et al., 2003) or those at high risk for schizophrenia (Brown & Cohen, 2010) show a negative bias when interpreting facial expressions or an attentional bias toward threatening facial expressions (Green, Williams, & Davidson, 2001). There is also evidence for other cognitive biases in individuals with schizophrenia such as attribution of negative events to external causes (Aakre, Seghers, St-Hilaire, & Docherty, 2009) or jumping to conclusions (Moritz & Woodward, 2005).

When discussing memory for affective stimuli, it is important to note that healthy adults show an enhanced ability to recall emotionally valenced versus neutral/non-valenced stimuli (Bradley & Mathews, 1983, 1988; Canli, Zhao, Desmond, Glover, & Gabrieli, 1999; Dolcos, LaBar, & Cabeza, 2004; Fiedler, Pampe, & Scherf, 1986; Hamann, 2001; Rusting, 1998). This bias for emotionally valenced information may be partially explained by the memory modulation hypothesis (McGaugh, 2004), which posits that the amygdala plays an important role in the encoding and recall of emotional experiences. Supporting this hypothesis, healthy adults who exhibit increased activation of the amygdala when encoding emotionally valenced stimuli have demonstrated enhanced recall of this information later (Canli, Zhao, Breuer, Gabrieli, & Cahill, 2000; Kensinger & Corkin, 2004). In contrast, evidence suggests that patients with schizophrenia have decreased amygdala volume (Lawrie & Abukmeil, 1998; Wright et al., 2000) and exhibit less amygdala activation when encoding emotionally valenced stimuli (Takahashi et al., 2004; Taylor, Liberson, Decker, & Koepp, 2002), although delayed memory for these stimuli has not been examined.

In considering the encoding-retrieval hypothesis, it seems a tenable hypothesis that anhedonia is associated with a lack of improved recall ability for pleasant stimuli compared to neutral stimuli. Such a deficit could explain the state-trait disjunction from an information processing standpoint, as individuals who lack an encoding/recall bias for positive stimuli might be more apt to offer responses downplaying their hedonic experiences when queried. As yet, data supporting abnormal memory performance for affective stimuli in schizophrenia are mixed. To our knowledge, nine studies have examined emotional memory performance in patients with schizophrenia, with five of the nine reporting some degree of emotion-specific abnormality (Calev & Edelstien, 1993; Hall, Harris, McKirdy, Johnstone, & Lawrie, 2007; Herbener, Rosen, Rhine, & Sweeney, 2007; Koh, Grinker, Marusarz, & Forman, 1981; Neumann et al., 2007) and four reporting no abnormalities at all (Danion, Kazes, Huron, & Karchouni, 2003; Horan, Green, Kring, & Nuechterlein, 2006; Koh, Kayton, & Peterson, 1976; Mathews & Barch, 2004).

When evaluating this literature, there are two important issues. The first is whether memory dysfunctions occur as a function of pleasant stimuli, unpleasant stimuli, or both. This issue is unclear because of inconsistency across studies. Three studies report that, compared to controls, individuals with schizophrenia have significantly poorer memory for pleasant stimuli (Calev & Edelstien, 1993; Herbener et al., 2007; Koh et al., 1981), one study reports poorer memory for unpleasant stimuli (Neumann et al., 2007) and one study reports poor memory for both (Hall et al., 2007). Thus, memory dysfunctions for pleasant stimuli have only been found in four of the nine studies to date. The second issue concerns the amount of time between encoding of stimuli and recall (Herbener, 2008), as it may be the case that the delay between presentation leads to abnormal decaying of affective memory traces. All of the null-finding studies listed above employed short encoding/recall delays – two studies had no delay (Koh et al., 1976; Mathews & Barch, 2004), one had a fifteen minute delay (Danion et al., 2003), and one had a four hour delay (Horan et al., 2006). In contrast, four of the five studies finding affective memory deficits had recall delays of at least one day, ranging from 24 h (Herbener et al., 2007; Neumann et al., 2007) to 48 h (Calev & Edelstien, 1993) to three weeks (Hall et al., 2007). In fact, in the two studies where both immediate and delayed recall were examined (Calev & Edelstien, 1993; Hall et al., 2007), individuals with schizophrenia did not differ in immediate recognition of emotional stimuli, but were significantly worse at recalling emotional stimuli after delay. From these studies, it appears that individuals with schizophrenia might not show abnormal memory for valenced stimuli after a brief delay period. With more extended delay, they appear to show difficulties with integrating or retrieving affective memories. Herbener et al. (2007) have proposed that anhedonia may result from an inability to consolidate experiences into long-term memory—a notion that nicely fits the aforementioned data.

5. Representational deficit

The fourth hypothesis explaining the state-trait disjunction in schizophrenia involves a specific deficit in the ability to mentally represent the values of stimuli. The representational deficit theory, advanced by Gold and colleagues (Gold, Waltz, Prentice, Morris, & Heerey, 2008), purports that patients’ ability to experience in-the-moment pleasure is not directly compromised, but their ability to develop a stable mental representation pairing valence and a specific stimulus is impaired. This in turn attenuates motivation to seek out the activity or stimulus in the future. Therefore, state experience is preserved, but in the absence of the stimulus, the hedonic properties of the stimuli are lost. While there is potential overlap between this theory and the encoding-retrieval deficit discussed above, the representational deficit theory is more focused on stimulus-valence representations than it is on effective encoding or retrieval of the stimulus itself. There is also potential overlap between this theory and the specific anticipatory pleasure deficit (outlined in section one), though the representational deficit theory examines the state-trait disjunction within a learning theory framework.

The representational deficit theory is supported by two bodies of findings. First, evidence suggests that the link between the experience of stimuli and encoded representations of these experiences is
abnormally disparate in patients with schizophrenia. Heerey and Gold (2007) asked stable outpatients with schizophrenia to rate their emotional experiences to standardized pleasant, unpleasant and neutral picture stimuli. They were also asked to make speeded button presses in the absence of each stimulus to indicate whether they wished to see it again. Consistent with the larger literature on state hedonic experience in schizophrenia, these patients reported similar subjective hedonic ratings to the stimuli as controls. In contrast to controls, who showed significant changes in button pressing behaviors across the pleasant, unpleasant and neutral stimuli, patients showed little behavioral change across these conditions. That is, patients showed a relatively consistent pattern of button pressing regardless of the valence of stimuli. Moreover, correlations between subjective ratings and button pressing behavior were much higher in the control than the patient group, suggesting that state experience in the presence of the stimuli has a closer tie to that experience in the absence of the stimuli in controls as compared to patients. Studies employing delayed discounting and probabilistic “gambling” decision making tasks have also supported this notion (e.g., Heerey, Robinson, McMahon, & Gold, 2007). Individuals with schizophrenia are significantly more likely than controls to select smaller immediate rewards over larger payoffs paid at a later date and are more likely to make much less optimal choices to maximize payoff. Taken together, these results suggest that individuals with schizophrenia show a deficit in the ability to mentally represent the value of a stimulus, whether it is a picture or more tangible reward. Thus, individuals with schizophrenia might be expected to report lower trait pleasurable experience in the absence of affective stimuli, despite intact state hedonic experience, because they have difficulty utilizing learning mechanisms to pair the hedonic reward of past experience with abstract representations of those experiences.

The second line of research used to support the representational deficit theory involves investigating reinforcement learning in schizophrenia. Gold et al. (2008) have proposed that individuals with schizophrenia show a differential deficit in “rapid learning systems,” which involve unexpected changes in reward contingencies, as compared to relatively intact “long-term knowledge” structures, which involve detection of irregularities over long-term stimuli presentations. Data from studies employing measures such as the Wisconsin Card Sorting Test (Prentice, Gold, & Buchanan, 2008), the probabilistic reversal learning paradigm (Waltz & Gold, 2007) and the reward sensitivity paradigm (Heerey, Bell-Warren, & Gold, 2008) have demonstrated that patients consistently show abnormally poor performance under conditions of changing reinforcement outcomes. These changing contingencies tap rapid learning processes and require the ability to develop an abstract, but implicit, representation of the stimulus valence. In contrast, when reinforcement contingencies are relatively stable over time, promoting the use of long-term knowledge systems, patients with schizophrenia show response accuracies similar to controls (Morris, Heerey, Gold, & Holroyd, 2008). That is, individuals with schizophrenia can employ feedback to effectively learn implicit stimulus–response pairings, but have difficulty updating values attached to these stimuli. Thus, as it is argued by Gold et al. (2008), dysfunctions in the ability to fully represent the affective value of stimuli, whether it be reinforcing or punishing, leads to a differential deficit in decision making and rapid learning. This deficit in mentally representing value in the absence of the stimulus leads patients to underestimate their predicted experience.

The representational deficit theory is consistent with conceptualizations of the neurobiology of anhedonia and negative symptoms more generally. For decades, models of schizophrenia pathology have drawn a link between the prefrontal cortex and negative symptoms, including Andreason’s negative syndrome (Andreasen & Olsen, 1982), Crow’s type II schizophrenia (Crow, 1985) and the deficit syndrome (Kirkpatrick, Buchanan, Ross, & Carpenter, 2001). The differential deficits in rapid, but not long-term learning implicated in the aforementioned studies, suggest dysfunctions in prefrontal structures. Of note, dorsolateral pre-frontal cortical structures are thought to play a critical role in delay discounting abilities (Bjork, Momanen, & Hommer, 2009) and orbitofrontal structures are implicated in updating and “on-line” adaptation to changing contingencies as used by “rapid” learning systems (Schoenbaum, Roesch, & Stalnaker, 2006). That these systems are largely dopaminergic in nature—a neurotransmitter central to schizophrenia pathology—and that other neuropsychological functions involving these regions are impaired, such as working memory and context processing (Barch, Carter, MacDonald, Braver, & Cohen, 2003; Park & Holzman, 1992) serves to further bolster this theory.

6. Social-specific hedonic deficit

A final explanation for the state-trait disjunction is that state emotion deficits are largely constrained to specific types of stimuli or specific domains. Affective experience can vary across a wide range of stimuli, and there is reason to believe that anhedonia in schizophrenia is restricted to social domains. In other words, experience of both state and trait pleasant emotion is disordered in individuals with schizophrenia in response to social stimuli only. Pleasurable experience of non-social stimuli, such as food, movies and cigarettes may largely be preserved. The social-specific hedonic deficit theory posits that the apparent “state-trait” disjunction is due to the fact that prior studies of state pleasant experience have largely investigated emotional experience in non-social domains.

Social anhedonia is an important feature of schizophrenia. Compared to healthy controls, individuals with schizophrenia consistently show elevated levels of social anhedonia using questionnaire-based assessments (Berenbaum & Oltmanns, 1992; Chapman et al., 1976) and in patients social anhedonia is associated with poorer social functioning (Blanchard et al., 1998). Research suggests that social anhedonia reflects a trait-like characteristic of the disorder. For example, when assessed over a period of one year, Blanchard, Horan, and Brown (2001) found that in patients social anhedonia remained stable even when symptoms remitted. This was not true of individuals with depression in which anhedonia tended to occur during mood episodes (Blanchard et al., 2001). Furthermore, in patients with schizophrenia, social anhedonia has been positively correlated with premorbid functioning rather than clinical status at the time of testing, while in patients with bipolar disorder and depression the opposite is true (Katsanis, Iacono, Beiser, & Lacey, 1992). There is also reason to believe that social anhedonia should be conceptualized and measured apart from non-social or physical anhedonia. In patients with schizophrenia, social anhedonia appears to be distinct from physical anhedonia in that social anhedonia is more strongly related to trait negative affect (Blanchard et al., 1998). Blanchard, Bellack, and Mueser (1994) also found that while questionnaire-measured physical anhedonia was related to attenuated pleasure experience with non-social stimuli (response to mood-inducing videos), social anhedonia was not. Therefore, the abilities to experience social and physical pleasure are not redundant with each other.

Social anhedonia appears to also be an important construct for understanding schizophrenia-spectrum liability more generally. From a theoretical perspective, social anhedonia has been postulated to be an important (but not sufficient) condition for schizophrenia pathology reflecting the product of a cascade of genetic and social learning influences (Meehl, 1990). This theory has contributed to the notion that social anhedonia is a marker of illness vulnerability, and can help identify “schizotypy” – a condition present in a putative categorically-distinct subgroup of the population with an estimated 10 percent prevalence rate. The specificity of schizophrenia-spectrum anhedonia to social domains as opposed to physical domains was theorized by Paul Meehl (1990), who noted that “schizoid anhedonia
is mainly interpersonal” (p. 833) and that even individuals with schizophrenia experience physical pleasure from sources such as watching television and smoking cigarettes (Meehl, 1990). In support of this conjecture, results from a ten year longitudinal study demonstrate the superiority of a scale assessing social anhedonia over one assessing physical anhedonia for predicting of schizophrenia-spectrum disorders (Kwapil, 1998). Social anhedonia is also associated with a number of schizophrenia-like maladies including impaired neurocognition (Cohen, Leung, Saperstein, & Blanchard, 2006; Gooding & Braun, 2004; Gooding & Tallent, 2003), physiological (Gooding, Shea, & Matts, 2005; Simons & Katkin, 1985) and genetic (Docherty & Spohnheim, 2008) anomalies. In this regard, social anhedonia appears to reflect a trait-like vulnerability to schizophrenia spectrum disorders.

The social cognition literature is also rapidly growing and offering insight into potential structures and pathways involved in processing social information. In the scientific literature concerning autism, a disorder that behaviorally resembles the negative/deficit subtype of schizophrenia, some have made the case that limbic structures, notably the amygdala are important in understanding social deficits. This is relevant in that both patients with schizophrenia and at-risk individuals show decreased amygdala volume compared to controls (Bogerts, Lieberman, Ashtari, & Bilder, 1993; Keshavan et al., 2002; Velakoulis et al., 2006; Wright et al., 2000). Also, a few studies of patients with schizophrenia have shown decreased activation of the amygdala in response to social emotional stimuli in the form of facial expressions (Kosaka et al., 2002) with corresponding overactivation of prefrontal regions (Fakra, Salgado-Pineda, Delaveau, Hariri, & Blin, 2008). This is consistent with a more effortful approach to interpretation of social stimuli in comparison with a more intuitive processing demonstrated by control participants, suggesting that these neurobiological abnormalities maybe especially manifest when patients have to interpret social stimuli. To our knowledge, only a handful of studies has directly examined whether social hedonic state experience is compromised in individuals with schizophrenia-spectrum pathology. The only study we are aware of to examine social hedonic experience in schizophrenia patients using “in the moment” assessment under controlled laboratory experiences (Aghevli, Blanchard, & Horan, 2003) did not find evidence for decreased experience of emotion in social situations in individuals with schizophrenia, only less emotional expressivity (i.e., facial expressions, hand gestures). Limitations of this study include a rather modest sample (n = 33) composed exclusively of male patients with schizophrenia. Moreover, the study used a social role play task to elicit emotions. Given potential cognitive limitations of patients with schizophrenia, including abstract representation, it is possible the role play was processed differently than in a genuine social interaction. For example, patients may have engaged in more effortful cognitive processing of the task (i.e., interpreting and remembering instructions, recalling past instances of similar social situations in real life). Furthermore, there is evidence that some individuals with schizophrenia (specifically paranoid) interpret non-genuine social stimuli differently than genuine stimuli (Davis & Gibson, 2000). It is possible that some patients had more difficulty than controls interpreting the role play as they would a real social situation. Additional studies are needed to examine whether these findings replicate in larger samples including female patients using other social stimuli. Further research should also examine emotional experience in social situations across different subtypes of schizophrenia, especially those with negative symptoms.

A handful of other “laboratory”-based studies has been conducted on individuals with schizophrenia. Quirk, Subramanian, and Hoerger (2007) found that individuals at risk for schizophrenia report less enjoyment from ambiguous social situations than healthy controls, perhaps suggesting that individuals lacking the resources to navigate ambiguous situations enjoy them less. These authors examined self-and other-report of enjoyment in novel/ambiguous (i.e., going to a party alone) and highly role-defined (i.e., visiting relatives) activities in both high and low schizotypy individuals. On both measures, the high schizotypy individuals enjoyed the more ambiguous situations, but not the highly role-defined activities, significantly less than the low schizotypy individuals. This is consistent with the argument that that schizophrenia spectrum anhedonia may be specifically social due to the inherent ambiguity of social situations, especially in light of the above-cited research suggesting that for individuals on the schizophrenia spectrum interpretation of social stimuli may be much more effortful and less intuitive. This may be the factor that leads to less pleasure in social situations (i.e. aversive drift; Meehl, 1990). Support for this idea is further garnered by research suggesting that individuals with schizotypy have difficulty identifying emotions in neutral and low intensity facial expression (the more ambiguous stimuli) rather than more obvious, higher intensity emotions (Brown & Cohen, 2010).

Another study (Kwapil et al., 2009) examined social experience using an Experience Sampling Methodology. College students who scored higher on the Revised Social Anhedonia Scale were more likely to be alone, to prefer being alone, and to report more positive emotions and less negative emotions when alone than with other people. Finally, in a study of explicit and implicit measures of social affectivity in schizotypy, individuals with schizotypy reported decreased experience of pleasure and increased experience of negative emotions compared to controls when asked explicitly and during a laboratory-based mood-induction procedure. Interestingly, a different pattern of results was observed with implicit measures of emotion (Cohen et al., Under Review), using lexical analysis of natural speech as subjects discussed their social relationships and an Implicit Association Test specially designed to examine implicit associations between valence categories (i.e., “good,” “neutral”) and social categories (i.e., “brother,” “friend”). Individuals with schizotypy and controls did not differ on any of these measures. These data provide further evidence that “state” social anhedonia is present in individuals with schizotypy, but that it occurs at a higher-order, more evaluative level rather than at a basic, automatic level.

In summary, it seems possible that particular aspects of social stimuli make them difficult to process for individuals with schizophrenia spectrum pathology. This may reflect a core deficit in the ability to enjoy social situations in the moment. Alternatively, these social-specific affective dysfunctions may reflect the inherent ambiguity and unpredictability of social situations, and also social cognitive deficits and/or biases. In this regard, it is not clear whether it is “social” stimuli per se that are difficult to process, or whether it is the inherent complexity or ambiguity of social stimuli. Though our knowledge of these processes remains incomplete, especially concerning etiological factors and neural mechanisms involved in social anhedonia, advances in the affective experience and social cognitive literatures lend support to the idea that state social anhedonia is an important aspect of schizophrenia spectrum pathology. Also unclear is whether individuals with schizophrenia show the same pattern of social affective experience as those with schizotypy. The single study of schizophrenia patients discussed above (Aghevli et al., 2003), although limited in some respects, suggests not. Thus, there is evidence of state social anhedonia in individuals with schizotypy but not for patients with schizophrenia. It is possible that individuals with schizotypy may have better insight into their social oddities or impaired social cognitive abilities which may limit these individuals’ ability to experience pleasure due to interfering negative emotions. More research is needed specifically on the “in the moment” affective experience of social stimuli in individuals with schizotypy and controls. Measures of self-confidence or perceived social skill may help clarify whether it is those situations in which schizophrenia spectrum individuals feel less competent or at ease that elicit less pleasure. It may also be the case that a lack of or deficient affiliative
tendency in combination with limited neurocognitive resources leads to a decreased social responsiveness and “aversive drift” in schizophrenia spectrum individuals (see Meehl, 1962 for elaboration). More research in all domains concerning the social life of schizophrenia spectrum individuals (social cognition, emotional processing, social behavior, underlying neural pathways) with corresponding integration of these findings is needed.

7. Summary and integration

In this article, we discussed five theories to explain the seemingly paradoxical “state-trait” disjunction in schizophrenia. Each theory has been previously suggested in the literature and the theories have varying degrees of empirical support. It is hopefully clear to the reader that these theories are not mutually exclusive. In fact, there is much overlap between these theories. For example, the “encoding-retrieval deficit” theory, based on the idea that patients have a dysfunction in remembering affective experiences, may fundamentally reflect a “representational” deficit in that stimuli and affective valence are not effectively coded together. Similarly, the “anticipatory” pleasure deficit, related to difficulties in affective forecasting, may be particularly pronounced due to co-activated negative emotions, as explained by the affective dysregulation theory. That these five theories are not distinct does not detract from their potential importance. Rather, it highlights the expanse of biological and neurocognitive systems that are associated with these affective deficits. That is, “anhedonia” appears to be associated with dysfunctions in anticipatory pleasure, affective regulation, memory, motivated behavior, reinforcement learning and social functioning systems. Carefully considering these systems together will be important for efforts toward developing a “unifying theory” of affective dysfunction in schizophrenia.

In developing an agenda for future research, some practical recommendations for future studies are indicated. First, pleasant and unpleasant emotion should be considered separately. There is ample evidence from the neuroscience literature more generally that the systems underlying pleasant and unpleasant emotions are independent and not necessarily antagonistic (Cacioppo, Gardner, & Berntson, 1997; Petty, Briñol, Petty, Fazio, & Briā±o et al., 2008; Priester & Petty, 1996), and we believe it is best to isolate these experiential systems when possible. Second, it is important to include measures of state and trait emotions that are closely matched in methods and psychometric properties. A notable limitation of the extant literature is that trait affect is measured using trait questionnaires while state affect is experimentally manipulated using laboratory procedures. It is difficult to directly compare findings from these disparate methods. Third, employing measures from cognitive psychology (e.g., delay discounting, affective memory recall paradigms) potentially improves the sensitivity for investigating affective processing—although it is important to consider affect across a range of cognitive functions. Considering reinforcement learning, encoding and retrieval within the same study will be important for establishing specific deficits. Finally, it is critical to consider stimulus modality. In particular, researchers should consider whether stimuli have a social or nonsocial theme. None of the first four theories discussed here account for the specificity of state affective deficit in social versus nonsocial stimuli. We believe this represents an important knowledge gap.

In conclusion, it is worth acknowledging the gains that have been made in understanding affective deficits in schizophrenia. While research has explored this issue for over a century, it is within the last decade that significant strides have been realized. This reflects renewed interest in the affective symptoms of schizophrenia—symptoms that are largely intractable with current treatments. It is, however, also clear that these gains have been possible by integrating findings from a broad range of disciplines. Borrowing theory and methods from cognitive and social psychology, basic emotion, neuroscience/neurobiology and other fields has paved the way for new insights. Further advances in understanding the affective deficits in schizophrenia will only be possible through similar multidisciplinary research.

References


