

COGNITIVE APPROACHES TO SCHIZOPHRENIA: Theory and Therapy

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■ **Abstract** A theoretical analysis of schizophrenia based on a cognitive model integrates the complex interaction of predisposing neurobiological, environmental, cognitive, and behavioral factors with the diverse symptomatology. The impaired integrative function of the brain, as well as the domain-specific cognitive deficits, increases the vulnerability to aversive life experiences, which lead to dysfunctional beliefs and behaviors. Symptoms of disorganization result not only from specific neurocognitive deficits but also from the relative paucity of resources available for maintaining a set, adhering to rules of communication, and inhibiting intrusion of inappropriate ideas. Delusions are analyzed in terms of the interplay between active cognitive biases, such as external attributions, and resource-sparing strategies such as jumping to conclusions. Similarly, the content of hallucinations and the delusions regarding their origin and characteristics may be understood in terms of biased information processing. The interaction of neurocognitive deficits, personality, and life events leads to the negative symptoms characterized by negative social and performance beliefs, low expectancies for pleasure and success, and a resource-sparing strategy to conserve limited psychological resources. The comprehensive conceptualization creates the context for targeted psychological treatments.

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INTRODUCTION

The clinical presentation of schizophrenia includes four separate sets of symptoms or behaviors: delusions, hallucinations, thinking/discourse disorder, and “negative symptoms” (John et al. 2003). Although factor analyses have consistently demonstrated that the first two sets load on a common factor, often named “reality distortion,” it is difficult to discern the meaningful connections among the sets of symptoms. Further, the relation of these symptoms as well as broader cognitive functions, such as reality testing, to the neurocognitive impairment is not clear. We attempt to address the following question in this chapter: What processes can account for the diverse apparently unconnected symptomology and its relation to structural and neurophysiological abnormalities? We explore this question in terms of the interaction of inadequate cerebral functioning leading to aversive life experiences, excessive psychophysiological reactions, and the consequent cognitive, affective, and behavioral abnormalities characteristic of schizophrenia.

Recent findings of improvement of the schizophrenic symptoms in response to cognitive therapy adjunctive to pharmacotherapy (Dickerson 2004, Rector & Beck 2001) suggest that the psychological understanding obtained from these studies and the experimental literature (Bentall 2003) might help to integrate the clinical and experimental findings from these different conceptual levels. We review these clinical trials and briefly describe the therapy.

Although experimental findings are insufficient to warrant an empirical review of the cognitive approaches to the basic mechanism of schizophrenia, a theoretical discussion would provide the framework for understanding the phenomenology and cognitive therapy of schizophrenia. The emphasis of this chapter, consequently, is on the analysis of the development, symptomatology, and therapy from a cognitive perspective. Where available, supportive empirical findings are presented. Based on the analogy of impaired cardiac function, the concepts of cognitive “insufficiency,” “decompensation,” and “failure” can be applied to the complex interaction of the predisposing neurobiological, environmental, cognitive, and behavioral factors in the development of schizophrenia.

THE DEVELOPMENT OF SCHIZOPHRENIA

A large literature has documented the impact of disturbances in the activity of neural circuitry on components of perception, cognition, and behavior. These disturbances have produced measurable cognitive impairments in executive function

and working memory. The cumulative effect of these impairments and other as yet unidentified impairments may also contribute to a reduction in the available pool of cognitive resources, as described by Nuechterlein & Dawson (1984).

Conceptualizing the pathogenic process from a clinical vantage point, we propose that the neurocognitive impairment in the premorbid state makes the schizophrenia-prone individual vulnerable to aversive academic/work and interpersonal experiences (for example, substandard school performance and social difficulties). These stressful conditions, in turn, lead to dysfunctional beliefs (for example, "I am inferior") and, consequently, dysfunctional cognitive appraisals and maladaptive behaviors (for example, social withdrawal). These problems evoke more aversive experiences that consolidate the dysfunctional beliefs and behaviors. The repeated dysfunctional appraisals increase the amount of psychophysiological stress.

The at-risk individuals, as well as patients, show a number of specific impairments demonstrated in neurocognitive tests: attentional problems, impaired working memory, and defective executive function (Nuechterlein & Dawson 1984, Walker et al. 1998). These indices of "cognitive insufficiency" impede the individuals' academic and social adjustment and when combined with hypersensitivity to stress, create relevant conditions for the development of schizophrenia.

The physiological component of the excessive stress reaction proceeds from an activation of the hypothalamic-pituitary-adrenal (HPA) axis leading to a cascade of corticosteroids producing a neurotoxic effect on the brain, especially on the hippocampus and prefrontal lobes. The release of corticosteroids also activates the dopaminergic system, which contributes to the development of delusions and hallucinations. Figure 1 illustrates the interaction of brain dysfunction with external events and psychological and physiological responses. The repeated cycling of these psychological and physiological reactions leads to "cognitive decompensation" and the clinical syndrome of schizophrenia.

The empirical evidence for the physiological overreaction to stress has considerable support in the literature (for example, Corcoran et al. 1995, Dickerson & Kemeny 2004, Walder et al. 2000). Schizotypal individuals, for example, show thinking problems analogous to those found in schizophrenics and similarly overreact physiologically to stress (Walker et al. 1998). Further, social rejection and low self-esteem are correlated with excessive cortisol release (Dickerson & Kemeny 2004).

Although specific regions of the brain and specific functions (for example, short-term memory and executive functioning) have been shown to play a central role particularly in the formation of negative symptoms and disorganization (Heydebrand et al. 2004, Kerns & Berenbaum 2003), these studies do not account for the delusions, hallucinations, and impaired reality that are characteristic of schizophrenia.

The concept of defective localized, specialized functions of the brain has dominated the research on higher brain function in schizophrenia in recent years. Phillips & Silverstein (2003) point out that these locally specialized functions must be complemented by processes that coordinate them, and propose that impairment

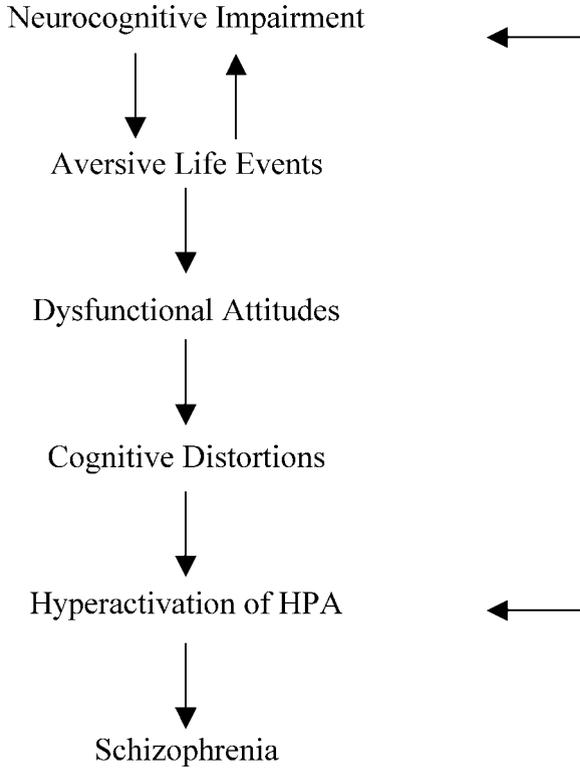


Figure 1 A diathesis-stress model of development of schizophrenia.

of these coordinating processes may be central to schizophrenia. They suggest that this important class of cognitive functions can be implemented by mechanisms such as long-range connections within and between cortical regions that activate synaptic channels and synchronize the oscillatory activity in the brain. The cognitive capabilities that these mechanisms provide have been shown to be impaired in schizophrenia.

A global view from the perspective of the impairment of the total integrative function of the brain can clarify the question of how the disorder develops. Given the limitations on the cognitive capacity of the brain, external stressors increase the cognitive load and divert resources to buffer the impact of the stressors and consequently reduce the available resources. Although certain cognitive functions such as those measured by intelligence tests may be preserved in some cases, the relative impairment of complex effortful psychological functions such as self-reflectiveness, self-monitoring, correction of misinterpretations, and responsiveness to corrective feedback from others facilitates the development of

dysfunctional ideas and impedes the development of interpersonal skills. Patients with schizophrenia may successfully utilize these cognitive skills in evaluating their own relatively neutral ideas or the erroneous ideas of others, but they generally lack the cognitive capacity to apply them to highly charged emotional ideas, particularly those associated with delusional beliefs. This deficiency leads to the clinical concepts of “impaired insight” and “deficits in reality testing.”

Reduction in available resources is reflected in a reduction of motivation, expressive affect, behavior, and cognition (the “negative syndrome”). The profound disengagement manifested by alogia (meaning “without speech”), affective flattening, and anergia are in the service of resource sparing. A similar weakening of cognitive inhibition appears in the thinking disorder (or discourse disorder) characterized by derailment and loss of referents, especially when the patient is experiencing external stress or is discussing an emotionally salient subject. Total disorganization may be conceptualized as an expression of “cognitive failure.”

The cognitive insufficiency obviously varies over time and is frequently improved, or compensated for, by pharmacotherapy. Individuals predisposed to schizophrenia often compensate for their deficits; for example, they protect themselves from stressful situations through social isolation (e.g., Lencz et al. 2004). Nonetheless, stressful situations or neurotoxic substances can lead to cognitive decompensation and the recurrence of symptoms. There is experimental support for these clinical constructs, as research has demonstrated that under cognitive load the cognitive functioning of patients with schizophrenia deteriorates (Melinder & Barch 2003, Neuchterlein & Dawson 1984). Defective pupillary responses (Granholm et al. 1987) and antisaccadic reactions (Curtis et al. 2001) are postulated indirect evidence of attenuated cognitive resources.

The interplay between attenuated resources, dysfunctional attitudes and appraisals, and salient life events in the development and maintenance of the major symptom domains in schizophrenia are addressed below.

DISORGANIZATION DISORDER

It is often difficult to discriminate formal thought disorder and disorganized speech since only speech is accessible to the listener. Hence, researchers (e.g., Docherty et al. 2003, Maher 2003) have referred to the terms “discourse disorder” or “aberrant utterance” based on observable data. In keeping with current practice, however, we refer to this cluster of symptoms as “disorganization disorder,” which may be manifested in a number of ways: speech incompetencies such as derailment, drifting, inexact or inappropriate selection of words, concrete responses, over-inclusive associations, and frank disorganization (Andreasen 1979, Chapman & Chapman 1973). Other observations include difficulty in maintaining a set, switching sets, and maintaining a context. When contextual cues are weak, patients with schizophrenia fail to use these cues (Chapman et al. 1977), but when the cues are stronger, the patients’ performance will approach that of normals. Also, patients

with schizophrenia prefer literal interpretations of proverbs to a more correct, abstract response, but may select a more abstract response when it is particularly common (Titone et al. 2002).

The clinical observations and experimental work suggest that the various aspects of formal thought disorder and/or communication disorder can be attributed to circumstances that place a burden on the patients' cognitive capacity. In some cases, the patients respond to the demands by conserving their attenuated resources (for example, choosing the easiest—although out of context—response, skipping over the more effortful part of discourse such as providing bridges and referents, and favoring literal over abstract interpretations). In other instances, they ramble, experience intrusions of seemingly irrelevant words or thoughts, and run on excessively in word-association lists.

The research findings and clinical observations can be considered in terms of two processes resulting from the limited cognitive resources: resource sparing and disinhibition. When the accessible resources are low, speech production becomes more difficult. Adhering to the rules of social discourse in preference to one's own train of thought and personal associations requires the expenditure of resources. The attenuation of resources for continuous, ongoing deployment of resources to a socially oriented activity such as carrying on a conversation results in reduction in bridges and referents between sentences (Docherty et al. 2003).

Resource sparing can also explain the patients' choice of the strong associates to stimulus words when these are inappropriate to the context. Since searching for the more appropriate association is more demanding, the choice of easy responses (literal or popular) reflects a resource-sparing cognitive bias. It is likely that patients find the demanding tasks aversive, and consequently they shy away from the extra burdens in favor of easier verbal strategies. Poverty of speech may also be a response to excessive demands on resources. The patients either spare their limited resources by limiting the duration of speech, and thus avoiding disorganization, or by stretching limited resources to produce more lengthy speech, but allowing disorganization to occur (Melinder & Barch 2003).

Intrusions result from associations that are more personally meaningful and highly charged than the subject of an ongoing conversation. If resources are not fully available for inhibiting such intrusions, the pressure of cognitive factors override, to some extent, the demands of the interpersonal situation. The resources to curtail these intrusions are further depleted when upsetting material is discussed with the patient. Worry and other disturbing concerns become more salient relative to external demands and divert the patients' attention to more meaningful associations. Thinking-disordered patients manifest an increase in discourse incompetencies when they discuss emotionally charged topics (Docherty et al. 2003). Similarly, they show intrusions of speech when criticized by their family (Rosenfarb et al. 1995).

Inhibition of intrusions also imposes a drain on resources. Disinhibition is observed clinically in phenomena such as derailment, drifting, and intrusions (Andreasen 1979). Certain associations may have a stronger charge than the topic

a patient is discussing, and owing to the patient's lack of inhibition, break into the speech. The diversion of resources to emotionally charged ideas and affect shifts the balance between goal-directed thinking and disinhibition of irrelevant ideas and speech in favor of the latter. There is research evidence indicating patients do not show the usual inhibitory responses in experimental situations (Peters et al. 2000) or loss of prepulse inhibition (Braff et al. 1992).

DELUSIONS

Although delusions are defining characteristics of schizophrenia, they are also observed in the context of a number of other psychiatric disorders such as depression, obsessive-compulsive disorder, and body dysmorphic disorder. It is an open question as to whether delusions in these disorders are formed in the same way as those observed in the context of schizophrenia. Nonetheless, delusions share many common characteristics irrespective of the specific disorders in which they occur.

Delusions are characterized by a variety of unique cognitive patterns and problems including externalizing, internalizing, and intellectualizing biases; categorical thinking; emotion-based and somatic-based reasoning; and inadequate cognitive processing such as jumping to conclusions. In addition, reality testing, particularly of delusional interpretations, is attenuated. During the formation or exacerbation of organized delusions, the patients' interpretations of their experience are controlled to a significant degree by biased information processing. The usual processing functions are preempted in these pathological states by hypersalient cognitive structures, or schemas, which distort the inputs (from the stimulus situations) and products (cognitions). These cognitions—interpretations, evaluations, and self-instructions—are shaped by idiosyncratic beliefs that constitute the content of the cognitive schemas. Since the schemas in delusions are rigid and relatively impermeable to ordinary corrective feedback, they shape the patients' perceptions of their personal world to the exclusion of consensually shared meanings with other people. Since the beliefs are deviant, the interpretations are deviant.

The understanding of delusions may be facilitated by examining their form as well as their content. The dimensions of the beliefs that generally impact individuals' information processing are essentially the same in delusional beliefs. Moreover, the formal dimensions of the beliefs observed in the nonpsychotic conditions, such as depression and anxiety, are also found in delusions (Garety & Hemsley 1987, Hole et al. 1979). These dimensions include (a) pervasiveness (the proportion of patients' interpretations that have a delusional stamp), (b) conviction (from absolute to minimal), (c) preoccupation, (d) lack of insight (the relative impermeability to contradictory evidence, logic, or reason), and (e) their impact on behavior. Although the etiology of poor insight in schizophrenia is still unclear, recent studies have supported the hypothesis that neuropsychological deficits are at the root of very poor insight that persists over time (Amador & David 2004).

A scale developed to measure the patients' ability to reflect on and distance themselves from their delusional thinking correlated well with the Birchwood insight scale and was found to be a robust predictor as well as a mediator of improvement in a study on cognitive behavior therapy for schizophrenia (Beck & Warman 2004).

Hypersalient beliefs tend to co-opt information processing and produce a high degree of conviction, pervasiveness, preoccupation, and impact. For example, a patient who believed totally (conviction) that everyone was an FBI agent (pervasiveness), could not consider that the belief might be wrong (inflexibility), and, as a result, hid in his room (impact). A test of the patients' inflexibility is their response to the "hypothetical contradiction of the delusions" (Hurn et al. 2002). Patients' willingness to consider contradictory information has been found to improve their prognosis (Garety et al. 1997).

The Role of Biases in Delusional Beliefs

The criterion for labeling an unusual, even bizarre, idea as delusional may be difficult to determine simply from the idea's content. Throughout history, many new ideas that were considered far-fetched or even irrational eventually became accepted. A clear marker that distinguishes delusional from nondelusional thinking is the coupling of two extreme biases: preemptive self-centered focus and external locus of causation. A critical measure of delusional thinking is the degree to which the individual perceives himself as the central component or object of events (egocentric bias). A person who endorses telepathy as a valid phenomenon, for example, will arouse suspicion of psychosis if he confronts people with complaints that they are reading his mind. He incorporates a popular theory into his system of beliefs about himself and uses the theory to explain his experiences with other people. The egocentric bias is also manifested in the patients' tendency to relate irrelevant events to themselves and consequently arrive at false conclusions. Thus, the sounds of whirling motors represent attempts by enemies to control their behavior, peoples' muffled voices are criticisms of them, and flashes of light are signals from God. The patients' personal domain may expand to encompass their entire social environment—everyone has personal relevance to the patient.

Patients with paranoid schizophrenia are especially prone to make extraordinary explanations for their ordinary experiences. Because of a powerful externalizing bias, they forgo plausible explanations in favor of unlikely or impossible external attribution. Thus, difficulty in performing manual tasks may be attributed to interference by alien forces, a headache to beams from a satellite, and command hallucinations as orders from God. Unlike most individuals with hypochondriasis who ascribe normal somatic sensations to medical illness, for example, paranoid patients may ascribe their subjective experiences such as aches or fatigue to the manipulation of an external entity. They focus their attention particularly on external (usually social) stimuli for signs of potential danger, instructions, or (in some cases) rewards. Like a soldier in a combat zone, they are hypervigilant to any environmental event that could signify danger. They detect danger when there is

none; they perceive enemies when there are none. Research supports these clinical observations: Individuals with paranoid delusions attend preferentially to social threat-related stimuli (Bentall & Kaney 1989, Fear et al. 1996), especially when the threat stimuli are emotionally salient (Kinderman 1994).

Their scrutiny progresses beyond the concerned vigilance of socially phobic patients. It is generally based on the assumption that other people are malevolent; therefore, they are continually suspicious of others' motives. Interestingly, despite their hypervigilance, the patients' actual perception of potential persecutors is often fuzzy. They seem to perceive a projection of their own images of what they believe their persecutors are like. A female patient, for example, believed that most of the people on her psychiatric ward were members of the Philadelphia police force. In a sense, she had "homogenized" their characteristics in a way similar to negative stereotyping.

Patients with grandiose ideas may similarly interpret environmental events as evidence of their extraordinary presence. A homeless patient perceived that other people were hostile to him and therefore he was Jesus, about to be martyred by them. When pressed, he was unable to describe their specific appearance—he could describe them only in general terms as enemies.

Based on clinical reports and experimental work, we propose that although paranoid patients are hypervigilant for threatening cues, they quickly turn their eyes away from the supposedly threatening stimuli. Phillips & David (1997), for example, found that paranoid patients spent less time looking at pictures with threatening poses than did nonparanoid patients. Presumably, this avoidant pattern interferes with their effective processing of the data, thus leaving their delusional belief uncorrected.

Of special significance is the imaginal creation of external entities (whether a computer chip in the brain or an influencing machine) engaged in aggressive activity to influence, harm, or protect the individual (intentionalizing biases). Thus, psychotic patients conceive of a causal chain proceeding from the external agent directing attention to the patient to intentionally influencing the patient (either for better or worse). The causal sequence is accepted by the patient as an incontrovertible fact. In contrast, the typical nonpsychotic patients are fixated on self-evaluative concerns rather than factual entities and sequences. A depressed patient, for example, might react to a poor report on the job or at school with the evaluative statement, "I am stupid," the patient with schizophrenia would respond with a factual declaration, "They have a conspiracy against me." Similarly, patients with obsessions interpret the intrusions as a sign that they are mad or dangerous while the patients with schizophrenia interpret them as a sign of mind manipulations from an external entity.

There appears to be a progression in the development of delusions from ordinary subjective experiences to delusional conclusions. Paranoid thinking often follows an inexorable sequence from fear or concern, to a feeling of self-consciousness and anxiety, to the actual occurrence of the feared event. A patient, for example, believed that the husband of each woman with whom he flirted would be jealous

and he eventually began to believe that men on the street were these jealous husbands who wanted to attack him. Another patient reported drug dealers to the police. He began to fear that the police were in cahoots with the drug dealers and subsequently decided that innocent-appearing strangers were undercover policemen, that ordinary vehicles were unmarked police cars, and consequently, that he was in imminent danger of being arrested or eliminated.

Conspiratorial delusions of being followed by members of a hostile group (CIA, Mafia, terrorists) are frequently preceded by a sense of being conspicuous in a group—often of appearing different in some way—associated with feelings of self-consciousness and anxiety. The patients, like the social phobics, then conclude because of this anxiety that other people are watching them and look away from them (emotion-based reasoning). Any concerns that they may have about being targeted by the inimical group intensifies their self-focus and subsequent physical sensations, such as tautness of the face, a tension in the eyeballs, and general uneasy feelings. Unlike nonpsychotic individuals, they interpret these sensations as evidence that they are under observation from unfriendly entities. Some patients who already believe they have enemies conclude that they are being watched by hidden video cameras wherever they go, while still others may decide that they are being followed by enemy agents. As a rule, they capitalize on coincidences and selectively attend to stimuli to validate their beliefs (confirmation bias).

A number of studies have shown that paranoid patients have a specific kind of explanation for ambiguous distressing events. Whereas some people (such as depression-prone individuals) may attribute the cause to their own personal deficiency and others will account for this by attributing them to circumstance (external-situational), paranoid patients have an exaggerated tendency to blame extraneous factors, particularly other persons' noxious traits ("external-personal"; Kinderman & Bentall 1996, 1997). Experimental studies comparing paranoid patients to depressed and nonpsychiatric controls find that paranoid patients show this excessive bias for external attributions for negative events, especially when the event is self-referential. Studies that distinguish between situational external attributions and personal external attributions further demonstrate that paranoid patients are more likely to blame people as opposed to situations (see Garety & Freeman 1999 for review). As such, they find it difficult to make "situational" attributions that an aversive event could be due to circumstances, chance, or accident. The tendency to make explanations in terms of external factors, even when internal explanations are more obvious, seems to be almost reflexive for normal individuals—the "primary attribution error" (Gilbert & Mallone 1995, Heider 1958).

In an elegant experiment, Gilbert (1991) also demonstrated that normal individuals tend to respond to events by making automatic external personal attributions. That is, they explain the occurrence in terms of the other individual's personality traits. Yet, given additional information, they generally correct their initial interpretation. However, if their cognitive resources are strained, they do not make this correction. The attentional and memory deficits during a psychotic episode limit

the patients' ability to focus their attention on alternative explanations, as Bentall (2003) points out. The same kind of limited resources will also affect patients' ability to make other corrections of highly charged interpretations. Although this conclusion is an extrapolation from data received from normal individuals, it does seem to fit the attributional problems of psychotic patients. Gilbert's model is currently being tested on delusional patients (Bentall 2003).

Although studied separately, the various biases are apparently different aspects of the same mental construction. The content of the delusional mode is organized into a story or plot consisting of a triad of internal representations. These include the subject: an active agent or agents; the agent's motivations: malevolent or benevolent; and the object: the patient. The basic scenario may appear in different ways depending on external circumstances. Moreover, the components may vary; for example, the subjects may change or their motivations may get worse or better. External stimuli such as traffic, conversations, or television programs are interpreted as signals to the patient (internalizing bias), and these external entities are represented as having a malevolent (or benevolent) motivation (intentionalizing bias). Also, internal experiences such as hallucinations, obsessive thoughts, or somatic sensations are attributed to an external agent (externalizing bias). As long as the mode is activated, the delusional plot is played out and leads to relevant affective (anger and/or anxiety) and behavioral (attack/flee) consequences.

Categorical Thinking

The homogeneous image of the Enemy reflects a more general disposition to engage in categorical thinking. The patients create an imaginary category to define the persecutory agents and then apply it vigorously to "suspicious" individuals. The paranoid category may be broad with loose boundaries (e.g., Philadelphia police) or narrow and well defined (e.g., coworkers or family members). Once the category is created, however, the individual characteristics of the identified persecutors are blended into the category. The same categorical thinking extends to invisible entities such as the Devil, spirits, or dead relatives. When the image of the defined entity has been created, the patients either "see" them or feel their invisible influence.

Related to their categorical thinking is the tendency of some patients to conceive of certain perceptual experiences in concrete materialistic terms. Further, their metaphorical descriptions of an experience become reified. A patient with a variety of dissociative symptoms, including a pervasive sense of unreality, initially described these in metaphorical terms as a numbness of the brain. Later when he transitioned into psychosis, he believed that his brain was literally dead. Also, many patients who think their food tastes different than previously perceive the sensation as poison. A patient who felt pressure in his eyeballs when he looked at (actually stared at) other people believed that their eyes emitted actual rays to his eyes. The materialistic model applied by psychotic patients contrasts with the medical model used by many nonpsychotic patients and normal individuals.

Patients with hypochondriasis, for example, view their somatic sensations (pain, stiffness, fatigue) as signs of a medical illness. Individuals with anxiety, depression, or obsessive-compulsive disorders, on the other hand, describe their symptoms in psychological terms. They could be said to use a “mental” model. These more normal models help define the problem and point to solutions. The materialistic model simply intensifies existing problems.

Patients with nonpsychotic disorders, moreover, recognize that their distress is generated within themselves, and that their own reactions to situations or to subjective experiences constitute a large part of their problem. In contrast, patients with schizophrenia locate the source of their symptoms as outside themselves and generally ascribe their distress to the work of a specific agent or agents. They construe the cause of their symptoms in terms of a definable entity that can be assigned to a concrete category—in preference to “natural” but less-defined constructs, such as emotions or beliefs.

An interesting theoretical challenge is the determination of what mechanisms or deficiencies facilitate crossing the boundary between nonpsychotic experiences, such as extreme self-consciousness, to delusional beliefs, such as being observed or followed. Considerable research has also focused on the problem of understanding other people’s thinking when the patient is in a psychotic episode. Generally labeled “Theory of Mind” deficits, this problem also has been used to explain the external attributional style. Taylor & Kinderman (2002) found that students who performed relatively poorly on a Theory of Mind task showed more paranoid-style external-personal attributions than did students who performed well. Although the study was limited to students, it does show the relationship between Theory of Mind skills and attributions. This study is currently being repeated with patients with psychosis (Craig et al. 2004). Presumably, problems in understanding other people’s motivations combined with the negative bias feed into beliefs that are malevolent.

Relation of Delusions to Dysfunctional Attitudes

The seeming bizarreness of the content of delusions and hallucinations becomes more comprehensible when understood within the interpersonal context of a person’s life. Commonplace concerns about being rejected and manipulated may become amplified into paranoid delusions; hypervigilance for and preoccupation with somatic sensations develop into somatic delusions. Several authors have found continuity between worry and concerns prior to psychosis and the continuity of the delusion (Harrow et al. 1988).

The understanding of predelusional psychological problems provides direct clues to the formation and content of the delusions. For instance, we have found that grandiose delusions may develop as a compensation for an underlying sense of loneliness, unworthiness, or powerlessness. Many of the patients with grandiose delusions have experienced prior life crises characterized by a sense of failure or worthlessness and subsequently begin to think of themselves of being famous, divine, or all-powerful. The proximal antecedents of a paranoid delusion, on the

other hand, may include the fear of retaliation for having done something that offended another person or group (Beck & Rector 2002).

Consistent with the hypothesis that paranoid delusions are associated with beliefs reflecting interpersonal vulnerability, a cross-sectional study demonstrated that excessive need for others' acceptance and approval (i.e., sociotropy), as measured by the Dysfunctional Attitude Scale, significantly predicted the presence and severity of persecutory delusions (Rector & Beck 2001). Among the items significantly related to the suspiciousness/persecution item on the Positive and Negative Symptoms Scale (Kay et al. 1987) but not to the passive/apathetic/social withdrawal item were: "I cannot be happy unless most other people I know admire me," "If others dislike you, you can't be happy," "It's best to give up your own interests in order to please other people," and "My happiness depends more on other people than it does on me." These items, generally considered sociotropic, show the importance of other people to paranoid patients. The precise chain connecting sociotropic items to paranoid items has not yet been determined.

It could be speculated that individuals with a high investment in being admired or loved might be more vulnerable to aversive events denoting depreciation or rejection. The subsequent pain from the trauma could lead to a defensive strategy of suspiciousness and hypervigilance. These in turn would strengthen beliefs that other people are treacherous and hostile.

Reality Testing

The delusions in schizophrenia are relatively impervious to reality testing. The patients are unwilling or unable to distance themselves from their delusional thinking and beliefs or even to consider that they might be wrong. In psychiatric terms, they have "impaired insight." Patients with depression, in contrast, are generally amenable to examining their negative interpretations and when questioned skillfully can acknowledge their errors in thinking and correct them (Beck et al. 1979). The capacity to recognize cognitive distortions and substitute more realistic appraisals has been termed reality testing or cognitive insight (Beck et al. 2004). Cognitive insight scores involve categories of self-reflection, reception to corrective feedback, and overconfidence; the scores in these categories discriminate schizophrenia from other psychotic disorders (Beck et al. 2004).

As noted above, a major question in understanding the phenomenology of delusions is why patients continue to adhere to delusional beliefs despite their bizarre or at least improbable, nature, and the lack of consensual validation. Why don't patients with psychosis draw on these life experiences and conventional notions of cause and effect and probability as well as correctional feedback from other people to question their unreasonable and frequently distressing ideas? Moreover, why can't patients with psychosis draw on the same important cognitive skills they use to challenge the delusional beliefs of other patients but not their own?

An approach to resolving this dilemma is to consider that the generation and maintenance of beliefs, as opposed to reality testing them, represent two totally

different psychological domains. In contrast to the automatic, reflexive, parsimonious notion of the primal mode, the self-questioning mode is reflective, deliberative, less automatic, and effortful (Beck 1996). Questioning beliefs, an important function of this system, requires first and foremost distancing from the beliefs (reorganizing them as mental products rather than reality), evaluating them, examining the evidence, and conceding alternative explanations—all of which draw on the individual's resources. If the beliefs are highly charged (and consequently carry a high level of connection), it is difficult to activate the self-questioning mode sufficiently to have an impact on them. Similarly, if resources are strained or unproductive, they may be insufficient to conduct reality testing. Since therapeutic interventions can often prime the self-questioning mode, it seems that the relative inaccessibility of the corrective information is the result of resource sparing rather than absolute absence.

HALLUCINATIONS

The formation and persistence of auditory hallucinations requires consideration of a range of psychological factors, including the natural continuum of hallucinatory experience, the overlap between stream of consciousness and content of voices, the role of predisposing factors, mechanisms in the threshold of perceptualization, salience of internal representations, cognitive and reasoning biases, delusional beliefs about the voices, as well as the appraisal of alternative behavioral responses. As detailed more fully elsewhere, these subcomponents connect and are guided by the broader cognitive organization of the person to produce hallucinated percepts (Beck & Rector 2003).

Hearing voices is the most commonly reported symptom of schizophrenia, occurring in approximately 73% of patients with the diagnosis (World Health Organization 1973). Contrary to popular belief, however, hallucinations are not specific to a diagnosis of schizophrenia. From 4% to 25% of the population report having auditory hallucinations sometime in their lifetime (e.g., Tien 1991). Auditory hallucinations are commonly reported during periods of bereavement and in adverse situations such as solitary confinement, hostage taking, and following significant sleep deprivation. Hallucinations also occur in a wide variety of disorders, including psychotic depression, manic-depressive disorder, and posttraumatic stress disorder. Auditory hallucinations have also been reported in a very broad range of organic and psychiatric conditions including neurological disorders, hearing loss, deafness, and tinnitus. These observations suggest that hallucinations lie on a continuum with normal experience. The main difference in the case of the psychotic hallucinations is that they tend to be more negative, are less responsive to corrective feedback, and the content is taken at face value despite evidence to the contrary (Beck & Rector 2003).

Voice content reported by patients is often similar to the automatic thoughts that are observed in other psychiatric conditions, such as depression, mania, and

social phobia, and include comments, criticisms, and ruminations. They may also be similar to the intrusive thoughts in obsessive-compulsive disorder (Baker & Morrison 1998). Thoughts in the first person ("I am a loser") may transition into a voice in the second person ("You are a loser"), but critical automatic thoughts are frequently in the second person. Many automatic thoughts are directed at the patient as an object: "You are dumb." Third-person voices frequently develop from ideas of reference. A patient noticing people looking at him thought, "They are talking about me," and then heard their voices saying, "He's a slob." A differentiating feature of the hallucinated cognitions is that the self is perceived as the object (e.g., "You should go for a walk") rather than the initiator or subject (e.g., "I think I'll go for a walk").

So why are patients with schizophrenia particularly predisposed to experience some thoughts as externalized voices? Patients with schizophrenia appear to have an unusual propensity for imagery in the auditory modality. Several studies have supported the hypothesis that hallucination-prone subjects or hallucinating patients have an unusual propensity for unintended or involuntary imaging in the auditory and visual domains. Bentall & Slade (1985) administered an auditory signal-detection task using white noise and periodic intrusions of a voice and found that hallucination-prone participants were significantly more likely to perceive a voice when it was not present (false alarm). A replication of the same experiment with hallucinating and nonhallucinating patient participants found that the hallucinators showed significantly more false positives in voice recognition than did the nonhallucinators. A similar study reported by Rankin & O'Carroll (1995) also found that subjects scoring high on hallucination-proneness overestimated the presence of a verbal signal. An earlier study by Margo et al. (1981) indicated that hallucination-prone individuals were more likely than was a control group to experience spontaneous auditory hallucinations when exposed to white noise.

Since hallucinators are prone to focus excessive attention on auditory stimuli, their hypervigilance may be reflected in expectancy for the occurrence of a voice. Aleman (2001) suggested that imagery and perception are closely related in hallucination-prone subjects, and thus are more difficult to distinguish from one another. He also presented some evidence showing that when volitional imaging is more salient than an actual perception, a patient is more likely to be an active hallucinator. Thus, it is the relative balance between imagery and perception that is relevant to the formation of hallucinations. Further, Shergill and colleagues (2001) utilized functional magnetic resonance imaging with hallucinating patients and concluded that auditory hallucinations may be mediated by distributed networks in cortical and subcortical areas. They also pointed out that the pattern of activation observed during auditory hallucinations was remarkably similar to the pattern of activation by healthy volunteers when they were asked to imagine another person talking to them (auditory verbal imagery). This finding provides support for the hypothesis that auditory hallucinations are an expression of "internal speech" that has become externalized. Hoffman (2002) also reports preliminary evidence that Broca's and Wernicke's areas are excessively coupled in voice-hearers most

of the time (i.e., the time course of their activations are more correlated than in normals).

The inconsistency in the experience of hallucinations suggests a corresponding variability in the threshold for perceptualization. The apparent on-off character of voices suggests the functioning of a threshold for perceptualization. The threshold may vary considerably depending on endogenous and external factors. It is lowered, for example, by fatigue, stress, reduction in external stimulation, and emotional factors such as anxiety, anger, and depression. The other major contribution is the pressure from the hypersalient cognitions. Thus, the combination of factors may result in a hypersalient cognition “crossing the sound barrier” (Beck & Rector 2003).

Although the preceding discussion helps to illuminate why certain thoughts become perceptualized as external voices, another puzzling aspect to consider is how internally generated phenomena can be experienced as identical to externally derived phenomena. Patients with ideas of reference misperceive other peoples’ talk (and other sounds such as a cough or a sneeze) as directed at them. The inner representation of “What people think of me” overrides the actual sensory stimulus and produces an auditory image (“He’s a loser”) just as real as the unencumbered transmission of actual sound. Hypersalient representations can produce perceptions in the absence of input from the sensory organs: hallucinations. When the internal representations co-opt the cognitive processing system, they create a false reproduction of the external world of sight and sound—a visual or auditory hallucination. Certain internal representations expressed in the form of cognitions, memories, or visual images are hyperprimed. At the same time, the usual constraints on the formation of endogenous perceptualization are diminished (disinhibited). The combinations of these factors subvert the normal functioning of the internal processing systems and produce hallucinations.

Finally, the study of hallucinating patients has demonstrated the presence of a cognitive bias to misattribute internal cognitive events to an external source. This externalizing bias is similar to the externalizing bias in paranoid delusions (Beck & Rector 2002, Bentall 1990). Studies by Rankin & O’Carroll (1995) and by Morrison and his colleagues (Baker & Morrison 1998, Morrison & Haddock 1997) found that hallucinating patients with schizophrenia, compared to nonhallucinating patients with schizophrenia, were significantly more likely to misattribute the source of self-generated speech to an external source.

Another question that arises is: Why is it that these initial errors in attribution are not corrected with the passage of time and the emergence of disconfirming evidence? As pointed out by Johns and colleagues (2002), people who hear sounds as a manifestation of tinnitus, or hear music or voices related to the aging process, may check the radio or television set or test their perceptions with others. In contrast, patients with schizophrenia do not go through processes of reality testing. Generally, patients hearing voices do not check or seek others’ opinions regarding the validity of their interpretation. If the voice *seems* to be real (that is, from an external agent) then it *is* real (that is, it cannot be internally generated). They seem

to lack the normal propensity to question the reality of an unusual experience. To override spontaneous appraisals and judgments requires not only extra energy but also sophisticated forms of self-correction—strategies that have been poorly developed in patients with schizophrenia. Other reality-testing functions also appear weak in patients with schizophrenia: the ability to consider alternate explanations, to suspend judgment pending accumulating more information, withdrawing attention from the hallucinations and delusions—viewing reasoning biases objectively (Beck & Rector 2003). These reality functions are not totally absent but are simply hypoactive during the psychotic episodes. In fact, when the psychotic episodes have passed, the patients frequently recognize that their erstwhile hallucinations were actually their own thoughts.

Because the patients are susceptible to premature closure in their judgments, they stick to the belief that the voice is “real”; that is external in origin. They follow the easier route of accepting real-seeming perceptions as real instead of undertaking the more taxing task of reconsidering and discounting their reality. However, even when they do not identify a particular agent, they do believe that the voices are coming from “somewhere.” The belief in the external origin of the voices hardens as the patients begin to accumulate supporting evidence (e.g., consequential reasoning). For example, a patient has a command hallucination telling her that she must run errands or she will regret it. If she does not comply, she hears a voice scolding her, and she feels bad and does indeed regret not complying. If she does comply, the voice may praise her—thus reinforcing the notion that the voice must be real. Also, the mere repetition of the voices impresses on the patient that they must be real and (generally) should be taken seriously. The lack of a critical attitude toward their mental constructions reflects a more general lack of awareness of their mental content. Further, if a particular experience is “real,” there is no motivation to question it.

The persistence of hallucinations and the belief that they are externally caused is facilitated by a number of factors. Birchwood & Chadwick (1997) suggest that the activation of the voices triggers delusional beliefs about the presumed agency and power of the voices, which then intensify their importance. The particular beliefs activated by the voices are not necessarily evident from the content of the voices. The content, for example, may be negative (“You always mess up”) and yet, because they have a benevolent belief about the voices, the patients may put a positive twist on them (“The voice wants to help me”). It is clear, however, that both the content of the voices and the beliefs about them influence affect and behavior. Further, beliefs may take a paranoid form, such as “They are after me”; a depressive form, “God is unhappy with me”; or a fearful form, “The doctors want to poison me.” As with many other aspects of the hallucinations, these beliefs themselves prompt the patient to focus on the voices in an effort to block them or damp them down. However, this increased attention to the voices tends to accentuate their potency and frequency and, consequently, to confirm the validity of the belief.

Patients who hear voices also engage in behaviors intended to mitigate the activation of voices, to neutralize the perceived negative consequences of hearing

voices, and/or to appease the perceived agent of the voices. Just as the patient with panic disorder avoids rigorous exercise in fear of producing symptoms of autonomic arousal that mimic panic sensations, or the social phobic wears light clothing to reduce the chances of sweating and drawing attention to himself in a meeting, the patient who hears distressing voices is prone to engage in overt as well as subtle covert behaviors that they believe aid in the management of their voices and reduce attendant distress. Analogous to the socially phobic patient, the reliance on safety strategies by hallucinators tends to maintain the hallucinations. Unfortunately, the effort spent on avoiding or neutralizing the voices leads to a curtailment in the scope of activities, which, in turn, increases isolation and, for many, leads to a paradoxical increase in voice activity.

NEGATIVE SYMPTOMS

Some of the earliest descriptions of the observed emotional, behavioral and social disengagement in schizophrenia (Bleuler 1911, Kraepelin 1913) mirror the current diagnostic description of negative symptoms: restrictions in the range and intensity of emotional expression (affective flattening), in the fluency and productivity of thought and speech (alogia), and in the initiation of goal-directed behavior (avolition) (American Psychiatric Association 2000). Patients experiencing flat affect speak in monotone, stare vacantly, and appear unresponsive. Alogia is reflected in brief and empty replies. Avolition is characterized by reductions in the pursuit of goal-directed activities. Although these symptoms can be readily distinguished, research has demonstrated that they are often assessed as an undifferentiated whole (Alpert et al. 2002).

The cognitive conceptualization of the negative symptoms starts with an explicit stress-diathesis model of schizophrenia (Rector et al. 2004) and recognition of the importance of biological substrates in their production. For instance, Dworkin and colleagues (Dworkin & Lenzenweger 1984, Dworkin et al. 1987) have demonstrated a stronger genetic loading for the development of negative than for positive symptoms of schizophrenia. Further, numerous studies have demonstrated that enlarged lateral ventricles are specifically associated with the negative symptoms in schizophrenia (Andreasen et al. 1982). In other research focused on structural abnormalities, the negative symptoms have been found to be associated with decreased frontal activity (see Liddle 2001 for review) and diminished dopamine activity in the prefrontal cortex (Weinberger et al. 2001). Structural and neurobiological abnormalities may lead to abnormal connectivity between various brain regions, resulting in poor integrative functioning of the brain and limited cognitive resources. Research has demonstrated that cognitive deficits and limited processing resources (Nuechterlein & Dawson 1984) correlate specifically with the presence of negative symptoms and appear to be independent of the presence of positive symptoms.

In addition to the importance of predisposing biological vulnerabilities, the role of preexisting personality and behavioral patterns has been discussed in relation to

the subsequent development of negative symptoms in schizophrenia (Bleuler 1911, Hoch 1910, Kretschmer 1925). For instance, Kretschmer (1925) suggested that trait indifference could be considered as both a clinical manifestation of schizophrenia and as traits of normal temperament that predispose to schizophrenia. This dimensional view of negative symptoms of schizophrenia resting on a continuum of expression of schizoid withdrawal has been retained in the current DSM classification system of the schizoid personality disorder. In support of the dimensional view from premorbid traits to negative symptoms, research demonstrates that up to 85% of schizophrenic outpatients have a history of premorbid schizoid, schizotypal, and avoidant personality disorders (Solano & De Chavez 2000). Importantly, the presence of these disorders correlates specifically with the presence of negative, but not positive, symptoms (Lindstroem et al. 2000, Peralta et al. 1991).

An important component of the schizoid personality pattern centers on negative attitudes toward social engagement. For instance, the tendency to minimize the importance of affiliation is reflected in the endorsement of such attitudes as "Having close friends is not as important as many say," "I attach very little importance to having close friends," and "I prefer hobbies and leisure activities that do not involve other people," and is a characteristic feature of those at risk for developing symptoms of psychosis (Chapman et al. 1994, Miller et al. 2002). These attitudes are also prominent in the biological relatives of individuals diagnosed with schizophrenia (Kendler et al. 1996).

In addition to the role of aversive attitudes toward social relations, patients with negative symptoms appear to demonstrate a negative cognitive set toward goal-directed activity in general. A cross-sectional examination of beliefs and attitudes, as assessed by the Dysfunctional Attitude Scale, was compared with clinical ratings on the Positive and Negative Syndrome Scale in patients with schizophrenia (Rector 2004, Rector & Beck 2003). Endorsement of attitudes such as "If I fail partly, it is as bad as being a complete failure," "If you cannot do something well, there is little point in doing it at all," and "If I fail at work, I'm a failure as a person," were found to be associated specifically with the presence and severity of negative symptoms. In this way, dysfunctional performance attitudes steer the patients to a "point of safety" and further disengagement.

In summary, there is evidence to support the view that negative symptoms reflect an exacerbation of preexisting personality traits. The consolidation of negative attitudes toward affiliation and preference for social distancing prior to the onset of the illness is more likely to give rise to and shape enduring negative symptoms than is the absence of these attitudes.

The Role of Negative Beliefs/Appraisals in Secondary Negative Symptoms

There is empirical support for the assertion that some negative symptoms emerge in response to positive symptoms, defined as secondary negative symptoms. For instance, research by Ventura and colleagues (2004) found that negative symptom

exacerbations (i.e., blunted affect, emotional withdrawal, and/or psychomotor retardation) were concurrent with positive symptom exacerbations over a three-year period. The behavioral responses conceptualized as “secondary” to active hallucinations and delusions often can be understood in terms of the person’s fears, attitudes, beliefs, wishes, and the like, and often serve as a form of protection against the putative threat. For instance, research by Chadwick & Birchwood (1994) found that the idiosyncratic delusional beliefs the person has about the voices’ power and authority determine whether they become engaged or disengaged with the voices (see Beck & Rector 2003 for review). As such, the person’s beliefs and appraisals about the voice activity appear to be more important to the development of secondary negative symptoms than does the mere occurrence of the voice activity.

The Role of Negative Beliefs/Appraisals in Primary Negative Symptoms

There are also cognitive factors that contribute to negative symptoms independent of positive symptoms and that reflect an exacerbation of negative attitudes toward social affiliation. Specifically, an outlook characterized by low expectancies for pleasure, success, acceptance, and the perception of limited resources are pivotal to the production and maintenance of negative symptoms. We posit that the distinct negative expectancy appraisals contribute to the development and particular expression of negative symptoms (Rector et al. 2004). Patients with prominent negative symptoms often think (and sometimes state), “What’s the point? Why should I bother? It’s too much work” when prompted to participate in an activity. They expect to derive little satisfaction for their efforts. These clinical observations have also been borne out in experimental research demonstrating that patients with schizophrenia maintain a negative expectancy set for anticipated pleasure (Germans & Kring 2000). However, research has also demonstrated that when patients with schizophrenia, including those with prominent and severe negative symptoms, are presented with pleasurable stimuli, they can and do derive pleasure from these experiences. As such, there is an observable disconnect between appetitive pleasure (i.e., anticipating that something will bring pleasure) and consummatory pleasure (i.e., the actual level of pleasure experienced from participating in an activity). This is similarly reflected in clinical observations where, despite their initial low expectancy for pleasure, patients demonstrate some enjoyment once engaged in a task.

Patients also demonstrate low expectancies for success in their day-to-day experiences. They expect to fail to meet goals and if they meet the goals, they tend to perceive substandard performance in comparison to their expected performance. This negative outlook affects their motivation to initiate and sustain goal-directed behavior, especially when under stress.

In addition to the real limitations produced by the symptoms of schizophrenia, patients with prominent negative symptoms incorporate stigmatizing views

into their self-construals. These have a negative influence on their perceived self-efficacy when they are faced with life challenges. It is very common for patients to state, "What do you expect, I'm mentally ill," or "It doesn't matter what I do, it's not going to change the fact that I'm just a schizophrenic," or "There's no hope for me since I've got schizophrenia."

Also contributing to a pattern of disengagement are beliefs pertaining to the perceived personal costs of expending energy in making an effort. When presented with the opportunity to participate in a putative pleasant activity, patients will state, "It's too much" or "I can't handle it" or "Why bother?" The subjective accounts of limited resources by patients is likely to reflect, in part, an accurate perception of diminished resources. However, we also propose that patients with prominent negative symptoms exaggerate the limited availability of resources as a result of a fixed defeatist cognitive set. Evidence that they have more available resources than they believe they have is apparent in their increased productivity when these negative attitudes are addressed in treatment (Rector et al. 2003).

The cognitive perspective on the negative symptoms highlights the interaction of neurological deficits, external stressors, personality vulnerability, dysfunctional beliefs, and negative expectancies in their production and maintenance. The symptoms of the negative syndrome, amotivation, anergia, asociality, alogia, and anhedonia may be viewed as an exaggeration of the preschizophrenic traits of avoidance, withdrawal, and negative expectancies.

COGNITIVE THERAPY APPROACH

A meta-analytic review of randomized controlled trial studies testing the efficacy of cognitive behavioral therapy (CBT) for schizophrenia found that interventions led to large clinical and statistical effects on measures of positive and negative symptoms (Rector & Beck 2001). Across controlled trials, the average effect size on measures of positive symptom functioning was 1.31 [standard deviation (SD) = 0.71] in CBT. Clinical improvements in the frequency and distress associated with hallucinations and delusions following CBT were maintained throughout the follow-up period (effect size = 1.48, SD = 0.95). Further, large treatment effects have been observed on composite measures of negative symptoms (effect size = 1.08, SD = 0.83) and throughout follow-up (effect size = 1.19, SD = 0.95). Moreover, the treatment effects in CBT have been found to be superior in comparison with supportive therapy interventions, with a mean weighted between-group effect size of 0.91 (SD = 0.14) in favor of CBT (Rector & Beck 2001). Recently conducted research has shown additional support for the approach. Dickerson (2004) has provided an updated review of 17 clinical trials testing the efficacy of CBT for schizophrenia across different treatment contexts. Recent studies comparing CBT with routine care and/or supportive therapies have found comparatively better outcomes in favor of CBT on selective measures of outcome in day-treatment settings, long-stay inpatient settings, with relapse-vulnerability populations, in older individuals with schizophrenia, and with patients with comorbid

substance abuse or anxiety disorders. Although little research has directly assessed whether improvements in symptoms following CBT convert into improvements in social functioning, a recently completed case-controlled clinical trial demonstrated the ability of CBT to produce large and significant effects on global psychosocial functioning at follow-up (Temple & Ho 2004).

As described elsewhere (Beck & Rector 2000, Rector & Beck 2002), cognitive therapy for schizophrenia is active, structured, time-limited (6–12 months), and can be delivered effectively in individual or group format (see Rector & Beck 2001 for review). The early sessions emphasize the development of the therapeutic relationship through guided discovery. The therapist aims to create a climate of openness and trust through empathic listening and nondirective questioning. The therapist gradually transitions to a more formal assessment of symptoms and the development of a cognitive conceptualization. Mutually prioritized goals are established. An integral component of therapy is the provision of education about the role of personal vulnerability, stressful life circumstances, and their combined roles in the development of the patient's particular symptoms. Treatment targets include delusional interpretations, the beliefs about voices, and harnessing engagement and the reduction of negative symptoms. There is considerable overlap in the strategies employed to treat psychotic symptoms with those for other psychiatric conditions, although there are special modifications to be considered.

The early phase of treatment focuses on building engagement and trust. Many patients expect their symptoms to be judged, dismissed, or ignored, and are often hesitant to speak openly about experiences and beliefs that may be construed as "crazy." Therapists listen carefully, show empathy, and work collaboratively with the patient to understand the meaning they give to these symptoms. Therapists aim to understand the patient's interpretation of important past and present events, and give special attention to those experiences that are identified as key to the development and persistence of their problems. As therapists listen to the meanings and explanations given for different experiences, they begin to normalize these experiences through psychoeducation and imparting of a stress-vulnerability view on the symptoms of schizophrenia. An integral component of therapy is the provision of education about the role of personal vulnerability and stressful life circumstances, and their interaction in the production of delusions, hallucinations, and the negative symptoms. This normalizing strategy conveys to patients the universal nature of their experiences, which serves to reduce stigma (Kingdon & Turkington 1994). Although acute paranoia and severe flattening can pose challenges to the successful development of the therapeutic relationship, alliance ratings from cognitive therapy studies demonstrate the ability of patients to quickly bond with their therapists in the early phase of treatment (Rector et al. 2002).

Further, as in the cognitive therapy for other psychiatric disorders, the therapist socializes the patient to the cognitive model by directing the patient's attention to the relations between their thoughts, feelings, and behaviors. Underlying beliefs and assumptions regarding self (e.g., "I'm unlovable"), others (e.g., "People are dangerous"), and the world (e.g., malevolent) are identified and linked to the

patient's past and present difficulties. This cognitive conceptualization provides an individualized map for navigation in therapy and for the timing of specific cognitive and behavioral strategies. Further, the establishment of an early questioning mode through guided discovery lays the foundation for more direct exploring of cognitive distortions, faulty appraisals, and dysfunctional beliefs associated with the positive and negative symptoms.

The therapeutic approach to treating delusions involves a number of cognitive and behavioral strategies aimed at undermining the rigid conviction, centrality, and distress of the delusion(s). In the assessment phase, the patients' pre-delusional beliefs are ascertained by inquiring about their preoccupations and daydreams. The therapist also attempts to identify proximal events critical to the formation of the delusions ("How were things going in your life when you started having this idea?") as well as current events that are likely to trigger the delusions. Specific triggers for delusions can be both external (e.g., a plane overhead) and internal (e.g., butterflies in the stomach). The specific emotional (e.g., fear, guilt, anger) and behavioral (e.g., avoidance, apathy, confrontation) consequences created by the activation of the delusion are also assessed. Once the therapist has a thorough understanding of the patient's delusional beliefs and the past and current events that are interpreted as supporting the belief, exploration of the evidence is undertaken. The cognitive approach is collaborative and Socratic, and the therapist initially deals with the interpretations that are peripheral to the more central and highly charged beliefs. Therapists' questioning mode provides the context for patients to generate a range of alternative explanations for these experiences. With repeated practice in generating alternative explanations in the therapists' office and then routinely as homework between sessions, the patient can begin to respond to life events more flexibly and thus reduce the delusional beliefs. In addition to verbal strategies, the cognitive therapist aims to institute change in delusional thinking by setting up behavioral experiments to test the accuracy of different interpretations.

In targeting auditory hallucinations, cognitive therapy is specifically aimed at helping patients with the distress that it is created by voices. Prior to implementing cognitive and behavioral strategies to help the patient construct an alternative view of their voices, a thorough assessment is undertaken with careful questioning of the frequency, duration, intensity, and variability of the voices. Patients report hearing voices more frequently when there are interpersonal difficulties, daily hassles, and negative life events (e.g., financial strain, housing crises, etc.) or alternatively, when they are isolated and lonely. Patients record the specific voice content between sessions with modified thought records to capture voice activity and reactions to the voices. Following from Chadwick & Birchwood's (1994) research showing that it is the idiosyncratic beliefs the patient holds toward the voices more than the actual occurrence of the voice activity itself that creates distress, the therapist aims to elicit all of the beliefs the patient has about his/her voices. What agents (God, the Devil, dead relatives, etc.) are purportedly talking to the patient? Beliefs about the voices can range from bizarre delusions to the

ordinary. All beliefs are identified and the evidence that has been interpreted as supporting these beliefs is recorded. As in the assessment of delusions, the therapist identifies the distal and proximal life circumstances to the initial onset of the voices. Next, the therapist enters a questioning mode to elicit alternative perspectives on both the voice content and the beliefs held about the voices. The approach to undermining the beliefs about voices is similar to the cognitive approach in treating delusions. The therapist explores the evidence that the patient has taken as support for his interpretations. Behavioral experiments are used to test out alternative explanations. For instance, the perceived omnipotence and omniscience of the voices can be tackled by setting up experiments that will demonstrate that the patients can ignore commands without consequence.

In addition to working with the evidence, patients are asked whether they have ever considered other explanations for their voices. Through collaboration, the therapist and patient attempt to generate as many alternative explanations for the voices as possible, with the ultimate aim of helping patients to recognize that the voices represent their own attitude about themselves and/or the attitudes they imagine others have about them.

The cognitive approach to treating negative symptoms includes behavioral self-monitoring, activity scheduling, mastery and pleasure ratings, graded task assignments, and assertiveness training methods. Cognitive strategies include eliciting the patient's reasons for inactivity and testing these beliefs directly with behavioral experiments; direct attempts to stimulate interests—either new interests or a reactivation of previously held interests; and identifying, testing, and changing rigid and defeatist negative expectancies for pleasure, successful performance, acceptance by others, and limited availability of resources. The negative symptoms are successfully reduced as a secondary response to reductions in positive symptoms (Sensky et al. 2000) or directly and independently from changes in positive symptoms (Rector et al. 2003).

THEORETICAL CONSIDERATIONS AND CONCLUSIONS

The recent studies of adjunctive cognitive behavioral therapy in the treatment of schizophrenia indicate that it is possible to alleviate the major symptoms by activating a number of the patients' "higher cognitive functions" such as distancing themselves from their dysfunctional interpretations, evaluating the evidence, and exploring alternative explanations, all of which are part of reality testing. The same kinds of introspective techniques are also used in identifying the underlying core dysfunctional beliefs.

Several theoretical points flow from these observations. Some portion of the causal basis of schizophrenia symptoms may represent a direct consequence of neural system dysfunctions, and the interaction of limited capacity with stress may help to explain some of the remaining variance in symptom expression, and perhaps in particular, temporal variations and their severity. These clinical findings

suggest a broader conception of the cognitive dysfunction in schizophrenia, viewing it not simply as a disorder stemming from domain-specific deficits, but as an impairment of the total integrative function of the brain. This impairment reduces the resources for dealing with stress and increases the vulnerability to aversive life experiences, leading to dysfunctional beliefs and behaviors. The resulting psychological disturbances further drain the limited cognitive resources and evoke more aversive experiences and dysfunctional beliefs and behaviors. This combination of cognitive insufficiency and the resultant stressful conditions provide the basis for the progression from the premorbid state to the cognitive decompensation manifest in the symptomatology of schizophrenia: the activation of dysfunctional schema and reduced reality testing as manifested in delusions and hallucinations, the resource sparing in the negative symptoms, and the collapse of organized structure in formal thought disorder. The loss of context or set described in thought-disordered patients may be attributed, in part, to the relative paucity of resources available for maintaining a set, adhering to rules of coherent communication, and inhibiting intrusion of inappropriate ideas. Resource sparing is evident in poverty of speech and the "easiness" bias, and resource stretching is evident in disinhibition. Pharmacological and psychotherapeutic treatments can damp down the hyperactive cognitive schemas and thus free up resources for further reality testing (cognitive compensation).

Finally, schizophrenia may be viewed as an outcome of the cycling interaction of neurocognitive deficits, stressful environmental events, and resultant dysfunctional beliefs and interpretations. Although cognitive behavioral therapy may not affect the basic neurocognitive lesions, it can modify the dysfunctional beliefs that can contribute to the physiological stress and exacerbations of the neurocognitive deficits.

The observation that the patient can be trained to modify the faulty beliefs that contribute to and aggravate delusions, hallucinations, and negative symptoms suggests that they have available cognitive resources that are made accessible through therapy.

We believe that many of the theoretical propositions are readily testable. Some headway has already been made in identifying certain core beliefs associated with the persecutory delusions, hallucinations, and negative symptoms. A study, for example, could examine the continuity of core beliefs during the prodromal phase and after the transition in psychosis. Another study could investigate the content of patients' automatic thoughts and hallucinations. Finally, brain-imaging studies could compare the changes achieved through a combination of CBT and pharmacotherapy with pharmacotherapy alone.

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