

Negative Symptoms and Formal Thought Disorder: Cognitive Characterization and Therapy

Dysfunctional negative beliefs and attitudes may play a role in negative symptoms and formal thought disorder. Cognitive therapy can be used to uncover such beliefs and to determine alternative viewpoints.

TEKST

Neal Stolar

Paul Grant

PUBLISERT 4. mai 2007

ABSTRACT:

Cognitive therapy is an effective treatment for schizophrenia; however, most of the empirical and theoretical attention in this field has heretofore been bestowed upon the symptoms of hallucinations and delusions. Here we consider the extension of cognitive therapy approaches to two of this disorder's prominent manifestations – the negative symptoms and formal thought disorder. Specifically, we argue that dysfunctional beliefs and attitudes play a pivotal role in the day-to-day exacerbations of negative symptoms and formal thought disorder. Cognitive therapy techniques can be used to uncover these beliefs, scrutinize their plausibility, and determine alternative viewpoints through examination of the evidence and the testing of new behaviors. We present here the theoretical basis, initial empirical findings, and suggestions for clinical applications of cognitive therapy for the treatment of negative symptoms and thought disorder.

Keywords: psychosis, cognitive therapy, negative symptoms, formal thought disorder

“Cognitive-free” Symptoms of Schizophrenia

Factor analytic studies of the symptoms of schizophrenia conducted in several cultures across the world (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; Andreasen et al., 2005; Barnes & Liddle, 1990; Fuller, Schultz, & Andreasen, 2003; John, Khanna, Thennarasu, & Reddy, 2003) all converge on, at minimum, a three factor solution: 1. psychotic symptoms (hallucinations and delusions), 2. disorganized symptoms (bizarre behavior and positive formal thought disorder), and 3. negative symptoms (flat affect, alogia, avolition, and anhedonia). Such studies are a part of an emerging database that corrects the overly narrow definition of schizophrenia that has enjoyed prominence in psychiatry over the past 40 years – that of being predominantly a psychotic disorder (Carpenter, 2006). While the three-dimensional solution returns the field to the richer and more accurate accounts of its pioneers (Bleuler, 1950; Kraepelin, 1971), the

cornerstone of schizophrenia treatment continues to be a pharmacotherapy that is largely an antipsychotic enterprise (Fenton, 2005). That the negative and disorganized dimensions are not the focus of treatment may go a long way toward explaining why these dimensions are associated with poor outcome in schizophrenia (Kirkpatrick, Fenton, Carpenter, & Marder, 2006; Norman et al., 1999).

Over the past 15 years, cognitive therapy has emerged as an innovative and effective treatment for individuals diagnosed with schizophrenia and schizoaffective disorder (Gould, Mueser, Bolton, Mays, & Goff, 2001; Pilling et al., 2002; Rector & Beck, 2001); however, this therapeutic effort has been, like pharmacotherapy, primarily focused upon the psychotic symptoms (Chadwick, Birchwood, & Trower, 1996; Fowler, Garety, & Kuipers, 1995; Nelson, 2005; Perris, 1989). One explanation for this emphasis is that the symptoms of psychosis are cognitive in nature: hallucinations often occur in the form of “voices” with reportable verbal content, potentially reflecting automatic or “hot” cognition (Beck & Rector, 2003); delusions, by definition, are reported beliefs (APA, 2000). The other two clusters of symptoms, by contrast, are typically devoid of meaningful verbal content: patients with negative symptoms are characterized by a reduction in verbal and non-verbal communication (Kirkpatrick et al., 2006), while the disturbance of formal thought disorder often renders verbal content difficult, if not impossible, to comprehend (Andreasen, 1979). Another factor is the widely-held perception that patients who are significantly disorganized or emotionally non-responsive or unengaged in their lives might be difficult, if not impossible, to engage in the efficacious collaborative therapeutic process entailed in cognitive therapy (Kingdon & Kirschen, 2006). Indeed, we note that researchers in the U.K. have recently advocated the adoption of the term CBTp (Terrier, 2005), which serves, among other things, to emphasize the idea that cognitive therapy predominantly addresses the psychotic dimension within schizophrenia.

Although negative symptoms and thought disorder have limited cognitive content of a meaningful nature, we propose that specific active psychological processes contribute to what on the surface might seem to be more behavioral, linguistic and emotional deficiencies. Our formulation is similar to, and owes much to, the framework employed by Beck (1963) in the early 1960s, in which he showed that cognitive aspects of depression, far from being epiphenomena, were fundamental participants that precede emotional and behavioral aspects. We hypothesize that those with negative symptoms or thought disorder have low expectations of success and/or pleasure in the activities of daily living such as education, employment, and social interaction. We further hypothesize that these attitudes are key cognitive participants in both dimensions of symptoms: when success or pleasure is not anticipated from an activity, desire to engage that activity is, often completely, reduced (negative symptoms); when the activity is engaged, more stress results, and, in the case of acts of communication, disorganization can result. While keeping this integrative hypothesis in mind, we will initially present the cognitive conceptualization of these classes of symptoms

separately, and then discuss a cognitive therapy approach that is tailored to negative symptoms and formal thought disorder.

Cognitive Conceptualization of Negative Symptoms

The negative symptoms include reduced verbal (alogia) and non-verbal expressivity (affective flattening), as well as limited engagement in constructive (avolition), pleasurable (anhedonia), and social (asociality) activity (Kirkpatrick et al., 2006). The consistency with which these symptoms cluster in factor analyses has been taken as evidence of the validity of the negative symptom construct in schizophrenia (Earnst & Kring, 1997). Further validation comes from the temporal profile: negative symptoms, relative to psychotic or disorganized symptoms, tend to appear earlier (Davidson et al., 1999; Lencz, Smith, Auther, Correll, & Cornblatt, 2004) and tend to be more stable across the chronic course of schizophrenia (Hafner, 2003). Furthermore, negative symptoms are associated with poorer outcomes, compared to the other symptom dimensions, over 5- and 10-year follow-up periods (Bromet, Naz, Fochtmann, Carlson, & Tanenberg-Karant, 2005; Milev, Ho, Arndt, & Andreasen, 2005). Antipsychotic medications have demonstrated minimal efficacy with regard to negative symptoms (Erhart, Marder, & Carpenter, 2006), making treatment innovation in this domain a priority of schizophrenia treatment research (Kirkpatrick et al., 2006).

Explanatory models of negative symptoms have, since the seminal writings of Hughlings Jackson (1931), appealed to degenerative neurobiology. One proposal is that loss of brain tissue causes the loss of capacity characteristic of negative symptomatology, as indicated by studies that find enlarged cerebral ventricles associated with prominent negative symptoms in schizophrenia (Crow, 1980). Another theoretical approach, using frontal lobe patients as analogy, proposes that pathology in the frontal lobes produces reduced activation levels which, in turn, causes loss of motivation, reduced emotionality, and minimal willful behavior (Miller & Tandon, 2001). Yet a third approach attributes the cause of negative symptoms to the cognitive impairment – deficits of memory, attention and executive function – characteristic of the vast majority of individuals diagnosed with schizophrenia (Heinrichs, 2005). While the evidence supporting these neurobiological models is mixed (Stolar, 2004), the formulations all characterize negative symptoms as biological deficits: the prominent negative symptom patient is represented as limited by his neurobiology such that he cannot engage constructive activity, generate expressive responses, etc. The recently published NIMH-MATRICES consensus statement on negative symptoms (Kirkpatrick et al., 2006) continues this trend, as conceptual, measurement, and research design issues are clarified so that the basic neuropathology of negative symptoms might be more effectively characterized and, importantly, made amenable to treatment by bioactive agents or devices.

We share Terrier's (2006) concern that the consensus achieved by the NIMH-MATRICES group is too narrowly focused upon the biological aspects of negative symptoms and places far too much emphasis on pharmacotherapy. Moreover, there is an emerging

literature that is beginning to chart the psychological aspects of negative symptoms in schizophrenia. Anhedonic patients diagnosed with schizophrenia, for example, have been shown to experience pleasure to an equivalent degree to normal control subjects (Gard et al., 2003), a result that contradicts the notion that these patients have a neurobiological deficit that hinders their ability to experience pleasures. Rather, a cognitive factor, expectation, distinguishes the two groups: the patients erroneously expect not to enjoy themselves and, thereby, engage in pleasurable activities to lesser extent than the controls. In a similar vein, Rector (2004) has found that elevated negative symptoms are associated with attitude endorsements such as “If I don’t do something well, there is no point in doing it at all.” Significantly, the correlation between the attitudes and negative symptoms holds when severity of depression is controlled. This set of beliefs endorsed by patients with negative symptoms has subsequently been labeled *defeatist performance attitudes* (Grant, 2005), because the attitudes are over-generalized, inaccurate and feed into a vicious cycle of avoidance, apathy, passivity, and isolation which may protect the patient from the pain of rejection, but lead to unhappiness and an empty life (Selton, Wiersma, & van den Bosch, 2000).

Integrating findings on the psychology of negative symptoms, Rector, Beck and Stolar (2005) have hypothesized that dysfunctional, negativistic beliefs contribute to the avoidance of constructive activity seen in individuals with schizophrenia. The relevant factors contributing to loss of motivation and avoidance are low expectancies for pleasure (e.g., “I won’t enjoy it.”), low expectancies for success at social and non-social tasks (e.g., “I am not going to be good enough.”), low expectancies for social acceptance (e.g., “What do you expect? I am mentally ill.”), and defeatist beliefs regarding performance (e.g., “If I am not sure I will succeed at a task, there’s no point in trying.”). The negativistic and overly general beliefs stymie the initiation of action (including speech and emotional expression). These cognitive causes (including demoralization and fear of rejection) may not be obvious, depending on the insight of the person afflicted, and thus, negative symptoms deemed as primary might arise from these negative attitudes, as well.

Ultimately, we propose a diathesis-stress framework in which defeatist beliefs are mediators in the causal chains that link cognitive impairment, negative symptoms and poor functioning in schizophrenia. Abnormal pruning, ventricular enlarging and other physiological disruptions may produce alterations in neuro-connectivity causing poor integrative functioning of the brain (McGlashan & Hoffman, 2000) and, hence, limited processing resources and poor neurocognitive performance (Bunney & Bunney, 1999). Cognitive deficits combined with limited availability of processing resources (Nuechterlein & Dawson, 1984) will likely provoke those with vulnerability for negative symptoms, producing adverse developmental stressors, such as social and academic failures (Lencz et al., 2004). There can even be a vicious cycle in which physiologically-caused negative symptoms lead to negative attitudes that perpetuate and exacerbate the symptoms of avolition and apathy.

Cognitive Conceptualization of Formal Thought Disorder

Formal thought disorder, along with inappropriate affect and bizarre behavior, comprise the disorganization dimension in schizophrenia (Andreasen et al., 1995; Fuller et al., 2003; Liddle, 1987). It is associated with poor educational, occupational and social functioning (Harrow, Marengo, & McDonald, 1986; Norman et al., 1999). Thought disorder is considered a subset of the language disorder found in individuals with schizophrenia (Covington et al., 2005). McKenna and Oh (2005) have recently summarized four theoretical approaches: thought disorder as 1. dysphasia, 2. communicative incompetence, 3. a dysexecutive phenomenon, and 4. a dyssemantic phenomenon.

As defined by Andreasen (1979, 1984b), positive forms of thought disorder include loosening of associations (various forms of getting off the track of the flow of a conversation, as well as tangential replies to questions) and idiosyncratic use of language (Andreasen & Grove, 1986; Peralta, Cuesta, & de Leon, 1992), such as neologisms (creating new words) and word approximations (using existing words in a new way). At its most extreme, positive formal thought disorder manifests as incoherence or word salad (random use of words). Negative thought disorder symptoms (Andreasen, 1984a) include blocking (interruption in the flow of thought) and poverty of content of speech, which itself includes concreteness, perseveration, clanging and echolalia (Marengo, Harrow, & Edell, 1993).

Formal thought disorder symptoms have been shown to worsen when the topic of conversation is emotionally salient (Docherty, Cohen, Nienow, Dinzeo, & Dangelmaier, 2003) or when the person is criticized by family members (Rosenfarb, Goldstein, Mintz, & Nuechterlein, 1995). This research accords with our clinical experience that the client with thought disorder is often quite lucid when discussing neutral topics (e.g., “How did you get here?”) or appetitive topics (e.g., sports), but becomes very thought disordered when discussing topics related to their treatment. Accordingly, we propose that situational and psychological aspects play a key role in the day-to-day experience of thought disorder. Specifically, formal thought disorder is, in part, a stress response to “hot” topics and situations. In this regard, thought disorder is analogous to stuttering – particular thoughts (e.g., “They won’t understand me?” “I am stupid”) elicited by certain situations lead to an increase in communicative difficulty. A vicious cycle, again, can be triggered by social feedback, as the people in the patient’s environment struggle to comprehend his/her speech and become increasingly frustrated and impatient, while, at the same time, the patient’s level of tension goes up and his/her experiences increased intrusive ideation regarding failure – the end result being even more disorganized speech. Repeated experiences of this sort could well lead to a preference, on the part of the patient, to avoid social interactions, which might partially explain the association between thought disorder and poor social functioning.

As in depression and anxiety disorders, specific types of automatic thoughts and distorted beliefs will lead to the occurrence of the thought disorder symptoms. In particular, defeatist attitudes regarding social performance and speech performance

seem to be likely candidates. Preliminary research conducted in our lab supports this idea: patients with elevated thought disorder also tend to endorse social aversion attitudes. This association holds even when psychotic, negative and depressive symptoms are statistically controlled. While this sort of research is in its infancy, rendering any conclusions as speculative, we believe that the understanding of thought disorder will be greatly advanced by studies exploring psychological aspects such as beliefs and expectations.

Cognitive Therapy for the Treatment of Negative Symptoms and Thought Disorder

While there is a tradition of psychosocial treatment of negative symptoms that has focused upon behavioral methods, such as skills training (Kopelowicz, Liberman, & Zarate, 2006), token economies (Kazdin, 1982), and psychiatric rehabilitation (Kopelowicz, Liberman, & Wallace, 2003), we are unaware of any such efforts targeting formal thought disorder. Cognitive therapy, despite its historical emphasis upon psychotic symptoms in schizophrenia, has, nonetheless, begun to establish a promising track record with regard to negative symptoms (Rector & Beck, 2001). Sensky and colleagues (2000), for example, in a study targeting treatment-resistant positive symptoms in outpatients, found that cognitive therapy produced a significant reduction in negative symptoms across a 9-month follow-up period as compared to patients in an informal support control group. This reduction in negative symptoms has proved impressively durable, as the cognitive therapy patients continued to show less negative symptoms five years after treatment subsided. Similarly, in a trial that is notable for explicitly targeting negative symptoms, Rector and colleagues (2003) found cognitive therapy, compared to enriched treatment as usual, to have reduced negative symptomatology over the 9-month follow-up period.

While we are encouraged by these results, it is our belief that more assertive tactics will, ultimately, add considerable efficacy to the treatment of both negative symptoms and thought disorder. Goal-directed cognitive therapy has been developed by Aaron T. Beck at the University of Pennsylvania with difficult to treat cases in mind. The general framework of this approach is to establish plausible long-term goals with the patient, and then to establish a series of short-term goals. As therapy proceeds, the therapist works collaboratively with the patient to move through the goals step-by-step. Thought disorder, low motivation and other symptoms such as hallucinations and delusions are addressed as they become obstacles to the patient reaching his goals.

Negative Symptoms

A protracted period of engagement with specific, direct questioning may be needed to access the goals of someone with negative symptoms. There often is a need to involve family and/or mental health staff to determine a patient's current needs as well as improvement as therapy progresses. Some modifications may be necessary when engaging in therapy with someone with negative symptoms. Patience is required in that responses may be slow and devoid of significant detail. Open-ended questions may need to be followed by direct or even forced-choice questions. The cognitive

impairment that often accompanies negative symptoms may necessitate the use of slow, repetitive speech on the part of the therapist. The patient may need to be asked to repeat key items to check for understanding. Agenda-setting may be limited by the patient's apathy. A therapist can follow a step-wise approach of allowing a more-than-usual amount of time to respond, asking about recent events and concerns about them, addressing common long-term goals (relationships, work, hobbies), and providing a choice of possible agenda items. As therapy progresses, a patient may be better able to formulate goals and agendas.

Given that biobehavioral models of negative symptoms postulate diminished activity in the systems responsible for motivated behavior (Stolar, 2004), external stimulation may energize the patient. In this regard, the therapist is a primary catalyst to help the patient identify goals and begin to ratchet up motivation and the successful execution of goal-directed behavior. The primary therapeutic tools of this activating process are activity scheduling with mastery and pleasure ratings and graded task assignments (Kingdon & Turkington, 2005). The more concrete the plan and the more immediate the outcome, the better. Accordingly, rewards, including frequent reminders of any incremental progress, should be immediate, and steps to achieve certain activities need to be specified and even written down as cues.

Cognitively, eliciting and addressing the negative attitudes about one's abilities can help reverse the cycle of resignation and disappointment. Rather than the presence of negative thoughts, there may be a lack of positive cognition in the sense that activities (including speech and facial expressions) may not be viewed as having any value rather than being viewed as having some negative consequence. Helping a client think of positive consequences of certain actions may help produce motivating thoughts. Cost-benefit analyses can aid in examining the advantages and disadvantages of doing some activity or doing nothing. Chronic patients' underlying assumptions and beliefs may be long-standing and not necessarily associated with recent situations. These assumptions may be reflected behaviorally in overcautiousness, passivity, and distancing the self from risks. These beliefs can be addressed as they interfere with the patient's new set of goals that involve more engagement with life and a better quality of life. Similarly, hallucinations and delusions that cause, via secondary psychological process, negative symptoms can become obstacles to the patient's ongoing goals, and can, as such, be addressed with appropriate cognitive and behavioral techniques (Chadwick et al., 1996; Kingdon & Turkington, 2005).

Formal Thought Disorder

Treatment begins, as in all types of therapy, with establishing rapport. If formal thought disorder is due to negative automatic thoughts related to social performance, including conversation, it may be reduced merely by establishment of the therapeutic alliance, as trust and confidence builds. One of the authors sees someone with schizophrenia who presented in the first session with severe thought disorder. However, it was virtually absent by the second session. The person admitted that she had been apprehensive

about changing therapists. She may have been most anxious in the initial session but less so in subsequent ones, leading to a dramatic reduction in formal thought disorder.

Cognitive assessment of formal thought disorder can be initiated as soon as its presence is detected. One component of this assessment concerns the content of disordered speech, as this content may have pertinent psychological meaning. Therefore, efforts should be made to discern as much as possible the ideas the patient is attempting to communicate keeping a particular eye to the emergence of themes. Being able to pose relevant questions to the patient serves a double purpose: the problem-solving activity of goal-directed therapy can be advanced at the same time that the patient appreciates that he is being understood. The latter aspect can then help reduce stress and potentially reduce the formal thought disorder itself.

Intermittent formal thought disorder, or fluctuations in its severity in session, calls for employment of a second cognitive assessment component – guided discovery for antecedent automatic thoughts that lead to the stress response manifested as disorganized speech. The therapist may be able to note what topics or situations (such as the presence of family members in certain sessions) lead to worsening of formal thought disorder in session. This can generate appropriate questions leading to the discovery of automatic thoughts preceding the exacerbation of formal thought disorder.

Given that people with schizophrenia exhibit more thought-disordered speech when discussing personal, emotion-laden items (Docherty, Evans, Sledge, Seibyl, & Krystal, 1994; Docherty, Hall, & Gordinier, 1998; Haddock, Wolfenden, Lowens, Tarrier, & Bentall, 1995), a useful strategy for reducing thought disorder symptoms would be to utilize therapeutic methods aimed at emotion regulation (Morrison, 2004) and stress reduction. Standard cognitive therapy techniques for managing depressive, anxiety and anger difficulties, as well as for ameliorating the emotional effects of hallucinations and delusions, can indirectly help to improve organization of speech.

Kingdon and Turkington (1994) suggest, additionally, using role plays to help the patients understand how their communication might not be comprehended by others and how to use clearer language by taking the position of the listener. This method has support from studies showing that patients are able to explain previously expressed thought-disordered discourse (Harrow & Prosen, 1978), provide meanings of neologisms (Foudraine, 1974), and improve communication after listening to audiotapes of prior conversation (Satel & Sledge, 1989).

Ultimately, therapist and patient work to improve communication, in part to facilitate the goals of therapy itself. Nelson (2005) recommends questioning patients directly when units of speech are not understood. In addition, Pinninti, Stolar, and Temple (2005) recommend 1) the five-sentence rule in which therapist and patient limit speech to five sentences at a time so that disorganization has less chance of worsening with length of conversation, 2) taking two-minute relaxation breaks using deep breathing, or

switching to a neutral topic when emotionally-laden material elicits thought disorder symptoms, and 3) asking about communication difficulties with others.

It is best to use reflective listening for those passages that are clearly understood so that the patient gets positive feedback for precise communication (and correction by the patient can be made if those passages are actually not understood correctly). Focus can then be directed at the incoherent items. More general questions (“What do you mean by ...?”) can be followed, if necessary, by suggested meanings based on the context and tone of the item in question. Clearly divergent, irrelevant material (such as clanging) may be ignored for the most part, but care should be taken to not throw out the baby with the bath water by mistaking emotionally relevant material for impertinent minutiae. For instance, the first word in a series of rhyming words may be important (e.g., “I’m depressed, oppressed, confessed, undressed.”)

Since the use of cognitive therapy for the treatment of schizophrenia has not yet focused on formal thought disorder, much work remains to be done to test the usefulness of these focused approaches. Improving the flow of speech can enable many people with schizophrenia and thought disorder to then engage in the approaches for hallucinations, delusions, and negative symptoms that previously would have been hampered by the thought disorder itself.

By determining what negative thoughts a person may be thinking prior to the moment of disorganized speech, strategies for cognitive therapy may be developed for that person and perhaps applied to other patients as well. This latter application is important since it may be very difficult to access the automatic thoughts of more severe cases of formal thought disorder. Automatic thoughts revealed in less severe cases may have to be applied in the treatment of more severe cases until formal thought disorder is diminished in these latter cases to the point that formal cognitive therapy can be initiated and idiosyncratic automatic thoughts accessed.

In addition to determining initial cognitive precipitants of formal thought disorder, it would also be useful to explore cognitive reactions to the social effects of formal thought disorder (which may lead to worsening or at least perpetuation of formal thought disorder). The possibility that disorganized thought, elicited by anxiety, may lead to a freezing of planning and behavior indicates that stress reduction methods, including relaxation techniques as well as cognitive treatment of depression, anxiety, anger and positive symptoms, may help reduce the negative symptoms. The basic cognitive technique of slowing one’s thoughts and examining them may help more directly in counteracting disorganized thinking to allow for meaningful behavioral output.

Summary

Although, on the surface, negative symptoms and formal thought disorder appear to be devoid of meaningful cognitive content and therefore inaccessible by cognitive therapeutic methods, we hypothesize that these symptoms emerge in part as a result of dysfunctional negative attitudes related to low expectations of success and/or pleasure.

These beliefs result in either limited activity (including minimal speech and affect) in the case of the negative symptoms, or in disorganization (including disorganized speech) that is secondary to the stress of attempting to engage in activities despite these negative expectations. Data supporting this view have been obtained in the case of negative symptoms, and preliminary findings suggest that this hypothesis applies to thought disorder as well. The standard cognitive therapy approach of eliciting negative automatic thoughts and examining the evidence can be applied to these symptoms, modifications being introduced when these symptoms directly impede the communicative aspects of therapy. Since patients can also have low expectations for success in the therapy sessions, engagement processes can be critical to achieving therapeutic gains. With further refinement of the use of cognitive therapy for negative symptoms and formal thought disorder, many more patients previously considered out of the reach of psychotherapy can start on the road to a more enriched life.

Neal Stolar M.D.-Ph.D.

Hospital of the University of Pennsylvania

10 Gates 3400 Spruce Street Philadelphia, PA 19101. Email: nstolar@juno.com

NEAL STOLAR

Neal Stolar is clinical associate professor, Department of Psychiatry, University of Pennsylvania; Medical Director at Project Transition – Audubon Unit, Audubon, PA; consulting psychiatrist at Creative Health Services, Pottstown, PA; and consulting psychiatrist at Beck Institute for Cognitive Therapy and Research, Bala Cynwyd, PA.

He has a private practice, and also works as a research scientist at Brain Behavior Laboratory, University of Pennsylvania. His research interests are cognitive therapy for schizophrenia and the neurobiology of schizophrenia.

Key publications

Pinninti, N. R., Stolar, N., & Temple, S. (2005). Five-minute first aid for psychosis. *Current Psychiatry*, 4, 36-48.

Rector, N. A., Beck, A. T., & Stolar, N. M. (2005). The negative symptoms of schizophrenia: a cognitive perspective. *Canadian Journal of Psychiatry*, 50, 247-257.

Stolar, N. (2004). Cognitive conceptualization of the negative symptoms of schizophrenia. *Journal of Cognitive Psychotherapy: An International Quarterly*, 18, 237-253.

PAUL GRANT

Dr. Paul Grant is the director of schizophrenia research and a fellow in the Psychopathology Research Unit, Department of Psychiatry, University of Pennsylvania. Having learned cognitive therapy from Robert DeRubeis and Aaron T. Beck, his research interests included cognitive psychopathological models of positive and negative symptoms, as well as cognitive therapy of schizophrenia.

Key publications

Grant, P., & Beck, A. T. (2007). Defeatist beliefs mediate cognitive impairment and negative symptoms in schizophrenia. Under review American Journal of Psychiatry.

Grant, P., Young, P. R., & DeRubeis, R. J. (2005). Cognitive and behavioral therapies. In G. O. Gabbard, J. S. Beck & J. Holmes (Eds.), Oxford textbook of psychotherapy (pp. 15-25). New York: Oxford University Press.

Warman, D., Grant, P., Sullivan, K., Caroff, S., & Beck, A. T. (2005). Individual and group cognitive behavioral therapy for schizophrenia: a pilot investigation. Journal of Psychiatric Practice, 11, 27-34.

Teksten sto på trykk første gang i Tidsskrift for Norsk psykologforening, Vol 44, nummer 5, 2007, side 555-561

TEKST**Neal Stolar****Paul Grant**[!\[\]\(5361750c22c4e047a52f4eac1ec2d4cc_img.jpg\) Vis referanser](#)**References**

Andreasen, N. C. (1979). Thought, language, and communication disorders: II. diagnostic significance. Archives of General Psychiatry, 36, 1325-1330.

Andreasen, N. C. (1984a). The Scale for the Assessment of Negative Symptoms (SANS). Iowa City: University of Iowa.

Andreasen, N. C. (1984b). The Scale for the Assessment of Positive Symptoms (SAPS). Iowa City: University of Iowa.

Andreasen, N. C., Arndt, S., Alliger, R., Miller, D., & Flaum, M. (1995). Symptoms of schizophrenia: methods, meanings and mechanisms. Archives of General Psychiatry, 52, 341-351.

Andreasen, N. C., Carpenter, W. T., Kane, J. M., Lasser, R. A., Marder, S. R., & Weinberger, D. R. (2005). Remission in schizophrenia: proposed criteria and rationale for consensus. American Journal of Psychiatry, 162, 441-449.

Andreasen, N. C., & Grove, W. M. (1986). Thought, language, and communication in schizophrenia: diagnosis and prognosis. Schizophrenia Bulletin, 12, 348-359.

APA. (2000). Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.). Washington, DC: American Psychiatric Association.

Barnes, T. R. E., & Liddle, P. F. (1990). Evidence for the validity of negative symptoms. In N. C. Andreasen (Ed.), Schizophrenia: positive and negative symptoms and syndromes (Vol. 24, pp. 43-72). Basel, Switzerland: Karger.

Beck, A. T. (1963). Thinking and depression: idiosyncratic content and cognitive distortions. Archives of General Psychiatry, 9, 324-333.

Beck, A. T., & Rector, N. A. (2003). A cognitive model of hallucinations. Cognitive Therapy and Research, 27, 19-52.

- Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias* (J. Kinkin, Trans.). New York: International Universities Press.
- Bromet, E. J., Naz, B., Fochtmann, L. J., Carlson, G. A., & Tanenberg-Karant, M. (2005). Long-term diagnostic stability and outcome in recent first-episode cohort studies of schizophrenia. *Schizophrenia Bulletin*, 31, 639-649.
- Bunney, W. E., & Bunney, B. G. (1999). Neurodevelopmental hypothesis of schizophrenia. In D. S. Charney, E. Nestler & B. S. Bunney (Eds.), *Neurobiology of mental illness* (pp. 222-235). New York: Oxford University Press.
- Carpenter, W. T., Jr. (2006). The schizophrenia paradigm: a hundred-year challenge. *Journal of Nervous and Mental Disease*, 194, 639-643.
- Chadwick, P., Birchwood, M., & Trower, P. (1996). *Cognitive therapy for delusions, voices, and paranoia*. Chichester: John Wiley and Sons.
- Covington, M. A., He, C., Brown, C., Naci, L., McClain, J. T., Fjordbak, B. S., et al. (2005). Schizophrenia and the structure of language: the linguist's view. *Schizophrenia Research*, 77, 85-98.
- Crow, T. J. (1980). Molecular pathology of schizophrenia: more than one disease process. *British Medical Journal*, 280, 66-68.
- Davidson, M., Reichenberg, A., Rabinowitz, J., Weiser, M., Kaplan, Z., & Mark, M. (1999). Behavioral and intellectual markers for schizophrenia in apparently healthy male adolescents. *American Journal of Psychiatry*, 156, 1328-1335.
- Docherty, N. M., Cohen, A. S., Nienow, T. M., Dinzeo, T. J., & Dangelmaier, R. E. (2003). Stability of formal thought disorder and referential communication disturbances in schizophrenia. *Journal of Abnormal Psychology*, 112, 469-475.
- Docherty, N. M., Evans, I. M., Sledge, W. H., Seibyl, J. P., & Krystal, J. H. (1994). Affective reactivity of language in schizophrenia. *Journal of Nervous and Mental Disease*, 182, 98-102.
- Docherty, N. M., Hall, M. J., & Gordinier, S. W. (1998). Affective reactivity of speech in schizophrenia patients and their nonschizophrenic relatives. *Journal of Abnormal Psychology*, 107, 461-467.
- Earnst, K. S., & Kring, A. M. (1997). Construct validity of negative symptoms: an empirical and conceptual review. *Clinical Psychology Review*, 17, 167-190.
- Erhart, S. M., Marder, S. R., & Carpenter, W. T. (2006). Treatment of schizophrenia negative symptoms: future prospects. *Schizophrenia Bulletin*, 32, 234-237.
- Fenton, W. S. (2005). Schizophrenia: integrative treatment and functional outcomes. In B. J. Sadock & V. A. Sadock (Eds.), *Kaplan & Sadock's comprehensive textbook of psychiatry* (8th ed., pp. 1487-1501). Philadelphia: Lippincott, Williams, & Wilkins.
- Foudraine, J. (1974). *Not made of wood; a psychiatrist discovers his own profession* (H. H. Hopkins, Trans.). New York: Macmillan.
- Fowler, D., Garety, P., & Kuipers, E. (1995). *Cognitive behaviour therapy for psychosis: theory and practice*. Chichester: Wiley.
- Fuller, R. L. M., Schultz, S. K., & Andreasen, N. C. (2003). The symptoms of schizophrenia. In S. R. Hirsch & D. L. Weinberger (Eds.), *Schizophrenia* (2nd ed.), (pp. 25-33). Malden, Massachusetts: Blackwell Publishing.
- Gard, D., Germans-Gard, M., Kring, A., Horan, W. P., John, O. P., & Green, M. F. (2003). Anhedonia in schizophrenia: distinctions between anticipatory and consummatory pleasure. *Society for Research in Psychopathology*. Toronto.
- Gould, R. A., Mueser, K. T., Bolton, E., Mays, V., & Goff, D. (2001). Cognitive therapy for psychosis in schizophrenia: an effect size analysis. *Schizophrenia Research*, 48, 335-342.
- Grant, P. (2005). *Dysfunctional attitudes mediate cognitive impairment, functional outcomes and negative symptoms in schizophrenia*. University of Pennsylvania, Philadelphia.

- Haddock, G., Wolfenden, M., Lowens, I., Tarrier, N., & Bentall, R. (1995). Effect of emotional salience on thought disorder in patients with schizophrenia. *British Journal of Psychiatry*, 167, 618-620.
- Hafner, H. (2003). Prodrome, onset and early course of schizophrenia. In R. M. Murray, P. B. Jones, E. Susser, J. van Os & M. Cannon (Eds.), *The epidemiology of schizophrenia* (pp. 124-147). Cambridge, U.K.: Cambridge University Press.
- Harrow, M., Marengo, J., & McDonald, C. (1986). The early course of schizophrenic thought disorder. *Schizophrenia Bulletin*, 12, 208-224.
- Harrow, M., & Prosen, M. (1978). Intermingling and disordered logic as influences on schizophrenic "thought disorders". *Archives of General Psychiatry*, 35, 1213-1218.
- Heinrichs, R. W. (2005). The primacy of cognition in schizophrenia. *American Psychologist*, 60, 229-242.
- Hughlings Jackson, J. (1931). *Selected writings*. London: Hodder & Stoughton.
- John, J. P., Khanna, S., Thennarasu, K., & Reddy, S. (2003). Exploration of dimensions of psychopathology in neuroleptic-naïve patients with recent-onset schizophrenia/schizophreniform disorder. *Psychiatry Research*, 121, 11-20.
- Kazdin, A. E. (1982). The token economy: a decade later. *Journal of Applied Behavior Analysis*, 15(3), 431-445.
- Kingdon, D. G., & Kirschen, H. (2006). Who does not get cognitive-behavioral therapy for schizophrenia when therapy is readily available?. *Psychiatric Services*, 57, 1792-1794.
- Kingdon, D. G., & Turkington, D. (1994). *Cognitive-behavioral therapy of schizophrenia*. New York: Guilford Press.
- Kingdon, D. G., & Turkington, D. (2005). *Cognitive therapy of schizophrenia*. New York: Guilford Press.
- Kirkpatrick, B., Fenton, W., Carpenter, W. T., Jr., & Marder, S. R. (2006). The NIMH-MATRICES consensus statement on negative symptoms. *Schizophrenia Bulletin*, 32, 214-219.
- Kopelowicz, A., Liberman, R. P., & Wallace, C. J. (2003). Psychiatric rehabilitation for schizophrenia. *International Journal of Psychology & Psychological Therapy*, 3, 283-298.
- Kopelowicz, A., Liberman, R. P., & Zarate, R. (2006). Recent advances in social skills training for schizophrenia. *Schizophrenia Bulletin*, 32 (Suppl 1), 12-23.
- Kraepelin, E. (1971). *Dementia praecox and paraphrenia* (R. M. Barclay, Trans.). Huntington, New York: Robert Krieger Publishing.
- Lencz, T., Smith, C. W., Auther, A., Correll, C. U., & Cornblatt, B. (2004). Nonspecific and attenuated negative symptoms in patients at clinical high-risk for schizophrenia. *Schizophrenia Research*, 68, 37-48.
- Liddle, P. F. (1987). The symptoms of chronic schizophrenia: a re-examination of the positive-negative dichotomy. *British Journal of Psychiatry*, 151, 145-151.
- Marengo, J. T., Harrow, M., & Edell, W. S. (1993). Thought disorder. In C. G. Costello (Ed.), *Symptoms of schizophrenia* (pp. 27-55). Oxford, England: John Wiley & Sons.
- McGlashan, T. H., & Hoffman, R. E. (2000). Schizophrenia as a disorder of developmentally reduced synaptic connectivity. *Archives of General Psychiatry*, 57, 637-648.
- McKenna, P. J., & Oh, T. M. (2005). *Schizophrenic speech: making sense of bathroofs and ponds that fall in doorways*. New York: Cambridge University Press.
- Milev, P., Ho, B., Arndt, S., & Andreasen, N. C. (2005). Predictive values of neurocognition and negative symptoms on functional outcome in schizophrenia: a longitudinal first-episode study with 7-year follow-up. *American Journal of Psychiatry*, 162, 495-506.
- Miller, D. D., & Tandon, R. (2001). The biology and pathophysiology of negative symptoms. In R. S. E. Keefe & J. P. McEvoy (Eds.), *Negative symptom and cognitive deficit treatment response in*

schizophrenia (pp. 163-186). Washington, DC: American Psychiatric Association.

Morrison, A. P. (2004). The use of imagery in cognitive therapy for psychosis: a case example. *Memory. Special Issue: Memory : Mental imagery and memory in psychopathology*, 12, 517-524.

Nelson, H. (2005). *Cognitive behavioral therapy with schizophrenia: a practice manual*. Cheltenham: Stanley Thornes.

Norman, R. M. G., Malla, A. K., Cortese, L., Cheng, S., Diaz, K., McIntosh, E., et al. (1999). Symptoms and cognition as predictors of community functioning: a prospective analysis. *American Journal of Psychiatry*, 156, 400-405.

Nuechterlein, K. H., & Dawson, M. E. (1984). Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10, 160-203.

Peralta, P. V., Cuesta, M. J., & de Leon, J. (1992). Formal thought disorder in schizophrenia: a factor analytic study. *Comprehensive Psychiatry*, 33, 105-110

Perris, C. (1989). *Cognitive therapy with schizophrenic patients*. New York: Guilford.

Pilling, S., Bebbington, P., Kuipers, E., Garety, P., Geddes, J., Orbach, G., et al. (2002). Psychological treatments in schizophrenia: I. meta-analysis of family intervention and cognitive behavioral therapy. *Psychological Medicine*, 32, 763-782.

Pinninti, N. R., Stolar, N., & Temple, S. (2005). 5-minute first aid for psychosis. *Current Psychiatry*, 4, 36-48.

Rector, N. A. (2004). Dysfunctional attitudes and symptom expression in schizophrenia: predictors of paranoia, negative symptoms and depression. *Journal of Cognitive Psychotherapy: An International Quarterly*, 18, 163-173.

Rector, N. A., & Beck, A. T. (2001). Cognitive behavioral therapy for schizophrenia: an empirical review. *Journal of Nervous and Mental Disease*, 189, 278-287.

Rector, N. A., Beck, A. T., & Stolar, N. M. (2005). The negative symptoms of schizophrenia: a cognitive perspective. *Canadian Journal of Psychiatry*, 50, 247-257.

Rector, N. A., Seeman, M. V., & Segal, Z. V. (2003). Cognitive therapy of schizophrenia: a preliminary randomized controlled trial. *Schizophrenia Research*, 63, 1-11.

Rosenfarb, I. S., Goldstein, M. J., Mintz, J., & Nuechterlein, K. H. (1995). Expressed emotion and subclinical psychopathology observable within the transactions between schizophrenic patients and their family members. *Journal of Abnormal Psychology*, 104, 259-267.

Selton, J.-P., Wiersma, D., & van den Bosch, R. J. (2000). Distress attributed to negative symptoms in schizophrenia. *Schizophrenia Bulletin*, 26, 737-743.

Sensky, T., Turkington, D., Kingdon, D., Scott, J. L., Scott, J., Siddle, R., et al. (2000). A randomized controlled trial of cognitive-behavioral therapy for persistent symptoms in schizophrenia resistant to medication. *Archives of General Psychiatry*, 57, 165-172.

Stolar, N. (2004). Cognitive conceptualization of negative symptoms in schizophrenia. *Journal of Cognitive Psychotherapy: An International Quarterly*, 18, 237-253.

Terrier, N. (2005). Cognitive behaviour therapy for schizophrenia - a review of development, evidence and implementation. *Psychotherapy and Psychosomatics*, 74, 136-144.

Terrier, N. (2006). Negative symptoms in schizophrenia: comments from a clinical psychology perspective. *Schizophrenia Bulletin*, 32, 231-233.