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Depression, Antidepressants and Cognitions: Silent Schemas in the Walking Wounded

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Abstract

Cognitive and pharmaceutical interventions and outcomes for depression are compared, with attention to their long-term effects on a range of negative schema. A subset of residual dysfunctional schema is identified and shown to influence patients during periods of mood or stress induction while under pharmaceutical treatment as well as following cessation of pharmaceutical interventions. The same dysfunctional schema, however, show a more effective and enduring pattern of positive modification in response to cognitive treatment. Medication is acknowledged to provide symptomatic relief from a subset of negative schema, without modifying a full complement of deeply rooted, tenacious negative beliefs. Patients medicated for depression, even in the absence of overt depressive symptoms, carry emotionally masked and dormant maladaptive schemas which may continue, through their carriers' thoughts and behaviors, to influence their own behavior and affect others in their environment who may or may not be medicated.

Keywords: Depression; Antidepressants; Cognitive therapy; Negative schemas

Introduction

This literature review sorts through a variety of cognitive and emotional variables to compare treatment paths and effects for those who suffer from depressive emotional symptoms. After surveying contemporary instruments used to assess depressive thoughts, attention is given to the roles cognitive schemas play in depression. The effects of both medication therapy and cognitive therapies are identified, with special attention to a subset of tenacious, residual, dysfunctional schemas. A case is made to demonstrate the differential effects of medication on moods versus schemas, suggesting that particular negative schemas are more resistant to pharmaceutical treatment than are moods, and that cognitive therapy more effectively addresses and ameliorates the underlying, resistant cognitive schemas.

In conclusion, medication is acknowledged to provide symptomatic relief without addressing a full complement of deeply rooted, tenacious negative beliefs otherwise shown to be amenable to advanced cognitive therapy techniques. Medicated patients, the 'walking wounded', carry emotionally masked and dormant maladaptive schemas even in the absence of overt depressive symptoms. Those 'silent' schemas, like walking viral pneumonia, have the potential to influence others while the walking wounded remain wrapped in a pharmaceutically 'protected' emotional cocoon. The latent and infective nature of particular schemas, in other words, tends to be masked or suppressed by psychopharmacological interventions.

Assessment Instruments

Cognitive assessment instruments address a subset of negative cognitions from a large continuum. The instruments vary in whether or endorsement intensity is identified by more than a dichotomous 'present' or 'absent' choice. Also, instruments vary in whether they measure clustered symptoms or thoughts. Thought content clusters, if identified, reveal important information about the presence of particular cognitive schemas and point back to developmental wounds, as conceptualized in Bowlby's internal working models [1]. Variations in degree and content of cognitions are key to understanding differential pharmacotherapy and cognitive therapy efficacy for patients with depressive symptoms. These variations also lead to a cultural phenomenon for patients who are medicated for depression and may best be described as the 'walking wounded.' Psychological patterns akin to 'walking pneumonia' characterize patients who function as if they were not carrying dis-eased cognitive filters, even while they are protected from mood dysphoria associated with their own thought patterns.

Contemporary instruments used to assess cognitions for mood-disorder research purposes include: Personal Beliefs Inventory [2]; Cognitions Questionnaire [3]; Cognitive Errors Questionnaire [4]; Beck's Depression Inventory-II [5]; Attributional Style Questionnaire [6]; Ways of Responding Questionnaire [7]; Automatic Thoughts Questionnaire [8]; Hopelessness Scale [9]; Sociotropy-Autonomy Scale [10], and Schema Questionnaire [11].

Of the two most popular instruments, the Alternative Thoughts Questionnaire (ATQ) [8] has demonstrated more sensitivity to and specificity for depression than the Dysfunctional Attitudes Scale (DAS) in most clinical groups. While the DAS discriminates between groups suffering from varying levels of depression, the ATQ provides more sensitive discrimination [12]. The DAS also tends to give a relatively high level of false positives. Both the ATQ and the DAS contain an inventory of negative thoughts which subjects endorse on Likert scales [12]. Cumulative scores are used for assessment purposes in most cases, with a few researchers also engaging in item analysis to reveal significant cluster response patterns. Although the ATQ is considered to be a sensitive measure of depression, it suffers from some diagnostic

inaccuracies [12]. The DAS is both less sensitive and even more often inaccurate diagnostically. Both instruments, however, provide important information about depressive symptoms and treatment efficacies [12].

Cognitive Schemas

Schemas, also known as patterns which are "imposed on reality or experience to help individuals explain it, to mediate perception, and to guide their responses," [13] are said to arise out of nuclear family experiences and subsequently become congruent with a person's self-concept, influencing perceptions and symptom development [14]. Attempts to disrupt schema patterns are met with intense levels of resistance [11]. Medicated or not, a person's schemas can be re-activated under the influence of particular environmental stimuli. Regardless of whether they are destructive, schemas provide individuals with a sense of predictability and comfort [15].

Etiologically, schemas are believed to originate in repeated early childhood experiences, and serve as "screening templates to determine what is processed and what is not" [16]. They narrow the perceptual field to match the brain's capacity for interpreting sensory data. The selectivity with which incoming data is processed further reinforces existing schema patterns.

Increased schema severity, characterized by more pervasive and intense thoughts, is associated with an increased number of stimuli or situations which could generate schema activation [13]. It is believed that the developmental matrix from which schemas arise is typically an unmet need associated with attachment, autonomy, freedom, spontaneity, or self-control. It is less clear to what extent genetic tendencies might predispose a person toward particular schemas in any given situation. Riso and McBride [17] posit "the notion that schemas are latent during non-symptomatic periods and become accessible and impact cognitive processing when they are activated" (p. 6). Since latency implies a dormant state, it is also unclear to what extent those dormant schemas influence one's thoughts and behaviors. If dormancy refers primarily to the host's personal mood-related symptom development, negative 'dormant' schemas remain integrated with the capacity to influence and impact others through the host's communication and behavioral patterns. Schema deactivation can be manifested by symptom (negative mood) reduction without schema modification, unless deep therapeutic techniques have potentiated modification processes.

Hope as a Cognitive Schema

A positive example of how significant schema change impacts therapeutic outcomes involves the presence or absence of hope, an underlying perceptual construct. As an early developmental construct, hope involves a cognitive matrix for adaptive information processing. Its absence, on the contrary, suggests the presence of self-perpetuating pathologies.

Erikson [18] proposed that hope is a virtue arising from successful resolution of the first developmental polarities, trust and mistrust. Dependent upon internalization of a maternal figure, individuals feel confident about the predictable presence of a trusted caregiver. Patterns arising from Erikson's first stage of psychosocial development meet the criteria for archaic schemas and are by definition highly resistant to change. As an indicator of therapeutic success, improvements to a deep level of hopelessness signal increasing flexibility, which may generalize to other dysfunctional cognitive realms as well [19]. High hopelessness levels at the onset of treatment for depression are predictive of treatment dropouts and also characterize those who are nonresponsive to therapy, meaning that their depressive symptoms do not subside even after a full course of interventions. However, changes in hopelessness during the first month of treatment have a high predictive value for positive treatment outcome [20]. Hope is a foundational positive schema upon which change is based and it appears that hope itself is amenable to change in the right therapeutic circumstances. Intractable hopelessness, on the other hand, typically characterizes patients with personality disorders, who maintain a rigid grasp on their problems, destructive patterns, and their related sense of identity [13].

Negative Beliefs as a Basis of Depression

Depression is hypothesized to be rooted in a bed of persistent negative cognitions (schema), out of which depressive symptoms arise [21,22]. Both thought content and process are implicated in this definition, including a dormant negative schema which can be activated or triggered by significant negative life events [23]. Regarding the distinction between levels of negative thoughts, automatic thoughts are considered to be relatively 'surface' byproducts, revealing their 'deeper' cognitive schema origins [14].

The onset and maintenance of negative thinking patterns are both related to clinical depression [24], although the link between neurochemistry or genetic predispositions and depressive schema remains unclear. Depression, therefore, is not just an emotion. At the very least, it also involves negative thoughts which can be assessed on instruments such as the DAS and ATQ. In general, high scores on the DAS are correlated with poorer medication treatment outcome, and indicate the need for additional treatment modalities [24]. There is a limit to how much medications can ameliorate symptoms in the presence of extensive negative thought networks.

When treatment is effective, primary symptoms related to depression can be expected to subside. Among those symptoms, fear of criticism, fear of rejection, and preference for affiliation are critical areas which varied over treatment time for participants with successful therapeutic outcomes, meaning that these specific depression symptoms subsided. Those three critical areas did not vary, however, for treatment non-responders [25]. Negative beliefs maintained a tenacious hold upon non-responders' emotional symptoms, more powerful than the expected effects of medication upon mood.

Antidepressants and Euphoria

According to Moncrieff and Cohen [26], antidepressants have not been shown to cure depression and depression is not caused by an abnormal brain in need of cure. Rather, antidepressants serve to create an abnormal brain state, which causes a variety of manifestations including sedation, cognitive impairment, and stimulation. Stimulation, depending upon idiosyncratic experience, can be either euphoric or agitating, and in low doses can help to improve attention and concentration. Sedation, on the other hand, may decrease the discomfort associated with high levels of arousal.

Common antidepressants such as Paxil, Prozac, Celexa, and Effexor are reported to generate central nervous system side-effects including euphoria (http://www.methamphetamineaddiction.com/side-effects-of-commonly-prescribed-anti-depressant-drugs/ Retrieved Feb. 1, 2017). This pleasing side-effect may be sufficient to neutralize depressive symptoms otherwise generated by residual or dormant dysfunctional

attitudes or thoughts. Although many of the symptoms associated with depression have been shown to subside under pharmacological treatment, certain underlying negative thoughts are also known to persist in a temporarily attenuated state outside of euphoria's reach.

Relationship between Medication and Automatic Thoughts

An automatic thought can be understood as the tip of the schema iceberg. It is a "stream of positive and negative thoughts that run through an individual's mind unaccompanied by direct, conscious deliberation" [14]. Automatic thoughts do not require conscious volition; they activate spontaneously from underlying schema and generate behavioral and emotional reactions. However, they are more accessible and apparent during stressful events, as though the automatic thought volume was raised during those times.

Any discussion about medication as a treatment for depression must account for placebo effects. Drug treatment demonstrates a statistical improvement in symptom reduction for patients who received medication compared to those who received placebos [24], thus mediating against the placebo effect.

However, evidence-based practice indicates that results attributed to pharmaceuticals treatment may reflect a psychological model of therapy. Therapeutic professional support is posited to be a primary causative factor for success even in pharmaceutical-mediated symptom reduction, surpassing other medication related factors [27]. The implication is that professional relationships associated with obtaining prescriptions may address cognitive schema patterns, generating a deeper level of change than would be attributable to chemical intervention alone.

A typical research study compared pre-medication automatic thoughts with post-medication automatic thoughts utilizing an abbreviated version of the Dysfunctional Attitude Scale (DAS) for depressed patients. Results demonstrated the reduction of a large percentage of particular negative beliefs or automatic thoughts when tricyclic antidepressants were administered [28].

The DAS was noteworthy in this study [28] for its ability to identify a statistically significant subset of depressive beliefs which persisted even up to one year following symptom resolution in the medication treatment group. The intractable thoughts endorsed by the subgroup revealed a common schema or cognitive organization style which leads to an intense and exhausting level of environmental scrutiny. Depressed patients were characterized by an emphasis on the need for personal strength, efficacy, and self-reliance; on pessimism; on the need for complete control; on the importance of others' opinions; and on urgency regarding resolution of interpersonal issues. The behavioral manifestations of these thought patterns are described as hypomanic.

In another study, a six-week psychopharmacologic treatment for moderately depressed patients resulted in a significant decrease of negative beliefs, thus confirming the biochemically state-dependent nature of particular cognitive dysfunctions [29]. This study utilized the DAS, but did not report on differences between specific cognitions or schemas. The results confirm that certain medications did reduce negative thoughts, even though additional qualitative information about those cognitions was not assessed.

DAS results suggested that mood is a causative factor in dysfunctional attitudes [24]. The study also noted residual trait effects in the treatment group, confirming the presence of surplus or remnant pathology. Curiously, patients with exogenous depression weighed in with higher initial DAS scores than the endogenous group; the exogenous patients endorsed a higher number of dysfunctional thoughts. For both groups, however, higher initial DAS scores were correlated with poorer responses to treatment. According to Beck and Dozois [14], "When an underlying vulnerability is strong, less stress is necessary to trigger the behavior or disorder; when the predisposition is weak, a greater amount of stress is typically needed before an individual develops the disorder".

An additional study [30] found that medication and cognitive therapy effected similar levels of improvement in participants with depressive symptoms. However, one significant difference emerged between treatment groups. Consistent patterns of improvement in hopelessness, self-view, and mood preceded positive changes in motivation and activity for cognitive therapy patients, but not for medicated patients. A pattern of structural change in schema and mood became the foundation for improved motivation and activity among cognitive therapy patients. The absence of an improved foundational pattern for medicated patients is correlated with a higher risk of recurrent symptoms which can be stimulated by environmental stressors, such as sad mood induction.

Sad mood induction, whereby participants are asked to feel the mood expressed in self-paced slide shows of other subjects' sad facial expressions [31], increasingly revealed the presence of dysfunctional attitudes in patients treated with pharmacotherapy. No similar increases in reactivity were seen in cognitive therapy patients, and this difference was predictive for depression relapse in subsequent years for the medication group [32]. Cognitive reactivity lay seemingly 'dormant' in medicated patients until sad emotions were evoked. Vulnerability to reactive thinking patterns and subsequent depression relapses were not addressed in most studies which otherwise claimed insignificant differences between medication and cognitive therapies.

In addition to the slide show induction technique, sad mood induction may also be initiated by asking participants to read a collection of negative cognitions [33], or recall a variety of unpleasant experiences [34]. Sad mood induction is a laboratory style environmental stressor which has been shown to uncover otherwise dormant negative thoughts in medicated patients.

Evidence indicates a hierarchy of symptom reductions in patients undergoing medication interventions [19]. Pharmacotherapy leads to an improvement in emotional symptoms, which in turn decrease certain aspects of negative thinking, albeit to a lesser degree and with less endurance than for participants who undergo a course of cognitive therapy. Reciprocal interactions can also be said to occur between emotions and thoughts, as though a semi-permeable membrane separates those two domains. When changes occur in thought patterns, especially in deep cognitive patterns known as schemas, emotions are affected. Similarly, but to a lesser degree, changes in emotions, such as those which are generated by medications, lead to a subset of cognitive modifications.

At the organic level, another difference between pharmaceutical and cognitive therapy has been demonstrated for unipolar depression patients [35]. Metabolic activity levels differed between the two treatment modalities, with cognitive therapy revealing cortical-limbic changes, whereas medications generated limbic-cortical pathway changes. The cognitive therapy group generated increased hippocampus and decreased frontal cortex activity. The

pharmacotherapy group results were reversed, with decreased hippocampus and increased frontal cortex activity.

Because the hippocampus is responsible for moving short-term to long-term memory, increased activation during cognitive reconstructions would be reasonable. The frontal cortex is responsible for planning, initiative, personality, and creativity, and is associated with other brain regions to support learning and memory [36]. Since the retention of longer term memories falls under frontal cortex control, an emphasis on creating new schema, which occurs during schema-based cognitive therapy, could require 'quieting' older schema contents. Medicated patients need not rely on new learning pathways, benefitting instead from euphoric treatment effects. Increased frontal cortex activity, for this group, may be generated by chemical stimulation or other unknown interactions.

Cognitive Therapy as a Treatment Factor

Multiple studies attest to cognitive therapy's effectiveness in reducing depressive symptoms during early treatment phases, which is best followed by more in-depth identification and modification of cognitive schemas as treatment progresses [19]. Without this shift from initial basic assessment and modification process to comprehensive world views, the treatment outcome more closely approximates that for medicated patients as well, meaning that there is a higher recidivism rate. While most cognitive therapy approaches have focused on acute phase states, pre-morbid core beliefs are attracting more attention in the interest of long-term improvement.

Research analysis differences may be partly responsible for what appear to be ambiguous results arising out of studies which seek to distinguish between medication and cognitive therapy efficacies. By contrast, when measures of analysis focus on the number of extreme automatic thought responses endorsed by participants, cognitive therapy demonstrates a positive effect over pharmaceutical therapy. Overall, many studies indicate a statistically significant decreased relapse rate for cognitive therapy participants in comparison to participants who received medication [19].

One schema-focused approach to cognitive therapy demonstrated high levels of efficacy [37] among a group of chronic depression patients. The three phased approach included an introductory period, a bonding phase, and up to sixty schema therapy sessions. The Hamilton Rating Scale for Depression revealed significant and enduring symptom relief even at the six month follow-up.

Summary

Research literature indicates that pharmacotherapy decreases depressive symptoms by elevating mood, inducing euphoria, and suppressing a limited number of depressive or dysfunctional thoughts, thus alleviating certain somatic symptoms. However, unexamined or residual negative schemas remain fully integrated despite medication, and their somatic effects are otherwise concealed until evoked by stressful situations. Even though stress and sad mood induction reveal or activate underlying schema doesn't imply that induction alone can activate those functions. Other environmental stimuli may be equally causal, though unexamined. Cognitive therapy, as compared to pharmacotherapy, appears to produce the "most generalizable change and the greatest prevention of relapse" [14].

What are the long-term implications of unveiling previously masked depressive symptomatology when particular schemas are triggered by life's inevitable difficulties? Does latency imbue negative thought patterns with increased or decreased power over time? These unanswered questions require further examination, as does the place of linguistic imagery effects.

The walking wounded carry dormant maladaptive schemas which may be emotionally masked by medication effects, but carry the power to reactivate archaic symptoms under stress. Relatively silent negative cognitive schemas are carried by medicated hosts who are protected emotionally from associated symptoms. Even when not activated, schemas serve to filter perceptions, attributions, actions, and decisions throughout life, especially under stressful conditions. Therefore, these schemas cannot be considered completely silent, and may impact non-medicated others with whom the walking wounded associate.

Cognitive therapy, as distinct from pharmacotherapy, more deeply attends to the negative cognitive schemas which generate depression's enduring causal links. Medication, on the other hand, tends to primarily modify superficial cognitions related to depressive symptoms, leaving a trail of life choices and actions rooted in deeply held negative schema.

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