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Define, Then Treat, the Origins of Depression

A Review of

Experiences of Depression: Theoretical, Clinical, and Research Perspectives
by Sidney J. Blatt
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Psychology's concern with evidence-based treatments, although appropriate from an outcome perspective, can have a deleterious side effect in that it can detract from the more important scientific work of defining the etiological origins, factors, and mechanisms that contribute to psychiatric diseases and disorders. Knowing that a treatment is efficacious without knowing how or why hardly represents a scientific advance, unless one can define the process by which the disorder occurs and demonstrate that the treatment has been designed specifically to address that process. Unfortunately some current psychotherapies, as well as pharmacotherapies, are susceptible to this criticism. For this reason, a book such as *Experiences of Depression: Theoretical, Clinical, and Research Perspectives* reminds us that treatment efficacy begins with thorough scholarship into the origins of the pathology.

Sidney J. Blatt, a distinguished academician and researcher, defines for the reader the experience of depression and then traces its origins to the idiosyncratic conceptualizations that are the products of early familial relationships and other developmental life experiences. In so doing, he demonstrates the logical intercourse between clinical observation, theoretical formulation, and empirical investigation by which psychological science seeks to define and explain the processes of pathology and its treatment. It is his eventual goal that an understanding of the life experiences and other etiological factors that lead to depression will guide the development of more specifically targeted and therefore more effective treatment approaches.

Drawing on his own and others' clinical observations and formulations, Blatt presents an integrated theory of the phenomenology and etiology of depression, encompassing classical and more contemporary psychoanalytic, object-relations, interpersonal, and cognitive-behavioral formulations. An extensive review of the relevant research literature, including the author's own research investigations, provides a compelling case and impressive empirical support for the formulation of two types of depression: (a) *anaclitic*, characterized by marked dependency and susceptibility to the loss of nurturing sources; and (b) *introjective*, characterized by marked self-criticality and susceptibility to guilt-inducing situations. Each is derived from relatively distinct etiological courses, developmental and familial histories, cognitive processes, and clinical experiences, thereby requiring distinct therapeutic interventions. As the author notes, this approach to depressive typology stands in contrast to the usual attempts to differentiate among types of depression based only on symptomatic description (e.g., the *Diagnostic and Statistical Manual of Mental Disorders* [4th ed., text rev.; American Psychiatric Association, 2000]). Because similar symptom patterns can be precipitated by widely divergent conditions, symptom-based typologies have criterion relevance only for symptom-focused treatments and contribute little to understanding of the origins and differential contributions of specific etiological factors.

The Role of Cognitive Structure

An analyst by training, Blatt is quite adept at melding analytic, interpersonal, and cognitive theories to explain the cognitive–developmental processes that account for the two types of depression. Mental representations, or cognitive schemas, acquired in the course of an individual's life experiences "can involve veridical representations of consensual reality, idiosyncratic and unique constructions, or primitive and pathological distortions that suggest psychopathology" (p. 125). Mental representations, so defined, are reminiscent of George Kelly's (1955) The *Psychology of Personal Constructs,* reflecting the defining role played by cognitive structure (vs. content) in the prediction and control of life experiences. Kelly's two-volume work is a comprehensive treatment of the role of cognitive process in psychopathology, and although his book is no longer in print, his influence on the field has been significant. *Structure* refers to the degree to which representations of objects—or the conceptual dimensions used to describe them—are articulated, differentiated, and integrated. Thus, the complexity of an individual's cognitive structure is reflected in the degree to which the individual is able to discriminate between or integrate stimulus objects and their characteristics along the individual's conceptual dimensions. *Pathology*, therefore, can be defined in terms of the degree to which the individual's cognitive structure is inadequate to the task of enabling the patient to adapt to challenging life events.

Blatt is mindful that the uniqueness of each individual cognitive system, derived from highly individual experience, poses complications for understanding and treating depression. Individuals experience and construe situations uniquely, and although there may be a certain degree of consensual agreement as the result of social interaction, individuals differ in their interpretation and experience of events. Thus, the definition of self, others, threat, stress, emotion, and every aspect of an individual's life experience is "in the eye of the beholder." How one responds to future persons and future events is—to varying degrees —cognitively "programmed" by past interpersonal experiences.

Because these cognitive systems are by-products of early life experience, they follow a Piagetian developmental course, enabling Blatt to identify the differential developmental levels and resultant conceptual structures associated with each of the two forms of depression. The author and his colleagues have developed instruments to assess the patient's depressive experience, quality and nature of significant formative interpersonal relationships, and developmental level of object representation. These and related measures have contributed empirical support for the two depression hypotheses. In addition, they also provide clinicians with valuable assessment tools, enabling the therapist to identify and target the patient's specific pathology and thereby individualize the intervention.

However, operationalizing pathology-specific interventions proves to be a challenge. All of the theoretical positions reviewed stress change in the patient's cognitive structures as the essential task in the treatment of depression. Although there are differentials in transference themes, interpersonal issues, and self-other definitions, the method of therapeutic intervention with the two types of depressed patients is essentially the same. "Exploration of the multiple . . . impairments, as well as their developmental antecedents, in the context of a therapeutic relationship, helps patients revise their conceptualizations of self and others in their actual and potential interpersonal relationships" (p. 256).

Blatt's treatment recommendations and the process by which he arrives at them are similar to McCullough's (2000) formulation of the Cognitive–Behavioral Analysis System of Psychotherapy (CBASP) designed for the treatment of chronic depression. Reviewing the relevant literature, McCullough formulated a treatment process that addresses the specific experientially produced, developmentally arrested (also Piagetian) cognitive impairments and transference issues of the chronically depressed individual. Focusing on current interpersonal situations, patients are taught new skills for assessing the accuracy and relevance of their interpretations of these interactions and the appropriateness of the behavioral responses they make to them. In the process, they learn to define realistic and attainable desired outcomes and the behaviors and

interpretations of the interaction that will best achieve their desired outcomes. Thus, patients learn a method for analyzing and changing their construction of events and avert repeated interpersonal failures on the basis of past, irrelevant, and/or inaccurate interpretations and inappropriate behaviors. The remarkable congruity between the Blatt and McCullough treatment modalities is noteworthy. Although they begin at different points along the depressive continuum, they follow similar methodologies in assessing the etiological conditions leading to depression, and then they arrive at similar targeted treatment methodologies.

Although assigning an important role to therapist empathy, Blatt observes that some cognitive–behavioral therapists place less emphasis on empathy as vital to the therapeutic process. This raises an interesting question. If *empathy* is defined as the therapist's ability to comprehend the patient's construction of his or her experience, to communicate this understanding to the patient, and have it confirmed by the patient, then similarity or compatibility in the cognitive structures of the patient and therapist is required for this to occur. Thus, as an interactive cognitive process, it would appear that empathy should be central to cognitive approaches to therapy (a major postulate in Kelly's theory dealt with the importance of patient-therapist cognitive similarity in effecting outcome). Thus, it seems ironic that any cognitive–behavioral therapist should minimize the role of empathy in treatment.

Biological Contributions

Although increased understanding of the etiology of depression is one of Blatt's stated goals, the formulation offered leaves several issues unanswered. The author proposes that a psychological perspective will contribute more to understanding of depression than a biological disease perspective based on a "presumed but as-yet-undocumented chemical imbalance" (p. 8). Anaclitic (dependent) and introjective (self-critical) depression are shown to be associated with specific conceptual structures, which in turn are associated with specific child-parent experiences, but as the author points out, how this occurs—that is, the mechanisms and processes that mediate these associations—remains unclear. The role of biological processes is minimally addressed in the book, although Blatt acknowledges that "A comprehensive developmental model . . . must consider the multifaceted ways in which constitutional, organismic . . . and environmental factors transact to affect development and create vulnerabilities to depression" (p. 228).

Granted, the relatively simplistic concept of a "chemical imbalance" adds little to understanding of the causes of depression. However, continuing study of the human response to chronic stress is revealing a complex mediational role for the combined neuroendocrine systems in affective disorders. In normally functioning individuals, stressful homeostatic challenges to the organism are met by relatively automatic, lower cortical level responses, whereas increasingly complex behavioral, cognitive, sociocultural, or environmental challenges require the adaptive and creative problem solving associated with higher level cortical functioning. Emerging evidence suggests that brain structures that play a critical role in higher level adaptive learning (e.g., the hippocampus) are impaired and even damaged by excessive levels of glucocorticoids in the blood stream, the result of sustained exposure to stressful life experiences or other chronic stressors (see reviews by LeDoux, 2002; Sapolsky, 1998). Thus, the cognitive anomalies associated with depression also appear to involve an impaired ability to make appropriate adaptive responses to stress, and it seems that this impairment is the result of neuroendocrine as well as psychological processes. Whether or how these processes are linked is yet to be determined, but their duality suggests that by combining pharmacological and psychological interventions, treatment effects could be amplified. Keller et al. (2000) conducted a national, multisite study of the efficacy of CBASP and selective serotonin reuptake inhibitors (SSRI), both alone and in combination, in the treatment of chronic depression. Over a period of 16 sessions, they found CBASP and the SSRI to be equally efficacious alone (55 percent and 52 percent improvement rates, respectively) but obtained an 85 percent improvement rate for the two treatments combined.

A Stress–Diathesis Model

In attempting to define the etiological factors and mechanisms by which a pathological condition develops, an inevitable question, familiar to epidemiologists, must be addressed. Many individuals are exposed to many of the risk factors contributing to depression. Why do some individuals get depressed whereas others do not? To address this issue, Blatt proposes a transactional stress-diathesis model. On the basis of the congruency hypothesis, the model "stipulates that psychopathology results from a co-occurrence of a particular genetic or psychological vulnerability (i.e., diathesis) with congruent stressful life events" (p. 232). Here again, we see Blatt's acceptance of the probability of biological involvement in predisposition. It is not that he is dismissing this as a contributing factor. Rather, he has chosen to make his contribution by exploring and defining the psychological parameters of that vulnerability, including its origins in early familial learning experiences, the resultant idiosyncratic cognitive structures that lead to two identifiable forms of depression, the congruent events that precipitate the depressive disorder, and a compelling body of supportive empirical evidence. This is the essential first step and, therefore, an enormous contribution to understanding of the processes that lead to depression.

It remains for future writers to build on the significant clinical, theoretical, and empirical integration achieved by Blatt and to further flesh out the complex interactions of contributing environmental, sociocultural, cognitive, behavioral, and biological (e.g., genetic, neuroendocrine) processes that contribute to the phenomenon of depression. Blatt provides a monumental psychological foundation and sets a high standard for subsequent scholarship in this area.

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