This review addresses the etiology of bipolar disorder and presents the literature within a psychological behaviorism framework (Staats, 1996; Staats & Heiby, 1985). The proposed theory attempts to provide an integrative developmental approach that is grounded in established behavioral principles. The bipolar theory posits 15 hypotheses based on past and concurrent biological and situational factors as well as their interactions with an individual's basic behavioral repertoires. Implications for subclassification and treatment research are noted.

During the past several decades, there has been a proliferation of psychological theories of adult unipolar depression that have been extended to children (e.g., Abramson, Seligman, & Teasdale, 1978; Beck, 1967; Ferster, 1973; Lewinsohn, 1974; Rehm, 1977; Staats & Heiby, 1985). Meanwhile, there has been a paucity of psychological theories of bipolar disorder since Kraepelin (1921) observed mania not only among adults but also among 3% of his child and adolescent patients.

The apparent assumption of most of the research evaluating the etiology and treatment of bipolar disorder has been that this is primarily a biological condition for which primarily biological interventions are indicated. Biological bias has left the psychological aspects of bipolar disorder largely unexplored and the biological research poorly integrated with advances in other areas of investigation (Depue & Iacono, 1989). Fortunately, a number of investigators have expressed concern about this state of affairs (e.g., Akiskal, 1966; Bebbington, 1986; Depue & Iacono, 1989; O’Connell, 1986; Perris, 1986; Rehm & Tyndall, 1993).

The recent developments of psychosocial (Craighead, Miklowitz, Vajk, & Frank, 1998), cognitive behavioral (Basco & Rush, 1996), and family (Miklowitz & Goldstein, 1997) treatments for bipolar disorder are promising but have focused primarily on enhancement of medication compliance. They are extensions of treatments developed for unipolar...
depression and chronic and severe disorders. These treatments are not based upon a psychological theory of the development of bipolar disorder and are not designed as behavioral prevention and change alternatives to psychoactive substances.

It is the purpose of this paper to suggest an integration of the bipolar literature on vulnerability factors in childhood and maintenance factors in adulthood. The guiding theoretical framework for this integration is Staats' (1975) social behaviorism, later referred to as paradigmatic (e.g., Staats, 1986) and more recently psychological behaviorism (Staats, 1996). The theory is an extension of classical and operant conditioning as well as developmental and cumulative human learning principles. Each revision of the theory was accompanied with a broader integration of levels of analysis (e.g., inclusion of organic factors) and additional behavioral principles (e.g., self-administered verbal-emotional stimuli that have directive, affective, and reinforcing effects). Psychological behaviorism (PB) was selected because it has been shown to have heuristic value for organizing the disparate research on intelligence (Leduc, Dumais, & Evans, 1990), unipolar depression (Heiby & Staats, 1990; Staats & Heiby, 1985), anxiety disorders (Hekmat, 1990), and other forms of psychopathology for which there is no generally accepted theory (Eifert & Evans, 1990; Staats, 1996). Although the application of PB theory to unipolar depression includes some mention of bipolar disorder, the utility of the theory for integration of the bipolar literature has not been evaluated previously.

First, bipolar disorder will be described. Second, a summary of general PB theory will be presented. Third, the research investigating the childhood and adult etiology of bipolar disorder will be organized according to the situational, behavioral, and organic factors proposed in PB theory. Finally, 15 hypotheses regarding the etiology of bipolar disorder will be offered and directions for the development of psychological treatments and subtyping are noted.

Definitions and Phenomenology of Bipolar Disorder

The reliability and utility of the distinction of bipolar from unipolar depression has been long established (Leonhard, Korff, & Shulz, 1962). The Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV; APA, 1994) identifies four types of bipolar disorders, all of which include a history of mania, hypomania, or some admixture of mania and depression. However, a review (Rehm & Tyndall, 1993) of the unipolar-bipolar distinction argues that bipolar disorder may involve numerous subtypes so that a history of mania, as defined in DSM-IV, is insufficient to specify a taxonomy.

The disparity of the literature on factors related to bipolar disorder is understandable given the episodic and highly variable characteristics inherent in the definition of a manic episode itself. The definition of a manic episode in DSM-IV (APA, 1994) is heterogeneous in terms of the emotion
involved as well as accompanying behaviors. An "elevated, expansive, or irritable mood" (p. 332) is one necessary criterion. This criterion permits excessive positive emotions (happiness; euphoria) or excessive negative emotions (anger; irritability). This inclusion of two dysfunctional moods under one category is in contrast to the DSM-IV criteria for a major depressive episode in which one negative emotional state, dysphoria or loss of pleasure, is the defining dysfunctional mood characteristic.

Heterogeneity is also illustrated in the DSM-IV criteria indicating that any three or four of a possible seven remaining symptoms can constitute the definition of a manic episode. These include inflated self-esteem or grandiosity, decreased necessary sleep, talkativeness, racing ideas, distractibility, increased activity, and excessive involvement in pleasurable activities such that there is a risk for eventual adverse consequences. For children and adolescents, these behaviors overlap with normal development at some ages as well as with irritable depression, conduct disorder, attention-deficit/hyperactivity disorder (APA, 1994), and schizophrenia (Goodyer, 1992), further obfuscating a clear identification of bipolar disorder among youth.

The characteristics of manic episodes are a challenge to establish because they are relatively rare and the onset is difficult to predict. Prevalence during childhood and adolescence is unknown. Childhood onset of mania among adults exhibiting bipolar disorder has been estimated to occur in 20% of cases (Goodwin & Jamison, 1990). Among adults, a 1 month U. S. adult population prevalence of 0.4% for manic vs. 2.2% for major depressive episodes has been estimated (Regier et al., 1988). Lifetime prevalence of a manic episode has been estimated to be 1.6% and of a depressive episode to be 17.1% (Kessler et al., 1994). Some epidemiological research has addressed the degree of variability in expression of the behaviors included in the DSM-IV (APA, 1994) definition of a manic episode. In their review, Rehm and Tyndall (1993) indicate that racing ideas may be present in 41% to 100% of manic episodes, pressured speech in 75% to 100%, delusions in 44% to 75%, hallucinations in 4% to 40%, heightened activity in 87%, and decreased sleep in 81%.

It is unknown what percentage of individuals exhibiting manic episodes express irritability. However, one review suggests anger may be as common as euphoria (Goodwin & Jamison, 1990) while one study found 8% exhibit irritability only, 30% euphoria only, and 62% both irritability and euphoria (Winokur & Tsuang, 1975). The presence of irritability seems like an important distinction as the affective states of euphoria and anger are not only subjectively different, but may also emit and reinforce quite different operant behaviors (e.g., prosocial yet unproductive gambling versus antisocial dangerousness to others).

Given the stark contrast between the behaviors defining depression and mania, it is not surprising that research comparing characteristics of depression between individuals exhibiting unipolar and bipolar depression has identified a variety of distinctions. This research most
likely involves a subclass of bipolar subjects, as the reported percentage of bipolar individuals exhibiting a history of depressive episodes has ranged from 5% to 100% (Goodwin & Jamison, 1990). The temporal nature of the mood change between depression and mania has been reported to range from less than 2 days to years (Rehm & Tyndall, 1993).

Compared to unipolar depression, depression in a bipolar disorder has been shown to differ on the following characteristics: (a) an earlier average age of onset (Rehm & Tyndall, 1993; Shulman, Tohen, Satlin, & Mallya, 1992); (b) more sporadic, frequent, and rapid severe mood changes but with less chronicity (Winokur, Coryell, Keller, Endicott, & Akiskal, 1993); (c) psychomotor retardation (vs. agitation); and (d) hypersomnia (vs. hyposomnia) (Rehm & Tyndall, 1993).

The importance of the distinctions between bipolar and unipolar depression is unclear. Rehm and Tyndall (1993) conclude in their review of major theories of mood disorders that current approaches are too narrow and very little is known or theorized about bipolar disorder among children and adults. They agreed with a suggestion by Craighead (1980) two decades ago that future subtyping of mood disorders integrate biological, environmental, and psychological factors. The following sections describe a psychological behaviorism theory of bipolar disorder as one step toward addressing the need to provide a framework to study the development of and heterogeneity in the expression of mania. The proposed framework is an extension of the then called paradigmatic behavioral theory of depression (Heiby & Staats, 1990; Staats, 1996; Staats & Heiby, 1985) which focused primarily upon unipolar depression and the determinants of a depressive episode.

Overview of Psychological Behaviorism

Staats' theoretical formulations date back to the 1950s and 1960s. At that time he argued that the further development of behaviorism required a comprehensive and integrative frame of reference, which could be applied to complex human behaviors (Staats, 1956, 1957a, 1957b, 1961, 1963, 1968). In this perspective, the learning principles of classical and operant conditioning are viewed as interactive processes, and stimulus situations are described as having three potential basic functions: (a) they can elicit affect and attitudes, (b) they can reinforce behavior, and (c) they can prompt the direction of behavior, that is, approach or avoidance of a stimulus. This aspect of PB theory is called "three function learning theory" (Staats, 1975, p. 34) or "A-R-D theory" (p. 86), referring to the affective, reinforcing, and directive properties of stimuli. Staats (1975) suggested that when these principles of PB theory are applied to complex human behaviors (e.g., those involved in psychopathology) another theoretical concept becomes useful—the concept of "basic behavioral repertoires" (BBRs; p. 246). BBRs are the result of an individual's developmental learning history and organic conditions.

For heuristic purposes the BBRs are divided into three types:
BIPOLAR DISORDER

emotional-motivational (e-m), sensory-motor (s-m), and language-cognitive (l-c). In actuality, however, most behaviors are regulated by all three types of BBR processes. An individual's BBRs encompass the various forms and patterns of responding which make up an individual's personality (Staats, 1975). BBRs can be regarded as personality characteristics, insofar as they are viewed as having a causal role in determining current behavior.

According to Staats (1975, 1996), there are individual differences in the e-m BBRs people learn, meaning people differ in what emotional responses that have become associated with various external and internal stimuli. These BBRs are theoretically important in explaining psychopathology because they define for the individual which stimuli have attitude-mood altering, reinforcing, and directive (A-R-D) functions. They also, in conjunction with environmental and organic conditions, determine affective states, which in themselves are central symptoms of many disorders.

The s-m BBRs consist of instrumental responses, including basic functional ability, vocational skills, educational skills, social skills, and recreational skills. The s-m BBRs are modified and maintained by operant conditioning and modeling. Skills relevant for adaptation obviously differ developmentally as do the s-m deficits that contribute to and characterize psychopathology. Indeed, a deficit in performance of social roles is included in most DSM-IV (APA, 1994) definitions of disorders, with problems at school being a common marker for children and problems at work a common marker for adults.

The l-c BBRs are conceived as covert language and imagery responses and stimuli, which follow the established principles of classical and operant conditioning. Thus, cognitions may be subject to modification as a result of stimulus contingencies (classical conditioning) as well as reinforcement contingencies (operant conditioning). Cognitions may also become conditioned stimuli or cues for further cognitions. According to Staats' theory, cognitions affect a person's behaviors and feelings, through their connections with the s-m BBRs and the e-m BBRs. Maladaptive cognitions are likely to be associated with performance deficits and excessive affect.

PB theory emphasizes five classes of factors that are likely to play an etiological role in psychopathology. Past organic and childhood situational factors (O1 and S1, respectively) are hypothesized to produce an individual's basic behavioral repertoires (BBRs). The BBRs interact with concurrent organic and concurrent situational factors (O2, O3, and S2, respectively) to produce the symptoms of psychopathology. The distinction between O2 and O3 is a recent development (Staats & Burns, 1992) in which O2 refers to concurrent somewhat stable organic factors which affect behavior through their effects on the BBRs (e.g., brain damage that limits language expression) and O3 refers to concurrent somewhat transient organic factors which do not affect the BBRs yet do have an impact on behavior (e.g., a headache temporarily affecting concentration). Thus, a list of potential etiological factors in
psychopathology involves past organic factors (O1), past situational factors (S1), the BBRs, concurrent organic factors which affect the BBRs (O2), concurrent organic factors which do not affect the BBRs (O3), and concurrent situational factors (S2). The relations among these six potential etiological factors are shown in Figure 1.

![Figure 1](image)

**Figure 1.** Psychological behaviorism's theory of bipolar disorder. Past psychosocial (S1) and organic (O1; O2) factors lead to the development of basic behavioral repertoires (BBRs), which interact with concurrent psychosocial (S2) and organic (O3) factors to produce bipolar symptoms.

**The Psychological Behaviorism Approach to Bipolar Disorder**

Individual differences in predisposing BBRs partly account for the onset and the heterogeneity of a manic episode. When these characteristics do not grossly impair functioning, they are often referred to as hypomania (APA, 1994). As pointed out by others (Rehm & Tyndall, 1993), hypomania can involve heightened reported well-being and productivity in the form of unusually elevated and persistent euphoria, active and creative thinking, and more productive physical activity.

PB theory posits that in the sensory-motor (s-m) BBR area, certain types of potentially adaptive instrumental skills, which yield short-term reinforcement and long-term punishment when exercised, may constitute risk factors for bipolar disorder. These assets are hypothesized to include persuasive conversational techniques, social manipulativeness, and other social, recreational, educational, or occupational skills required for high risk activities. The focus on childhood development of pathogenic assets rather than deficits in the s-m repertoire is a unique aspect of PB theory.

In the language-cognitive (l-c) area, bipolar vulnerable individuals may tend towards grandiose self-labeling. "Positive self-esteem" is usually considered to be an adaptive characteristic for children and adults. However, some individuals may have concomitant deficits in cautionary cognitive processes, which are needed for inhibiting potentially harmful behaviors and for ascertaining negative consequences. Additionally, there may be poor problem-solving skills in generating options and anticipating possible consequences.

In the emotion-motivation (e-m) area, individuals at risk for bipolar
disorder may respond with excessive or inappropriate positive arousal to certain psychosocial stimuli. For example, they may have inappropriately pleasant emotional responses to dangerous situations and imagery. Euphoric mood can also constitute a state of well-being that evokes positive responses from others, again suggesting e-m characteristics of hypomania may function as assets under certain circumstances. This is consistent with studies suggesting that hypomania can constitute an adaptive emotional state (Kendall, Howard, & Hays, 1989).

The sporadic and abrupt nature of manic episodes also suggests that e-m characteristics may have both adaptive and maladaptive functions. Although research cited throughout this article involves diagnosis of a manic episode without a distinction of the e-m states of euphoria vs. irritability, it is possible that these seemingly incompatible mood states involve different determinants. The current PB theory of bipolar disorder primarily considers mania in the form of maladaptive euphoria. However, PB theory recognizes that the determinants of irritability need further explication. This type of negative affect in a manic episode may represent a distinct mood disorder with different determinants and course.

Staats and Heiby (1985) hypothesized that individuals at risk for bipolar disorder exhibit s-m BBRs, such as an adolescent street racing or an adult playing casino poker, that at some points receive short-term reinforcement (S2) from the successful exercise of their predisposing skills. The reinforcing stimuli also elicit positive emotional responses that would elevate mood and cue further BBR activity. The short-term reinforcement and emotion elicitation would increase the frequency of the s-m skilled behaviors, thus producing more short-term reinforcement and more positive emotion that would add up to excessive euphoria. Furthermore, there would be little inhibition of these short-term rewarding, yet risky, activities, due to the deficit cautionary l-c BBRs. The short-term reinforcements could then elicit increasingly numerous and exaggerated positive self-statements, also due to l-c BBRs. These positive self-statements, in turn, would elicit and add to the euphoric e-m responses. Thus, bipolar vulnerability BBRs would make an individual susceptible to a process in which s-m, l-c, and e-m BBRs interact to accelerate the emergence of manic symptoms, referred to as the compounding principle (Staats, 1996). This process could progressively flood a bipolar individual with feelings of elation and success and escalate to an episode of mania.

This compounding process would be reversed when the long-term punishments stemming from the exercise of pathogenic skills (risky s-m behaviors) start to emerge. Manic behavior could result in a mounting number of set-backs and failure experiences. For the individual with the attitudes (e-m) and cautionary, problem-solving (l-c) and instrumental (s-m) skills needed to restore a positive situation (S2), euthymia rather than a depressive episode would be expected to follow setbacks. However, if these failure experiences elicit negative self-statements (l-c) and negative emotional responses (e-m), the cumulative effects of unpleasant events (S2) interacting with vulnerable bipolar BBRs would ultimately produce
dysphoria and symptoms of a depressive episode (Staats & Heiby, 1985). Thus, the constellation of bipolar vulnerability BBRs would contribute to both the manic/hypomanic as well as the depressive aspects of a bipolar disorder, depending on the adequacy of dysphoria-regulating skills.

As stated above, the bipolar BBRs are hypothesized to specifically yield the type of behaviors that result in both short-term rewards and long-term punishments. Because the long-term punishments are delayed, the punishments would not be automatically associated with the skill behaviors, and thus the inappropriate s-m skill behaviors would not necessarily diminish. This would also be true for the deficits in e-m and l-c repertoires. Thus, the individual would be left with the predisposition for further manic (and, for some, depressive) episodes. Normal mood periods would be experienced during times when the individual's life situation (S2) provides few or no stimuli that would lead to a compounding episode. For example, the person may be preoccupied with a harmless hobby, such as an adolescent frequently skateboarding in the parent's driveway. Then, when opportunity presents itself, such as an invitation from skateboarding peers to road race, exercise of the pathogenic skills would again start a manic compounding process. In this sense, the concurrent psychosocial environment would exert an influence over the course of bipolar disorder with an interaction of BBRs and S2.

It is also hypothesized in PB theory that O1, O2, and O3 factors may be causal in bipolar disorder. The O1 factors could involve genetic or prenatal conditions such as temperament with heightened tendency to euphoria. The O2 and O3 factors would involve direct biological factors heightening euphoria, such as injury to the frontal lobes or diet-related insulin dysregulation, respectively. Given the complexity of the potential causal factors proposed, it is not surprising that there is a great deal of difficulty predicting the temporal and behavioral characteristics of bipolar disorder (Gottschalk, Bauer, & Whybrow, 1995; Rehm & Tyndall, 1993).

The following sections will focus on past research pertaining to each of the hypothesized etiological factors of the PB framework—O1, O2, O3, S1, S2, and the BBRs. After the summary, additional etiologic hypotheses based on the PB formulation are generated.

Past Organic Factors (O1)

In principle, factors in this category may include all organic variables that affect learning of the BBRs including genetics, early brain injury, birth trauma, and exposure of the fetus or infant to toxic substances. Of all these possible past organic factors most of the literature on bipolar disorder has focused on genetic factors in terms of family concordance. Partially, this may be because of DSM-III-R (APA, 1987) diagnostic criteria, which called for a diagnosis of organic Mood Disorder if a definite organic cause can be ascertained, and DSM-IV (APA, 1994) criteria, which define a special category for Mood Disorder Due to a General Medical Condition.

Several studies, reported in Schlesser and Altsheuler (1983), have
indicated that an estimated 80% to 90% of bipolar individuals have at least one first-degree relative with a mood disorder. For unipolar depressed individuals this rate is only an estimated 40% to 60%. Monozygotic concordance rates are about 75% for bipolar disorder and 41% for unipolar depression. Dizygotic concordance rates are much less—about 19% for bipolar disorder and 13% for unipolar depression (Kendler, Pederson, Johnson, Neale, & Maathe, 1993; Schlesser & Altshuler, 1983). In addition, a review of studies using pairwise concordance indices of genetic transmission for bipolar disorder concludes an average of 56% concordance among monozygotic twins and 14% among dizygotic twins (Torrey, 1992).

Mendlewicz and Rainer (1977) ascertained mood disorders among biological parents of four groups: (a) bipolar adoptees, (b) bipolar non-adoptees, (c) normal adoptees, and (d) non-adoptees who had poliomyelitis living with their biological parents. The percentages of biological parents with disorders involving mood (bipolar, unipolar, schizoaffective, and cyclothymic) were 31%, 26%, 2%, and 10%, respectively. They also found that for the bipolar adoptees mood disorders were significantly more frequent among biological parents than adoptive parents.

The evidence for a genetic predisposition to bipolar disorder is generally considered to be strong (Biehar, Weissman, Gershon, & Hirschfeld, 1988; Kendler et al., 1993; Schlesser & Altshuler, 1983). However, the family and twin studies do not rule out a psychosocial transmission of bipolar disorder, for example, through cross-generational modeling and greater environmental similarity for monozygotic twins than for dizygotic twins. Adoption studies come closest to ruling out the effect of psychosocial factors but fall short in that there is usually some degree of psychosocial contact with biological parents before adoption. For example, Mendlewicz and Rainer (1977) included adoptions within the first year of life.

In summary, it is probably warranted to say that genetic endowment (O1) may have an etiologic role in some cases of bipolar disorder. However, one may also argue that the genetic research provides an indication of the existence of psychosocial etiological factors because the monozygotic concordance of 56% to 75% for bipolar disorder leaves 25% to 44% of the variability of the occurrence of bipolar disorder to be explained in terms of other factors. Similarly, Hoffman's (1987) finding of differential responses to lithium among bipolar identical twins suggests that nongenetic factors also play a role in bipolar symptomatology. However the evidence for genetic factors is interpreted, one implication is that there should be behavioral risk factors apparent in early childhood.

Past Psychosocial Factors (S1)

O'Connell (1986) called for greater attention to psychosocial factors in theorizing about bipolar disorder. His argument was cogent, but, unfortunately, he presented little evidence for childhood psychosocial etiology. There is a paucity of research on behavioral interventions for bipolar
disorder (Craighedt et al., 1998). Couples therapy alone (Davenport, Ebert, Adland, & Goodwin, 1977) and group therapy in combination with medications (Wulsin, Bachhop, & Hoffman, 1988) have been shown to reduce bipolar symptoms. Obviously, far more research is needed in this area because there are many unexplored hypotheses about early learning factors that have theoretical and prevention implications.

The poor understanding of S1 factors in bipolar disorder is in stark contrast to the state of the science for unipolar depression, for which there is a large learning-based treatment literature indirectly implicating early learning causal factors and thereby supporting the investigation of early prevention and behavioral and situational change techniques. For example, a recent review comparing behavioral versus antidepressant medication interventions for unipolar depression concluded that the former is more effective and less expensive (Antonuccio, Thomas, & Danton, 1998). If past psychosocial events were demonstrated to play an etiological role in bipolar disorder one would need to explain the causal mechanisms involved. The PB frame of reference may provide one explanation by describing the childhood development of basic behavioral repertoires (BBRs) that are vulnerability factors for psychopathology and targets for treatment and prevention programming. For an explication and summary of the empirical support for the learning principles guiding the development of the BBRs, see Staats (1975, 1996).

At present, PB theory only suggests the type of learning histories that can result in pathogenic BBRs. For example, bipolar histories including significant others with naively optimistic or happy-go-lucky attitudes can encourage an overly positive emotional conditioning to high-risk situations (e-m BBR) and can result in bipolar individuals failing to learn prediction of long-term future danger (a deficit l-c BBR). Also, a childhood environment providing learning opportunities for risky behavior (s-m BBR) and grandiose self-labeling (l-c BBR) can increase the risk for bipolar disorder. For example, significant others in a bipolar person’s developmental history may have provided positive emotional temporal associations (classical conditioning), modeling, and reinforcement for the thoughts, feelings, and actions involved in gambling and exaggerating one’s prospects.

Studies reporting the family history of bipolar disorder have not looked specifically at learning factors and environmental conditions (S1). They do, however, report a significant relationship between a history of bipolar disorder by a nuclear family member and meeting the diagnostic criteria for a manic episode (e.g., Winokur et al., 1993). One study of family interactions suggested the parents of adults exhibiting bipolar disorder demonstrated more odd word usage and sentence construction than parents of controls (Miklowitz, Velligan, Goldstein, Nuechterlein, & Gitlin, 1991). The authors did not specify whether the odd word usage by parents involved modeling the language-cognitive characteristics of bipolar disorder during childhood or whether the language was necessarily odd in order to respond to the individual speaking during a manic episode. Another study found that adults with bipolar disorder were
more likely than matched controls to be treated as a "special child" in the family (Glassner & Haldipur, 1985) suggesting an unspecified effect of training. Similarly, children living with bipolar vs. normal parents tend to exhibit more irritability, more positive self-image, greater number of interests and hobbies, and generally more emotion (Goodwin & Jamison, 1990), although it is unknown if these children developed bipolar disorder.

Although the above findings do not provide causal evidence and possibly involve a confound of O1 and S1 factors, they do permit the speculation that some early learning of the language deficits in bipolar disorder derives from parental modeling and reinforcement of pathogenic BBRs. Further research into the behavior of significant others is needed to establish the mechanisms by which bipolar vulnerability BBRs develop during childhood and adolescence.

Basic Behavioral Repertoires (BBRs)

The concept of the BBR role in bipolar symptoms implies the existence of personality risk factors. Personality has played a small role in theories of bipolar disorder since the development of psychodynamic theory (e.g., Freud, 1933), which has received little support in positing that mania is a defense for depression. Similarly, attempts to identify bipolar personality types have not been successful (Perris, 1992). However, one behavioral position has included a focus on personality vulnerability factors. Depue and Iacono (1989) suggested that bipolar symptoms are correlated and regulated by an integrated behavioral system, called the "behavioral facilitation system" (BFS; p. 458). They hypothesized that, among bipolar individuals, this BFS is susceptible to dysregulation by reinforcement and punishment. The concept of a BFS is useful insofar as it provides a target personality construct for investigating the effects of psychosocial and organic factors, although this has not been made explicit.

In terms of evidence for bipolar personality factors, one starting point is the clinical observation that bipolar individuals have greater and more frequent than average mood swings (DSM-IV; APA, 1994). If this phenomenon reflects a personality characteristic, one would expect a chronicity of greater than average variation in mood—even during so-called euthymic periods, as several authors have argued (Depue & Fuhrman, 1987; Depue et al., 1981; Wetzel, Cloninger, Hong, & Reich, 1980). At least one study (Depue et al., 1981; Study V) has documented such pervasive mood variation. These authors utilized a self-administered checklist and found greater than average behavioral variability (including variability of mood) on a day-to-day basis among subclinical bipolar individuals than among noncase individuals.

One review of the comorbidity of personality disorders and depression concludes there is a small body of research involving bipolar disorder individuals that reports rates of at least one personality disorder ranging from 23% to 62% (Shea, Widiger, & Klein, 1992). The studies reviewed reported over nine different personality disorders listed in DSM-
III-R (APA, 1987), with no apparent common disorder represented. This may be caused in part by small sample sizes, the heterogeneity of bipolar disorder, and the lack of specificity of personality deficits defining personality disorders (Goodwin & Jamison, 1990) that may be relevant to mood regulation.

It appears, however, that the search for bipolar personality risk factors may benefit from an analysis in terms of e-m, I-c, and s-m BBR patterns. The PB theory's tripartite view of BBR risk factors is consistent with prior formulations of bipolar behavior in which personality characteristics are classified as involving mood (e-m), cognition (I-c), and activity (s-m) (Akiskal, Djenderedjian, Rosenthal, & Khani, 1977). Global personality traits, such as aggressiveness, extroversion, or dependency, often involve more than one type of BBR and are likely to obscure specific bipolar BBRs. Also, there is little evidence that global personality traits are significantly deviant among bipolar individuals before or in between mood episodes. For example, MacVane, Lange, Brown, and Zayat (1978) compared 35 bipolar and 35 normal subjects and found no differences on the Social Desirability Scale (Crowne & Marlowe, 1964), Internal and Powerful Other Locus of Control Scales (Levenson, 1972), Personal Orientation Inventory (Shostrum, 1966), Embedded Figures Test (Witkin, Oltman, Raskin, & Karp, 1971), or the Brief Psychiatric Rating Scale (Overall & Gorham, 1962).

Angst and Clayton (1986) reported similar results with regard to premorbid personality traits among bipolar subjects. Scores on the Freiburg Personality Inventory (Fahrenberg, Selg, & Hampel, 1970) were available from the 1971 Swiss military examination for 87 male subjects with various psychiatric diagnoses. The bipolar men scored within the normal range on each of the three factors of the inventory—aggression, extroversion, and autonomic liability.

Results such as those referred to above call for different methods of assessment focusing on more specific aspects of the e-m, I-c, and s-m repertoires. In the following sections, studies which are more specifically relevant to bipolar BBRs have been grouped together according to their possible primary association with Staats' (1975, 1996) three types of BBRs. This separation is mainly useful for heuristic purposes, particularly in devising prevention and intervention strategies. For example, I-c deficits may be best treated with cognitive restructuring, s-m deficits with operant and modeling techniques, and e-m deficits with exposure/desensitization approaches.

**Emotional-motivational (e-m) BBRs.** So far, the literature has not directly addressed the task of mapping out bipolar e-m response patterns as they develop during childhood and adolescence. This is an odd state of affairs as bipolar disorder is defined largely in terms of affect. PB theory hypothesizes that people with bipolar disorder have learned since childhood to experience exaggerated emotional responses to certain stimulus situations. As indicated earlier, the e-m characteristics of bipolar disorder with maladaptive euphoria rather than irritability is the major focus of the present PB analysis.
There is some indirect evidence for e-m deficits in bipolar disorder. Montag and Birenbaum (1986) administered the Clinical Analysis Questionnaire (CAQ; Delhees & Cattell, 1971) and the Sensation Seeking Scale (Zuckerman, 1979) to 765 male job applicants. The results yielded a relationship between sensation seeking and hypomania (scores on the agitation scale of the CAQ). Their study provided evidence for a possible subclinical personality characteristic. This finding is in keeping with the PB hypothesis that individuals with bipolar vulnerable e-m systems respond differently than those with normal e-m systems. Specifically, it may be that threatening situations for most people are experienced as pleasantly thrilling by euphoric bipolar individuals both before and during a manic episode. Additionally, it may be positive psychosocial events rather than negative ones (S2) which are important in eliciting exaggerated bipolar emotional responses, because it is the elicitation of euphoria as opposed to anxiety that initiates bipolar symptoms in PB theory. There is some indirect evidence in support of this notion that negative life events are not necessarily etiologic. Depue, Kleiman, Davis, Hutchinson, and Krauss (1985) presented cyclothymic and normal individuals with a mild stressor (simple mathematics problems) and found no differences in subjective ratings of stress on a 5-point scale nor in changes of cortisol secretion.

The PB model calls for research on whether children at risk and bipolar persons overrespond to positive events, particularly when predicting mania involving euphoria rather than irritability. Such evidence is scanty, but there is some suggestion that bipolar individuals may manifest relatively positive evaluations of situations involving risk or threat (e.g., Montag & Birenbaum, 1986). The area of e-m BBRs needs further studies on differential euphoric and anger responsiveness to stimuli in order to identify excessive emotional arousal as a vulnerability factor for bipolar disorder.

Sensory-motor (s-m) BBRs. The s-m BBRs consist of instrumental responses to the environment, including basic functional ability, vocational skills, educational skills, social skills, and recreational skills. Staats and Heiby (1985) hypothesized that bipolar individuals have potentially adaptive skills for activities that provide short-term euphoria or a reduction in irritability and long-term dysphoria. Such activities may include promiscuous dating, skillful high speed driving, gambling, financial investments, or various forms of crime. Therefore, both assets and deficits of the s-m repertoire are in need of investigation. There is little research in this area, possibly because the proposed focus includes assets related to bipolar disorder.

There is some indirect evidence relevant to s-m deficits. Kochanska, Kuczynski, Radke-Yarrow, and Welch (1987) found that bipolar severity was associated with deficits in child-rearing skills. Others have shown a correlation between bipolar disorder and marital (e.g., Dunner, Gershon, & Goodwin, 1976) and occupational instability (Tohen, Wateraux, & Tsuang, 1990). A few studies directly measuring social skills among adults with bipolar disorder indicate that under some conditions they can
be charming (e.g., gregarious) while under other conditions the same behaviors can be alienating (e.g., excessive talking) (Segrin, 2000). These findings encourage further investigation of the adaptive and maladaptive s-m characteristics and effects upon family, school, work, and peer relationships (S2) that may be relevant to bipolar disorder.

**Language-cognitive (I-c) BBRs**. The importance of cognition in bipolar disorder has been recognized since the original writings of Kraepelin (Kendler, 1986). Kraepelin found that thought disorganization occurred in both manic-depressive illness as well as dementia praecox, but that it differed qualitatively. Kraepelin recognized that thinking is slowed down during depression. During mania, thinking is characterized by a flight of ideas, (i.e., a jumping from idea to idea with little relatedness between ideas and without completing any one train of thought). Schizophrenic thought, in contrast, is more often characterized as involving elements such as perseveration, use of neologisms, blocking, or clang associations. More recently, the role of cognitions in bipolar disorder has been suggested by the development of a cognitive behavior therapy for this condition (Basco & Rush, 1996) although the focus of this treatment is upon medication compliance rather than modification of the I-c BBRs that contribute to and characterize the disorder.

Within the literature dealing with the psychological functioning of bipolar individuals, studies of cognition have been the most numerous, perhaps because Kraepelin provided a framework to guide such research. Studies have documented rather general I-c deficits among bipolar individuals. Bipolar thinking may be characterized by deficits in memory formation (Calev, Korin, Shapira, Kugelmass, & Lerer, 1986; Henry, Weingartner, & Murphy, 1971; Rosen & Fox, 1986), slow information processing (Saccuzzo & Braff, 1986), speech degeneration (Fraser, King, Thomas, & Kendall, 1986; Miklowitz et al., 1991), playful and loose combinations of ideas (Holzman, Shenton, & Solovay, 1986; Solovay, Shenton, & Holzman, 1987), and overinclusive thinking (Andreasen & Powers, 1974). A review of cognitive theory and research in mood disorders indicates that there has been little progress in specifying more precise cognitive aspects of individuals who exhibit a manic episode (Rehm & Tyndall, 1993). In addition, the definition of I-c deficits in children and adolescents at different ages needs explication. For example, a loose combination of ideas may be normative in a 5-year-old but deviant in a 15-year-old.

The above findings are in keeping with PB formulations. The deficits in bipolar cognitive functioning may engender poor problem-solving and deficit ability to foresee the potential risks in certain actions. Also, reduced cognitive functioning may facilitate less critical acceptance of grandiose self-labeling, or exaggerated beliefs in personal control (Berrenberg, 1987). Research is still needed to determine the extent to which such deficits are produced at different stages of development by inadequate I-c BBRs and interactions with concurrent psychosocial (S2) and organic factors that do not affect the BBRs (O3). If the BBRs are involved, one should find I-c
aberrations during so-called euthymic periods or before onset of bipolar disorder among children and adolescents. In addition, such I-c deficits should be responsive to prevention and treatment training strategies.

Although a few I-c deficits have been identified in bipolar disorder, it is noteworthy that some aspects of these I-c characteristics, such as playful ideas (Holzman et al., 1986), have adaptive potential, as indicated earlier in the sections on hypomania. In a paper entitled "Healthy Thinking," Kendall (1992) concluded that hypomanic cognitions can involve adaptive distortions in the prediction of future events, perhaps when S2 does not involve long-term punishment.

**Concurrent Organic Factors (O2 and O3)**

A number of physical factors have been found to increase the risk of a manic episode. In most cases definite causal relationships have not been established because of ethical limitations of experimentation. Also, the literature has usually not distinguished between O2 and O3, with the former referring to organic conditions affecting the BBRs and the latter to transient conditions directly evoking the manic mood. One exception is a study focusing on late life onset of mania in which it is suggested that geriatric individuals are less likely to report a family history (O1; S1) of affective disorders and more likely to report recent organic factors that may directly affect mood (O3), such as drug side effects (Young & Klerman, 1992). Most research on organic factors has involved electroconvulsive therapy (ECT), antidepressants, and sleep-reduction among adults.

Various studies have documented the mood-elevating properties of sleep deprivation among individuals with bipolar disorder. For example, Wehr, Sack, and Rosenthal (1987) observed that 9 of 12 subjects with bipolar disorder experienced mania or hypomania following one night of sleep deprivation. Similar mood elevating properties have been noted for electroconvulsive therapy (Lewis & Nasrallah, 1986) and for antidepressants (Wehr & Goodwin, 1987), although the mechanisms relating these organic factors to euphoria have not been explicated.

The evidence for the mania-inducing properties of sleep deprivation, ECT, and antidepressants has primarily implicated persons who already have or are at risk for developing bipolar disorder. Thus, the evidence fits the PB notion of an interaction between concurrent organic factors and other etiological factors. There appears to be no evidence that current organic factors (O2 and O3) are either necessary or sufficient in producing bipolar disorder. Partly, this is due to the consensus, indicated earlier, that the definitional criteria for a manic episode should exclude mood disturbances with obvious organic precipitants (DSM-IV; APA, 1994, p. 332).

One type of indirect evidence for O2 and O3 involvement in the etiology of bipolar disorder derives from the psychopharmacological treatment outcome literature. Although the effects of such treatments do not provide evidence of a biological cause, they would be consistent with such a possibility. In other words, if a concurrent organic factor (e.g., lithium) can reduce bipolar symptoms then, perhaps, there is a concurrent organic factor that is maintaining the bipolar symptoms.
Dysregulation in the dopaminergic system is one hypothesized organic precipitant of excessive positive affect (Depue & Iacono, 1989; Shelton, Hollon, Purdon, & Loosen, 1991) although it remains to be established whether such dysregulation is genetically transmitted (O1), disrupts behavioral expression of BBR assets (O2), and/or directly elicits manic affect (O3). Such distinctions must await synthesis of various neuromodulatory systems and the investigation of the role of such systems in mood (Goodwin & Jamison, 1990). Furthermore, the organic level of irritable mood in some cases of mania remains to be explored. However, it must be kept in mind that the efficacy of biological treatments does not weaken the argument for psychosocial etiology, in some cases. In recent reviews, Tyrer (1985) and O’Connell (1986) reported that the overall response rate to ongoing lithium treatment was about 70%. While it is important to consider dosage, compliance, concomitant medications, and so forth, this response rate suggests that a substantial proportion of individual differences must be accounted for by factors which lithium does not counteract. These might include the BBRs and S2.

Concurrent Psychosocial Factors (S2)

Some reviews of the literature conclude that life crises may precipitate a first mood episode in bipolar disorder but that later episodes seem less dependent on stress and that life stress is neither necessary nor sufficient in causing a mood episode (O’Connell, 1986; Post, 1992). In some studies, no relation between stressful life events and onset of mania has been found (e.g., Sclare & Creed, 1990). Unfortunately, studies of precipitating life events have been correlational or retrospective and have not permitted causal inferences or an inspection of interactions with organic or personality factors, as is true of most studies relating to the etiology of bipolar disorder.

Chung, Langeluddecke, and Tennant (1986) interviewed first admissions with diagnoses of hypomania, schizophrenia, and schizophreniform psychosis to assess life stress for the 6 months preceding onset of their symptoms. Only the schizophreniform psychotics had experienced significantly more stressful life events than their controls. At least for hypomania, precipitating life stress does not appear to be greater than for schizophrenia. However, it is proposed by PB theory that in many cases the onset of bipolar symptoms is preceded by pleasant events to which bipolar-prone individuals overreact (Staats & Heiby, 1985).

Inappropriate social support may be another concurrent psychosocial factor that may increase the risk for developing bipolar disorder. In one study, deficient social support predicted lithium treatment outcome among bipolars, in contrast to demographic factors such as social class, education, or age, which did not predict outcome (O’Connell, 1986). Also, Miklowitz, Goldstein, Nuechterlein, Snyder, and Mintz (1988) found that high rates of family members’ critical comments and emotional over-involvement (expressed emotion) and unsupportive family interactions with the bipolar patient (affective style) predicted relapse of discharged
bipolar patients at a 9-month follow-up. Inappropriate current family interactions with a bipolar member were reported in a similar study (Miklowitz et al., 1991), mentioned earlier in the section on S1 deficits. Inappropriate social support could include encouragement to engage in high-risk activities or failure to discourage such activities. Also, a finding that social supports are inadequate among some bipolars would raise the question of social skills deficits (BBRs) as etiologic (Segrin, 2000). In contrast, Miller, Kabacoff, Keitner, Epstein, and Bishop (1986), assessed families with the Family Assessment Device (FAD) (Epstein, Baldwin, & Bishop, 1983) and found that bipolar patients' families were significantly less pathological than depressed patients' families on General Functioning and Communication. Moreover, the bipolar patients' families did not differ significantly from nonpsychiatric control families on any of the FAD variables (Problem Solving, Communication, Roles, Affective Responsiveness, Affective Involvement, Behavior Control, and General Functioning). Thus, family social factors may affect the manifestation of bipolar symptoms, but there is little evidence that bipolar families in general are particularly dysfunctional.

Although research on possible psychosocial factors has been increasing (e.g., Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990), the emphasis on psychosocial stress still lacks a theoretical link between psychosocial events and bipolar behavior. Covert stimulus-response mechanisms, manifested as BBRs, may be the missing link. In addition, it may be necessary to measure pleasant events given that some cases of bipolar disorder involve an euphoric (versus irritable) emotional response.

Discussion

The PB theory of bipolar disorder provides a framework for integrating the disparate research into six types of potential etiological factors—O1, O2, O3, S1, S2, and the BBRs. While there is a paucity of research relevant to many of these six types of factors, the theory does offer an additional 15 hypotheses regarding the etiology of bipolar disorder listed below. The theory also has implications for subclassification and psychological treatment and prevention evaluation efforts.

The greatest amount of information is available for the basic behavioral repertoires (BBRs) (especially for the language-cognitive [l-c] BBRs), past organic factors (O1), and concurrent organic factors which do not modify the BBRs (O3). It appears that bipolar l-c functioning is deficit and that there is an exaggerated belief in internal control (Berrenberg, 1987), except in the case of bipolar mothers perceiving little control over their children's maturation (Kochanska et al., 1987). Also, it is clear that past organic risk factors (O1) include a genetic predisposition (Schlesser & Altschuler, 1983). Organic risk factors close to the actual time of onset of bipolar disorder (O3) may include ECT (Lewis & Nasrallah, 1986), antidepressants (Wehr & Goodwin, 1987), or sleep deprivation (Wehr et al., 1987).

Information on the other PB factors in the childhood development of
vulnerability for bipolar disorder is sparse and is mainly based on indirect evidence or clinical observations. It is in these areas that the PB model is likely to make the greatest contributions, by providing a rationale for hypotheses that are amenable to empirical investigation. The most salient of these hypotheses are listed below.

**Past Psychosocial Factors (S1)**
1. Bipolar individuals have had developmental histories in which significant others modeled and shaped risky types of activities, such as gambling, crime, or substance abuse.
2. Significant others punished a cautious or realistic attitude towards the future and modeled and reinforced impulsivity and, perhaps, denial or disregard of consequences.
3. Bipolar individuals learned (from the modeling and shaping by significant others) to use grandiose self-labeling and denial as ways to cope with stress as well as to enhance feelings of elation and confidence in response to superficial successes.

**Concurrent Psychosocial Factors (S2)**
4. Manic episodes involving euphoria may be triggered (compounding principle) by pleasant events, such as superficial successes (e.g., winning at gambling) and major life events (e.g., school honors; job promotion).
5. Bipolar individuals may lack adequate social support systems that involve significant others who model and reinforce euthymic emotions, veridical self-labeling and cautionary statements, and safe behavior.
6. Significant others may provide reinforcement for elevated emotions, grandiose self-labeling, and risky behavior.

**Transient Concurrent Organic Factors (O3)**
7. Medical problems, such as sleep deprivation, may precipitate the onset of bipolar symptoms.
8. Medical treatment, such as antidepressant medication, may be iatrogenic.

**Emotional-Motivational Basic Behavioral Repertoires (e-m BBRs)**
9. Bipolar emotional responses to certain stimulus situations are excessive (i.e., nonveridical).
10. Bipolar individuals respond with positive emotional arousal to situations that others would find threatening or anxiety provoking.

**Sensory-Motor Basic Behavioral Repertoires (s-m BBRs)**
11. Bipolar individuals have above average skills for engaging in risky types of activities, which produce short-term reinforcement and long-term punishment. These may include persuasive conversational techniques or social manipulativeness.
12. Bipolar individuals have deficit social skills in maintaining adequate social support networks.

Language-Cognitive Basic Behavioral Repertoires (l-c BBRs)
13. Bipolar individuals are deficit in estimating long-term negative consequences.
14. Bipolar individuals tend to employ grandiose self-labeling and denial to elevate mood.
15. Bipolar individuals have poor cognitive problem-solving skills.

In considering the variety of potential etiological factors it is important to realize the role of interactions between these influences. For example, genetic factors (O1) may play a role in the acquisition of bipolar-related BBRs. They may, to some extent, underlie the finding of slow information processing (Saccuzzo & Braff, 1986) and memory deficits (Calev et al., 1986), emotional lability (APA, 1994), and perhaps even conversational skills (Fraser et al., 1986). Also, there are likely to be interactions between the BBRs and concurrent factors (O2, O3, and S2). For example, bipolar BBRs may interact uniquely with situations of high “expressed emotion” (Miklowitz et al., 1988) to produce bipolar symptoms, whereas persons with healthy BBRs may simply find ways to avoid such situations without overreacting.

One of the important gaps in the literature is in the area of psychosocial factors. This is an important gap because for many it might create the impression that the sole source of risk for bipolar disorder is found in genetic factors rather than childhood learning. There is increasing evidence that this is not the case (O'Connell, 1986). It is not likely that genetic factors are direct determinants of all bipolar BBR characteristics. For example, a tendency towards grandiose self-labeling or skills required for potentially harmful activities is difficult to explain without the notion of past learning histories. The problem regarding bipolar etiology is discovering the type of learning that interacts with genetic factors to increase the potential for bipolar symptoms. According to the PB theory, such psychosocial factors exist, and need to be identified. The resulting understanding would then guide the development of psychological treatments and prevention strategies for bipolar disorder as well as more accurate and useful subclassifications.

Treatment Perspectives
The attention given to psychological variables in the PB model holds implications for the development of psychological treatments for bipolar disorder. The indication in this and other reviews (e.g., O’Connell, 1986) that bipolar symptoms respond to psychosocial factors suggests that reliably effective psychological treatments and prevention strategies may be developed. The development of an effective psychotherapy for bipolar disorder may help lessen the problems of medications. The available drug treatments are not universally effective, produce side effects (Lerer, 1985; O’Connell, 1986; Tyrer, 1985), and have not been evaluated systematically for children. It is becoming apparent that bipolar disorder is
not a purely biological disturbance, and this calls for a more comprehensive approach to its treatment. There are presently no established psychological treatments for bipolar disorder (Goodwin & Jamison, 1990; Lerer, 1985). However, the recently proposed psychosocial (Craighead et al., 1998), cognitive behavioral (Basco & Rush, 1996), and family (Miklowitz & Goldstein, 1997) treatments for bipolar disorder may stimulate this type of research. Psychological behavioral prevention and treatment of bipolar disorder would focus on training directed at avoiding or ameliorating BBR deficits as well as improving concurrent organic and psychosocial conditions. This review suggests that evaluation of prevention and treatment outcome for bipolar disorder should focus on grandiose self-labeling, assessment of potential long-term risks, control of euphoric responses to pleasant events, and adaptive use of highly developed social, educational, occupational, and recreational skills.

Subclassification
There is variation in bipolar symptoms from individual to individual, as indicated earlier in the section on definitions and phenomenology of bipolar disorder. Differentiation of subtypes can be useful as this may lead to greater specificity and efficacy of treatments. The various manifestations have received different labels over time. These have included “bipolar I disorder,” which involves mania and usually some degree of depression, “bipolar II disorder,” which involves hypomania and the occurrence of significant depression, “rapid cycling bipolar disorder,” which involves at least four mood episodes per year, and “complicated mania” which involves mania plus another physical or psychiatric disorder (APA, 1987, 1994; Black, Winokur, Bell, Nasralla, & Hulbert, 1988; Dunner & Fieve, 1974). However, the evidence shows that such variations in bipolar symptoms do not constitute unrelated subtypes of bipolar disorder, and similarity across subtypes has been documented. For example, cyclothymia and bipolar I disorder have demonstrated similar rates of family psychopathology, lithium response, and type of symptoms (Akiskal et al., 1977; Alarcon, 1985; Coryell, 1982; Depue & Fuhrman, 1987; Depue et al., 1981). However, proponents of subclasses as well as proponents of a continuity model of bipolar disorder have focused on a description of symptoms in seeking support for their positions.

The PB model, in contrast, also draws attention to differences in etiology as a basis for subclassification. This approach is in keeping with Person’s (1986) arguments for a psychological rather than diagnostic analysis of psychopathology. This may be a more legitimate starting point because differential etiology might suggest differential prevention and treatment. Bipolar individuals may differ with regard to predisposing BBR constellations and concurrent psychosocial and organic factors that gave rise to symptoms, despite behavioral similarities. An etiology-based classification of bipolar disorder suggests specific targets for assessment and intervention. Also, different treatment methods may be appropriate for
BIPOLAR DISORDER

different etiologies. As mentioned previously, l-c deficits may be best treated with cognitive restructuring, s-m deficits with operant and modeling techniques, and e-m with exposure/desensitization approaches. Early and concurrent psychosocial factors, such as modeling cautionary statements and highly expressed emotion within family settings, may require environmental engineering, such as in family therapy.

References


