ELSEVIER

Journal of Affective Disorders 109 (2008) 221 – 232

Journal of Affective Disorders

www.elsevier.com/locate/jad

Special review article

Hypomania: A depressive inhibition override defense mechanism

Brad Bowins *,[1]

University of Toronto Student Services, Psychiatry Service, 214 College Street, Main Floor, Toronto, Ontario, Canada, M5T 2Z9

Received 28 June 2007; received in revised form 17 January 2008; accepted 24 January 2008
Available online 5 March 2008

Abstract

Objective: Evolutionary perspectives on bipolar disorders can further our understanding of the origins of these conditions, and assist clinicians in distinguishing normal from abnormal states. Hypomania is unique amongst bipolar conditions in that it seems to have beneficial aspects and can be difficult to diagnose, in contrast to full-blown mania and depression. A theoretical perspective regarding the evolution of hypomania as a defense mechanism is presented.
Method: Literature review focused on the fitness reducing aspects of depression and the fitness enhancing aspects of hypomania/mania.
Results: Of all the adversity inherent in depression, inhibition of physical and mental activity—depressive inhibition—has the most detrimental consequences, and throughout our evolution would have significantly reduced fitness. It is proposed that hypomania evolved as a depressive inhibition override defense mechanism, typically operating in a short-term time frame, to restore physical and mental activity to fitness sustaining or enhancing levels. Over-activity and not mood enhancement enabled hypomania to function as a defense mechanism against the fitness reducing state of depressive inhibition. Contributing to depressive inhibition are the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS), two basic motivational systems. Depressive inhibition consists to some extent of low BAS and high BIS. As human intelligence evolved cognitions inhibiting BAS and activating BIS became amplified, resulting in intensified depressive inhibition.
Limitations: A theoretical perspective.
Conclusions: Given its ability to override depressive inhibition hypomania might be viewed as a natural treatment as opposed to a problem to treat, producing maximal improvement in areas where functioning has suffered the most while typically enhancing social behavior.

Keywords: Hypomania; Mania; Defense mechanisms; Depression; Depressive inhibition; Behavioral activation system (BAS); Behavioral inhibition system (BIS)

Contents

* Tel.: +1 416 978 8070; fax: +1 416 978 7341.
E-mail address: brad.bowins@bellnet.ca.
[1] Private Practice, 2200 Yonge Street, Suite 1700, Toronto, Ontario, M4S 2C6. Tel.: +1 416 322 7935; fax: +1 416 322 7951.

0165-0327/$ - see front matter © 2008 Elsevier B.V. All rights reserved.
doi:10.1016/j.jad.2008.01.028

Despite the extensive literature on bipolar disorder there is minimal attention given to evolutionary aspects and the origins of bipolar states. Akiskal (2005) emphasizes the importance of incorporating evolutionary considerations and principles in understanding the origins of affective disorders. Beyond providing a conceptual framework for understanding these disorders, evolutionary perspectives can help set boundaries of what is normal and abnormal. It is proposed that hypomania evolved as a defense mechanism designed to override depressive inhibition, a state that would have been very fitness reducing during our evolution.

1. Depressive inhibition

Since 'Homo sapiens' evolved just under than 200,000 years ago there has been no change in brain capacity relative to body size (Henneberg, 1998; Previc, 1999; Lang, 1996). Hence, what we experience psychologically and emotionally now was almost certainly experienced throughout our evolution, depression included. In an evolutionary context the most damaging aspect of depressed states would have been inhibition of volition. Himmelhoch, 1998 believed that all the phenomenology of manic-depressive illness derives from depressive inhibition, and that impediment of volition is the predominate clinical feature of depression—"all action of the will is extremely difficult…the transformation of impulses of the will into action meets with obstacles that cannot be overcome without difficulty, and often not at all by the patient's own strength. Depressive inhibition is a fundamental concept and a preferential symptom of depression (Dufour, 1978).

Depressive inhibition impairs motivation, physical behavior, and cognition (Weygandt, 1899 cited in Marneros, 2001; Kraepelin, 1921 cited in Marneros, 2001; Dufour, 1978; Benazzi, 2004, 2007). A depressed person typically finds it difficult to get up and face a day that seems overwhelming, even when the same day a month prior would have been fine. Physical and emotional energy are in short supply, as is motivation to set and achieve goals however small. Concentration is limited making it difficult or impossible to progress with any goals set. A pessimistic outlook with themes of personal inadequacy and probability of failure dominate the cognitive landscape. Libido is diminished or absent and there is little desire to socialize. Nothing seems interesting and there is no pleasure to be found. Physical movements are often slowed and there is no spark to the person's appearance or demeanor.

Within the context of our ancestral past when food and water shortages, and threats from predators and competing hunting-gathering groups were very real dangers, symptoms of depression would have significantly impaired evolutionary fitness. The physical and cognitive limitations arising from depressive inhibition detracted from a person's ability to look after oneself and offspring, avoid predation, function effectively in the social group, and secure a mate. Neglecting important obligations within the social grouping could have resulted in ostracism and banishment, and opportunities for social advancement would be missed. Nonverbal and verbal expressions of the depressive inhibition state would have signaled to others that obligations might not be repaid and that the person lacked desirable genes, given that a positive upbeat appearance and manner is suggestive of desirable characteristics attracting potential mates and allies (Hess and Kirouac, 2000).

Relevant to the concept of depressive inhibition are what have been referred to as the Behavioral Activation System (BAS) and Behavioral Inhibition System (BIS) (Gray, 1987; Fowles, 1988). BAS and BIS are very ancient general motivational systems with the former approach oriented and based on positive appetitive incentive, and the latter regulating sensitivity to threat and non-reward cues, and guiding inhibition or avoidance responses (Gray, 1987; Fowles, 1988; Kasch et al., 2002; Johnson et al., 2000). BAS and positive affectivity are related concepts, sometimes used interchangeably, as are BIS and negative affectivity (Kasch et al., 2002; Campbell-Sills et al., 2004). Positive and negative affectivity can be viewed as expressions of BAS and BIS, respectively (Kasch et al., 2002). High BIS is clearly associated with anxiety disorders, particularly social anxiety (Gray, 1987; Biederman et al., 2001; Schneier et al., 2002; Fowles, 1988; Mick and Telch, 1998; Gladstone and Parker, 2006). High levels of anxiety naturally entail an

inhibition of behavior, as for example a socially anxious person being fearful of speaking out in a group and hence remaining silent.

BIS has also been linked to depressive states (Fowles, 1988; Kasch et al., 2002; Meyer et al., 1999). Kasch et al. (2002) administered the Behavioral Inhibition and Activation Scale to 62 patients with Major Depressive Disorder (MDD) and 27 non-depressed controls. The depressed participants had significantly higher BIS scores. In another study (Meyer et al., 2001) BIS scores showed a concurrent positive association with depression severity. In neither study did BIS scores predict the course of depression. Evaluating high school students over a 4-year period Hayward et al. (1998) found that fearfulness, a component of behavioral inhibition, increased the risk for depression and social phobia. Depression can involve activation of BIS when there is uncontrollable punishment, absence of expected rewards, or malfunction of a neurophysiological substrate (Fowles, 1988).

BAS is closely associated with depression because there is typically a disruption of appetitive motivation (Fowles, 1988). Loss is a core theme associated with depression, and loss implies a reduction in, or absence of, both rewards and cues for reward (Fowles, 1988). For example, loss of your job will mean a loss of income and status, and the cues that activate behavior leading to these rewards. In the study by Kasch et al. (2002) depressed subjects scored significantly lower on BAS than did the non-depressed controls. The BIS/BAS Scale has 3 BAS subscales—BAS Reward Responsiveness (BA-RR), BAS-Drive, and BAS-Fun. In Kasch et al. (2002) clinically depressed subjects also obtained lower scores on all three BAS subscales. BAS-RR and BAS-Drive subscale scores further predicted the course of depressive symptoms over an 8-month period. Following patients diagnosed with MDD weekly for 6 months, McFarland et al. (2006) found that baseline BAS predicted number of MDD symptoms at follow-up, average weekly level of depression, and time to recovery. Kasch et al. (2002) indicate that lower responsiveness to reward and a decreased motivational drive to pursue rewarding stimuli, serve to maintain a depressive episode by making it less likely that a depressed person will seek out positive stimuli or engage in pleasurable activities.

BAS inhibition appears more specific to depression whereas BIS activation is present in a broader range of psychiatric conditions (Mineka et al., 1998; Scholten et al., 2006). BAS and BIS typically operate in a fashion that is complementary, even synergistic. For example, increased aversive motivation lowers appetitive motivation (Fowles, 1988). BIS and BAS are independent entities as is depressive inhibition. The independence of BIS and BAS from one another is reflected in how high BIS contributes to many psychiatric conditions, likely via its influence on neuroticism one of the Big 5 personality traits (McCrae and Costa, 1999), while BAS is more restricted and closely linked to affective disorders. Depressive inhibition appears to be somewhat derived from high BIS and low BAS (Fowles, 1988).

One of the primary differences between depressive inhibition and BIS/BAS resides in the interaction with environmental conditions. Depressive inhibition is largely detached from environmental circumstances with a life of its own, as experienced by the patient and as observed by clinicians. The sun might be shining and circumstances in the person's life objectively quite good, but the inhibition of volition persists. A person in such a situation is frequently told to get over it, a statement reflecting how we commonly link our perception of what mood should be like to environmental circumstances. To a person trapped in the vice grip of depressive inhibition this type of advise makes about as much sense as get over gravity. Even when opportunities for reward are right in front of the person, the inhibition of volition persists triggering frustration for both the sufferer and concerned onlookers.

Unlike depressive inhibition that persists largely independent of environmental circumstances, BIS and BAS are more responsive to changing conditions. When the physical and/or social environment is threatening BIS is activated inhibiting actions. When circumstances communicate that the potential for reward is very low BAS is inhibited leading to conservation of existing resources and reduced efforts to secure additional resources. Seasonal Affective Disorder (SADS) is an example of mood responsive to environmental circumstances. Throughout evolution low light levels implied cold weather, vastly reduced vegetation, and scarcer prey. These circumstances threaten survival and indicate that expending resources is very unlikely to yield a net positive return (Davis and Levitan, 2005). Hence, with SADS there is a feeling of wanting to hibernate, hide away, and not expend resources.

The responsiveness of this condition and BIS/BAS to changes in environmental circumstances is evident in how SADS sufferers respond almost immediately to high intensity light from both artificial and natural sources. Whereas, a person with depressive inhibition is not likely to react to a winter break in a sunny tropical destination, a person with SADS responds in hours or days coming out of their mental and physical hibernation with vastly increased activity. Even those who do not suffer from SADS usually find that sunny warmer days trigger a more active state. One of the dangers for animals living in cold climates is too early warming, such as with deer,

who will become more active metabolically and behaviorally, only to suffer when the freezing temperatures return. BIS and BAS can also respond rapidly to internal states such hunger, thirst, cold, and emotions including anger, fear, and disgust. For example, thirst, hunger and cold overcome high BIS and low BAS, but with unmitigated depressive inhibition people frequently fail to look after their own physical, let alone emotional, needs and might even require hospitalization. Anger can interrupt high BIS and low BAS within seconds to prompt adaptive behavior, whereas, with depressive inhibition anger is likely just turned inwards leading to intensified volitional inhibition. BIS and BAS are very responsive to changes in circumstances that impact on the net positive or negative return from behavior.

BAS and BIS are basic motivational systems present throughout human evolution (Gray, 1987; Fowles, 1988). The most defining feature of our evolution has been the development of intellectual capacities far exceeding those of even out closest cousins the great apes. Intelligence enabled our ancestors to design and utilize technology, function in complex social groupings, and become the top predator with limited natural body weaponry (Calvin, 1994; Morwood et al., 2005). In prior papers I describe how the evolution of human intelligence has amplified emotional states as a byproduct—the Amplification Effect (Bowins, 2004, 2006). Virtually all emotions arise from conscious and unconscious cognitive activating appraisals (Clore and Ortony, 2000; Lazarus, 1984, 1991). Human intelligence amplifies emotions by making the cognitive activating appraisals more intensive and extensive, and adding a temporal dimension. For example, you lose your job and think about all the associated losses, such as missing lunches with coworkers, thereby intensifying the loss. Your thoughts might also extend the loss, by for example thinking, I won't be able to get another job if my skill level got me fired. The temporal aspect can be most damaging with thoughts about the loss occurring over days, weeks, months, and even years. Instead of experiencing the loss strictly in the moment as many animals likely do, the loss is perpetuated over time.

By amplifying emotions generally human intelligence makes us the most emotional of all creatures (Hebb, 1949). By amplifying negative emotions human intelligence contributes to depression and anxiety disorders, and ushered in the need for psychological defense mechanisms (Bowins, 2004, 2006). The same process applies to BAS and BIS, whereby human intelligence amplifies the cognitions that activate or inhibit the relevant motivational system. For example, perceptions of threats in the social environment might be amplified leading to stronger activation of BIS. Fowles (1988) indicates that cognitive distortions influence the impact of environmental cues, potentially amplifying or attenuating their ability to activate the relevant system. During our evolution amplified cognitions for BAS inhibition and BIS activation would have resulted in intensified depressive inhibition. The fitness reducing impact of this intensified depressive inhibition in turn set up the need for a defense mechanism capable of overriding it.

2. Hypomania as a depressive inhibition override defense mechanism

Defense mechanisms traditionally refer to psychological constructs designed to block conscious awareness of unacceptable unconscious stimuli (Vaillant, 1977). In a broader sense, defense mechanisms protect against fitness reducing states (Nesse, 1998). Intensified depression and anxiety states arising from the amplification effect of human intelligence, necessitated the ongoing operation of psychological defense mechanisms, similar to how our immune system must always operate to defend against pathogens (Bowins, 2004, 2006). To Homo sapien psychology, defense mechanisms are what the immune system is to biology. Given the fitness reducing effect of depressive inhibition, it is logical that a defense mechanism would evolve to override it. Imagine a depressed individual in a hunting and gathering context at a time when food resources are scarce and predators about. The depressive inhibition state would severely impair the person's motivation and capacity to seek out difficult to find food while displaying the concentration and vigilance needed to protect oneself and offspring from predation.

Depressive inhibition would also have impaired fitness within the social grouping in several ways. Life within a hunting-gathering group is very dynamic with shifting alliances, sophisticated obligation/entitlement relationships, and ever changing opportunities and restrictions. We often tend to idealize it as an easier way of life, but this fantasy hinders us from appreciating the complexity of life within such a group. To successfully navigate the small p politics, recall and respond to obligations/entitlements, take advantage of opportunities and avoid social violations an individual must be alert and responsive. Furthermore, expression of the depressive inhibition state would have communicated that the individual is not desirable as an ally or mate, increasing the likelihood of social rejection and perhaps ostracism.

Beyond the inability to navigate complex social environments, persistent depressive behavior typically results in reduced social support in anything but the very short run, as any depressed patient will readily confirm. Ostracism and banishment from the hunting-gathering

group could well have followed from persistent depressive inhibition. In circumstances of physical isolation while suffering from volitional inhibition, fitness would have been severely impaired. The possibility of successfully entering into another hunting-gathering group and taking advantage of this unique opportunity, would also have been all but impossible in a state of depressive inhibition. Rejection would have been inevitable, thereby sealing the fate of the individual. In our evolutionary hunting–gathering context depressive inhibition greatly impaired fitness, regarding both adaptation to the physical and social environments. Given that the depressed state would not simply lift in a matter of hours or days, evolution appears to have solved this problem by providing a mechanism to temporarily override the depressive inhibition and allow fitness enhancing behavior to ensue, such as seeking out food while being vigilant for predators.

A defense mechanism designed to override depressive inhibition would have to rapidly increase physical and mental activity to be successful. Mood state would not be as important because the detrimental impact of depressive inhibition mainly results from severely impaired activity (Kraepelin, 1904 cited in Himmelhoch, 1998). As formulated in DSM-IV-TR, euphoric mood is prominent in hypomania and mania (First et al., 2002). However, this perspective stands in contrast to substantial research evidence indicating that over-activity is a more central feature (Benazzi, 2004, 2006, 2007; Benazzi and Akiskal, 2003; Bauer et al., 1994). Kraepelin (1913 cited in Benazzi, 2004) believed that over-activity (increased busyness) is the 'most striking feature' of hypomania. Hecker in 1898 noted that productive over-activity is a core feature of hypomania, as did Falret in 1854 (cited in Benazzi, 2007) in describing circular insanity. Kraepelin, Hecker, and Falret believed that hypomania does not impair functioning, does not require hospital admission, and that over-activity and not mood change is the primary feature (Benazzi, 2004).

Moving ahead to recent research, Benazzi (2007) interviewed 137 bipolar II and 76 MDD patients looking for hypomanic symptoms during various prior episodes. Over-activity (increase in goal directed activities) was the most common hypomanic symptom, and of all such symptoms it had the strongest association with BP-II. BP-II was present in 81% of patients with a history of over-activity, while BP-II was present in only 63.3% of patients with elevated mood. When there were five or more hypomanic symptoms over-activity was present 89.5% of the time. Applying factor analysis Benazzi found three factors—elevated mood including elevated mood and increased self-esteem, a mental activation factor including racing thoughts, and a behavioral activation factor with over-activity—consistent with Kraepelin's three-domain structure of hypomania involving euphoria, flight of ideas, and hyperactivity (Benazzi, 2004). Mood change, either elevated or irritable, was not associated with mental or behavioral over-activity. Assessing 197 consecutive BP-II patients Benazzi (2004) found that 92.8% experienced over-activity compared to 70.5% with elevated/expansive mood. Factor analysis revealed 3 factors—mental activation consisting of racing thoughts, high mood including elevated/expansive mood, and behavioral activation including over-activity. Benazzi (2004) indicates that several other studies have also found an over-activity factor despite different patient populations and methodologies.

Several features support the upgrading of over-activity to a stem criterion for hypomania according to Benazzi based on his 2007 study. These include: (1) Over-activity was the most common hypomanic symptom, (2) over-activity, compared to other hypomanic symptoms, had the strongest association with BP-II, (3) factor analysis found that over-activity factors (both mental and physical) were dimensions independent of mood change, (4) most patients with a history of over-activity were BP-II patients and had a mean of 5.6 hypomanic symptoms during the hypomanic episodes, (5) over-activity had a high positive predictive value for BP-II (81%), and (6) patients with hypomanic episodes with 5 or more symptoms very often showed over-activity (90%), more often than elevated mood (77%).

Benazzi and Akiskal (2003) assessed 126 major depressives and 187 bipolar II patients for a lifetime history of manic/hypomanic symptoms. The hypomanic symptoms present in more than 50% of the combined sample were racing thoughts, increased energy and social activity, and irritability. Racing thoughts represent mental over-activity, while increased energy and social activity comprise physical over-activity. Factor analysis revealed two factors—Energized-Activity and Irritability-Racing Thoughts—both having an over-activity aspect. Irritability-Racing Thoughts characterize a depressive mixed state. The Energized-Activity factor was more applicable to hypomania accounting for the largest variance of the phenomenology of hypomania. This factor consisted almost entirely of psychomotor and social activation items. An interesting aspect of their results pertains to mood—the only 'hypomanic mood' state incorporated into the factor structure of hypomania was irritability, not euphoria. Benazzi (2006) indicates that the core feature of hypomania seems to be activation/over-activation more so than mood changes. Based on an 11 year prospective observation period Akiskal et al. (1995) found that the triad of mood lability, energy activity, and daydreaming (mental activation) predicts switching from unipolar

major depression to BP-II. An increase in physical and mental activity is then very much a part of hypomania both in regards to its initiation and behavioral expression.

For a hypomanic episode to effectively override depressive inhibition over-activity, both physical and mental, is essential. An upbeat or euphoric mood state might well assist with motivation but is not crucial. With increased physical and mental activity a person in our evolutionary past could overcome depressive inhibition and effectively engage in the search for sources of water and food, and defend against predators and competing hunting-gathering groups. There would be more of a focus on the goal at hand and mental alertness to detect predators and valuable resources would be enhanced. Social functioning would improve greatly enabling a person to avoid fitness reducing social rejection and ostracism, and take advantage of sudden social opportunities such as the availability of a mate or shifting alliances. Given that a hypomanic state is associated with a more energetic and positive presentation, it would have a positive signaling effect suggesting that the person is worth being allied with, and indicating 'good' genes to a potential mate.

3. Time frame of hypomania

The time frame of hypomanic episodes is relevant for the perspective that such episodes represent a depressive inhibition override defense. DSM-IV-TR indicates that hypomanic symptoms must be present for at least 4 days (First et al., 2002). However, research shows that the modal distribution for hypomania is 1–3 days (Wicki and Angst, 1991; Akiskal and Pinto, 1999; Benazzi and Akiskal, 2001). Circumstances arising in our evolutionary past where fitness would be greatly diminished by depressive inhibition were probably resolved for better or worse within a very short time frame, such as for example the approach of predators, a raid by a competing hunting and gathering group, or search for water. Preparation and effective actions would transpire over hours or days in most cases.

Speed of onset of a hypomanic episode is another important dimension of the time frame, given that a rapid onset over hours or a most a day or two would be highly advantageous. At least two lines of evidence indicate that the shift from either a depressive or euthymic state into hypomania is sudden. First, rapid eye movement sleep preceded by a night or so of sleep reduction has been found to trigger these shifts (Sitaram et al., 1978). Second, antidepressant-induced shifts from depression into actual mania can transpire very rapidly, as any experienced clinician who has witnessed the phenomenon will vouch for. The patient might be slightly irritable late afternoon and by next morning is in a full-blown manic state. The speed is so fast that discerning hypomania as a transitional state is very difficult. Within our evolutionary context, a defense designed to override depressive inhibition would have to engage quickly and perform optimally over hours or days to confer a selection advantage.

4. Mixed states

Given that the function of hypomania is to override depressive inhibition in the short term, the elimination of all depressive symptoms is not required and would not be expected with a period of activation of 1–3 days. Research suggests that a mixture of hypomanic and depressive symptoms is if anything the norm and certainly not the exception (Marneros, 2001; Carlson and Goodwin, 1973; Cassidy et al., 1998; Akiskal et al., 1998; Benazzi and Akiskal, 2001; Bauer et al., 1994). Kraepelin (1921 cited in Freeman and McElroy, 1999), noted that "very often we meet temporarily with states which do not exactly correspond either to manic excitement or to depression, but represent a mixture of morbid symptoms of both forms of manic-depressive insanity." Weygant (1899 cited in Marneros, 2001) stated "The co-existence of the main symptoms of both typical episodes of manic-depressive insanity, mostly of short duration, is extraordinarily frequent; in some cases the mixed features can occupy the entire episode or at least the greater part of its duration." Marneros (2001) indicates that depending on the definition used 20–74% of affective patients have mixed bipolar states and that this figure has been stable over the last 100 years.

Research supports these perspectives, such as Carlson and Goodwin (1973) who in conducting longitudinal studies of inpatients discovered that dysphoric features persisted throughout the course of manic episodes. Cassidy et al. (1998) found substantial rates of depressive symptoms including dysphoric mood in patients with "pure" mania. Akiskal et al. (1998) noted that 47% of the manic patients in their sample had at least one depressive symptom. Mixed mania occurs in up to 40% of patients with bipolar disorder hospitalized for an acute manic episode (McElroy et al., 1995). Benazzi and Akiskal (2001) found that 73.1% of BP II patients in their sample had a Major Depressive Episode combined with two or more simultaneous intra-episode hypomanic signs and symptoms. Bauer et al. (1994) examined 37 outpatients with at least one prospectively observed manic or hypomanic episode. They found that 73.3% of hypomanic subjects had some depressive symptoms during the episode. They state, "The presence of some depressive-spectrum symptoms during hypomania is the rule rather

than the exception." A mood state that is commonly associated with mania is irritability, perhaps arising from the dissonance experienced when there is both happiness/euphoria and sadness/depression. Cognitive dissonance arises when two inconsistent cognitive events occur simultaneously (Festinger, 1957; Draycott and Dabbs, 1998). The simultaneous occurrence of both happiness/euphoria and sadness/depression definitely constitutes a dissonant cognitive state, and irritable mood is probably the expression of this dissonant state.

5. Adaptive aspects of hypomanic behavior

In overriding depressive inhibition a hypomanic episode would be expected to involve productive and adaptive behavior. While we cannot observe exactly what this would have looked like during our ancestral past, we can examine functioning during a hypomanic state to determine if the physical and mental behavior is beneficial. Akiskal and Pinto (1999) indicate that when there is depression with hypomania (BP II) there can be "supernormal periods of functioning," and that a "considerable number of these patients are able to rebound from their difficult periods, to attain new conjugal or occupational status." Akiskal and Pinto (1999) list the signs and symptoms of a hypomanic episode that speak to the rebound success—cheerfulness and jocularity, gregariousness and people-seeking, increased sexual drive and behavior, talkativeness and eloquence, confidence and optimism, disinhibition and carefree attitudes, reduced sleep need, eutonia and vitality, and over-involvement in new projects. When preceded by depression, hypomanic episodes were observed by Akiskal et al. (1979) to be ego-syntonic, usually pleasurable, socially adaptive, and described by some clinicians as a "flight into health."

Articles detailing the constructive aspects of bipolar states often do not fully distinguish between various expressions such as sub-threshold hypomania, hypomania, mania, or hyperthymic temperament—a dimension of personality characterized by ongoing hypomanic-like behavior (Akiskal, 2005; Johnson et al., 2000; Eckblad and Chapman, 1986). However, each describes behavior that at least under certain circumstances can be highly adaptive. For example, Akiskal (2005) describes extroversion, proficiency in 3 or more languages, eminence, creativity, novelty-seeking and excess activity (activity junkies), and sexual excesses. Johnson et al. (2000) posit that whereas goal attainment in non-bipolars leads to "coasting," the same success in bipolars produces intensified goal seeking. Increases in BAS associated with mania or hypomania is credited with producing this effect. Relevant to this perspective is the finding that in a sample of undergraduate students high BAS was associated with hypomanic symptoms accounting for 27% of the variance (Meyer et al., 1999). Koukopoulos et al. (2003) describe how in rapid cyclers a "nuance" of hypomania including slightly less need for sleep, more active, and more talkative, always remains that does not impair functioning or quality of life, and on the contrary, has many positive aspects.

Examining the matter of positive aspects of hypomania from the perspective of the self, Jamison et al. (1980) asked 35 bipolar and 26 unipolar depressed patients set questions about perceived short and long-term benefits in regards to psychological sensitivity, sexual enjoyment, productivity, creativity, and social outgoingness and ease. The unipolar patients only indicated an increase in sensitivity. In striking contrast the great majority of bipolar men and women believed each of the attributes had either somewhat or very much increased during hypomania. The most significant change for men was increased social outgoingness and ease during hypomania, followed by creativity. Women were equally divided on increased sexual intensity, productivity, and social outgoingness and ease. The authors suggest that a baseline effect applies—the lower the level of functioning prior to the hypomanic episode the greater the change with it. Prior to a hypomanic episode social anxiety and inhibition in men might be worse than their libido, resulting in a greater improvement in social outgoingness and ease than libido during a hypomanic state. By maximally increasing performance in areas of weakness hypomanic episodes can be highly beneficial and adaptive. As Kraepelin (1921 cited in Jamison et al., 1980) mentions "the volitional excitement which accompanies the disease may under certain circumstances set powers free which otherwise are constrained by all kinds of inhibitions."

A very interesting aspect of depressive inhibition that hypomania can set free is social inhibitions. In the Jamison et al. (1980) study both men and women felt more social outgoingness and ease during hypomania, and for men this was the most significant change. Himmelhoch (1998) posits that social phobia and the volitional inhibitions of depression are similar states, and that in some cases social anxiety is an intrinsic presentation of the depressed phase of BP II disorder. Both result in a relative paralysis of instrumental, purposeful behavior. With "pure" depressive inhibition there is an inability to initiate action, and with social anxiety there is inhibition related to fear of humiliation. Himmelhoch (1998) noted that treatment with MAOI's triggered hypomania in 14 subjects with social anxiety. In the hypomanic state these individuals became much more self-assertive to the point where several relatives and friends were offended. In 6 of the 14 the change was described as "optimal adaptation."

Social anxiety and major depression are often comorbid—more than one-quarter of patients with MDD have been reported to have comorbid social anxiety disorder (Fava et al., 2000). Social anxiety often starts at an earlier age than depression and many comorbid patients attribute their depression to the suffering imposed by social anxiety (Schneier et al., 2002). Examining the relationship between bipolar disorder and anxiety, Perugi et al. (1999) found that in their sample generalized social phobia preceded the onset of bipolar disorder and showed a complete remission during hypomania.

Hypomania typically produces social extroversion and diminished social inhibitions. Himmelhoch (1998) found that social phobics who became hypomanic demonstrated extroversion and good social skills such as warmth and humor. According to Benazzi and Akiskal (2003) social activation accounts for a large part of the variance of the phenomenology of hypomania. Examining "hypomanic personality" Eckblad and Chapman (1986) evaluated undergraduate psychology students and compared those scoring high on the Hypomanic Personality Scale to a lower scoring control group. Relative to the controls those with hypomanic personality reported twice the number of close friends in high school, more social interactions, greater propensity to be a leader, and rated themselves higher on sociability and outgoingness. During the interview the hypomanic group was described as poised and articulate, relaxed, and acting older than their age. These individuals tended to experience "speeded up" periods once or twice a month lasting 2–3 days.

Relating hypomania/mania to the larger social system, Price (1967) equated mania with a rise in the social hierarchy and depression to a decline. Extending this concept Gardner (1982) suggests that an alpha status is associated with the manic side of the bipolar spectrum while social submissiveness (omega status) is linked to the depressive side. In an evolutionary context, improved social functioning derived from overriding depressive inhibition could well have meant increased likelihood of assistance with food, shelter, and defense when required, higher social status with better access to resources, more successful mating, and maximal capacity to take advantage of opportunities. Fitness reducing social rejection and ostracism would also have been minimized. These hypomanic benefits were derived from both heightened positive activity and enhanced signaling of desirable traits.

An interesting consideration is hypomania as a personality variant and hypomania as a state. "Hypomanic personality," more commonly known as hyperthymic temperament, is to a large extent the personality equivalent to hypomania (Akiskal, 1996). According to Akiskal (1996) it represents a permanently elevated baseline of hypomanic adjustment. Hyperthymic temperament is positively associated with leadership qualities and novelty seeking, and negatively associated with harm avoidance (Akiskal and Akiskal, 2005). From an evolutionary perspective there are several possibilities regarding the relationship between hyperthymic temperament and hypomania. Two of these possibilities consist of, one, hyperthymic temperament might have served as a template for hypomania as a defense, and second, the success of hypomania as a depressive inhibition override defense might have led to its incorporation into the substrates of personality.

With hyperthymic temperament present in at most 1% of the population (Placidi et al., 1998) and hypomania much more common (Akiskal, 1996), it seems plausible that hypomania evolved first. Akiskal (1995) conceptualizes affective temperaments as the proximal behavioral phenotypes in the pre-morbid course of these disorders. Affective temperaments, such as hyperthymic, are said to be milder expressions or variants of bipolar disorder (Akiskal and Pinto, 1999). Perhaps the fitness enhancing features of hypomania in the context of depressive inhibition led to progressively longer and more personality-based expressions of the defense. Hyperthymia characterized by exploration, high activity, and socially outgoing behavior likely acquired a foothold in personality via its similarity to extroversion, one of the Big 5 personality traits. Akiskal and Pinto (1999) indicate that a hyperthymic temperament protects against clinical depression, and those who succumb usually do so later in life. Hyperthymic temperament is effectively a proactive defense against depressive inhibition, reducing the likelihood of depression and the inhibited state that detracted from evolutionary fitness.

Within an evolutionary context the sequence of BIS/BAS, depressive inhibition, and defense mechanisms, including hypomania and hyperthymia, appears to have been as follows: BIS/BAS predated hominoid evolution and were responsive to fluctuating environmental circumstances. Intelligence as a cornerstone of 'Homo sapien' evolution interacted with primary emotions and BIS/BAS to produce an amplification effect. With emotions and BIS/BAS states amplified, depression and anxiety conditions including social anxiety arose. Most significant from the perspective of evolutionary fitness was depressive inhibition, severely impairing adaptation to the physical and social environments. To counteract the fitness reducing impact of depression and anxiety states generally, psychological defense mechanisms evolved. Given its independence from external and internal circumstances, and tremendous persistence, a mechanism capable of temporarily overriding depressive inhibition, namely hypomania, evolved. Precedents for an override defense mechanism

existed in the form of various physical and emotional states being capable of interrupting BIS/BAS, and brain systems that override others such as predator evasion mechanisms interrupting sleep (Cosmides and Tooby, 2000).

A proactive version of this highly successful override defense mechanism gradually evolved in the form of hyperthymia closely linked to the Big 5 personality trait of extroversion. With the advent of agriculture and towns came more specialized divisions of labor and interaction with strangers as part of the exchange of goods and services. Resource accumulation became possible and acceptable in ways not even remotely feasible amongst hunting-gathering groups. This fairly recent scenario likely ushered in the widespread dissemination of genes for hyperthymic personality. To be hypomanic in an ongoing fashion, is highly adaptive in an environment where huge inequalities in resource accumulation are both feasible and socially acceptable. The hyperthymic temperament reaches its ultimate in adaptive potential in modern industrial society. To be a real go-getter never tiring of life's challenges can be highly rewarding. People even seem to artificially induce hyperthymic behavior as evidenced by the high consumption of stimulants, foremost caffeine in coffee, and in drinks such as Red Bull and pop. Hyperthymia probably represents a personality variant currently undergoing rapid allelic spread and modification. Research in human evolutionary genetics indicates that advantageous alleles modifying existing systems, much like hypomania-hyperthymia alleles modifying depressive inhibition, can spread rapidly particularly in larger populations (Cochran et al., 2006).

6. Mania: a defense over the edge

In comparison to hypomania, which can be difficult to detect clinically because it is typically adaptive, mania is usually obvious given its maladaptive features (Akiskal and Pinto, 1999). While there is justification in separating these two entities, there is more of a spectrum from mild sub-threshold manifestations of hypomania to hypomania to milder mania to full-blown mania where psychosis dominates the clinical picture (Angst and Marneros, 2001). Somewhere along this spectrum a manic state is identified (Akiskal and Pinto, 1999). Considering that physical and mental over-activity is the key feature of hypomania enabling depressive inhibition to be overridden, it is easy to see how this is a defense on the edge. With the brakes off the mental and physical activity accelerate to the point where contact with reality is hard to maintain. For example, thoughts race, attention shifts too rapidly from one stimulus to another, the draw to reinforcers such as sex or drugs becomes irresistible, speech spills out, energy surges to unsustainable levels, and sleep/rest periods disappear.

The main feature distinguishing hypomania from at least full-blown mania is psychosis (mania means psychosis in Greek), such as with grandiose delusions where the person believes he or she has special powers. Setting the stage for such delusions are the positive cognitive distortions that are such an integral part of the mental over-activity of hypomania. In a hypomanic state a person typically has inflated confidence and optimism regarding their capacities and likelihood of success (Akiskal and Pinto, 1999; Eckblad and Chapman, 1986). Eckblad and Chapman (1986) found that while their hypomanic personality subjects displayed higher self-esteem and ambition they did not provide evidence of achievements superior to the non-hypomanic control group, emphasizing the positive cognitive distortion aspect as applied to the self. They also discovered that these individuals put a positive spin on their depressive periods preferring to label them as sadness or feeling down.

More intense distortions of these self-perceptions transform them into delusions. For example, with increased self-confidence during a hypomanic episode a person might believe that he or she has unique insights into religion that need to be shared. With an intensification of this positive cognitive distortion the person develops the belief that the key to religious conversion of the masses resides exclusively in their special God-given knowledge. A positive self-enhancing cognitive distortion has been transformed into a delusion. Akiskal (1996) emphasizes "heightened perceptions" as a cardinal feature of mania. "Coupled with distractible attention and the rush of ideas so characteristic of severe mania, this heightened perceptual set can produce a variety of hallucinatory and delusional experiences." Gardner (1982) posits that mania arises when the social rank expressed by the individual is out of context with reality. The manic person believes he or she is superior to what the social reality allows, an occurrence that rarely happens in the animal world where competition for higher status is ever present. With humans the social distortion can be hidden for some time and hence grow completely out of context with reality.

Whereas hypomania is generally adaptive, full-blown mania is usually not given the disconnect with reality, disturbed functioning, lost resources, and damaged interpersonal relationships that frequently arise from behavior displayed during this state. However, in all but the most extreme manic states there resides adaptive potential, at least in select contexts. For example, in a completely disinhibited, although still coherent state, a male might seduce a mate or mates not attainable

otherwise, or successfully attack a high-ranking individual in the social group to secure privileged resources. With delusions of grandeur a person in a full-blown manic state might attract followers, such as some fringe religion leaders have been known to do, and acquire their resources including mating opportunities. As very clearly seen in clinical practice, though, the full-blown manic state involves disorganized thinking and behavior that reduces adaptive functioning, such as a person acting so belligerent that potential mates stay well clear. Furthermore, delusions of grandeur usually shift to delusions of persecution giving rise to intense fear, further impairing functioning.

If hypomania typically progressed to mania the value of this defense in overriding depressive inhibition would be greatly diminished. Akiskal estimates (1996) that 3–6% of the general population worldwide has hypomania or cyclothymia giving a 1:1 unipolar–bipolar ratio. Subthreshold hypomanic symptoms conceivable might be even more commonplace. Mania is much less common and is estimated to occur in about 1% of the population (Weissman and Myers, 1978). BP-II likely accounts for at least 50% of all depressions observed in clinical practice (Akiskal, 2005). The greater prevalence of hypomania than mania and it being roughly on par with depression, supports its role as a defense against depressive inhibition. Most of those with hypomania will not develop a manic episode and hence will demonstrate predominately adaptive behavior. Even when there are manic episodes 50–60% will also experience milder hypomanic episodes (Cassano et al., 1992; Coryell et al., 1989). Studying an adolescent bipolar group Klein et al. (1996) found that most remained stable with hypomania and did not progress to mania. Amongst cyclothymics only 6% will develop mania (Akiskal, 1996).

While it is difficult to say why some individuals develop mania, factors such as genetic vulnerability to psychosis and perhaps mania itself, excessive alcohol and drug use common in bipolar disorders (Akiskal et al., 2003), antidepressant-induction, and stress might all play a role, particularly if mania is not preceded by hypomania. It is also feasible that when hypomania fails to override depressive inhibition the physical and mental over-activity will intensify to compensate. At manic levels the volitional inhibition is overridden but at a substantial cost in terms of compromised functioning. Ideally there would be a feedback mechanism, whereby when the costs of defense intensification exceed the value of overriding depressive inhibition, further intensification of the defense ceases. In some individuals with bipolar disorder this feedback mechanism might well operate preventing progression to mania whereas in others it does not work. Conceivably, the presence of this feedback capacity might even distinguish BPII from BPI. Antidepressants, alcohol, and illicit drugs probably impair it, increasing the likelihood of hypomania shifting into mania.

7. Conclusion

Depressive inhibition, comprised to some extent of low behavioral activation and high behavioral inhibition, would have greatly reduced fitness in an evolutionary context. Hypomania provided a short-term override capacity effectively restoring adaptive physical and mental activity. Operating as a defense mechanism, hypomania is activated in response to a depressive episode, or proactively to short-circuit the development of one. In the latter case, there will likely be a prior history of depression and early signals of it might trigger hypomania. The ultimate proactive version of this defense consists of hyperthymic temperament providing physical and mental over-activity on an ongoing basis. The physical and mental over-activity inherent in hypomania increases productivity, creativity, sensitivity, sexual functioning, and social outgoingness and ease. When social anxiety is involved in a depressive episode, hypomania overrides social inhibitions to produce for the most part socially adaptive functioning.

Beyond providing a conceptual framework for understanding the origins of hypomania and mania, this evolutionary based perspective suggests that hypomania is normal and adaptive in response to a depressive episode, or even proactively to prevent one from unfolding. Hypomania might best be viewed as a natural treatment for depressive inhibition as opposed to a problem to treat. Jamison et al. (1980) note that some patients discontinue lithium when depressed or facing problems to induce a hypomanic-manic state. Based on the premise that the active change mechanism in cognitive-behavioral treatments is the behavior component, a specific type of therapy—Behavioral Activation Treatment for Depression (BATD)—focuses on increasing patient activity and access to reinforcement (Hopko et al., 2003; Lejuez et al., 2001). Research indicates that adding automatic thought modification to behavior activation does not improve outcomes, and that behavioral activation therapy is just as effective as cognitive therapy for altering negative thinking and dysfunctional attitudes (Jacobson et al., 1996). Over a 24-month follow-up period combined therapy has been shown to be no more effective at preventing relapse than behavioral activation therapy alone (Gortner et al., 1998). For more severely depressed patients behavioral activation therapy is comparable to antidepressants, with both more effective than cognitive therapy (Dimidjian et al., 2006).

By simply encouraging exercise, involvement in constructive activities, and social participation clinicians might help foster sub-threshold hypomanic or actual hypomanic behavior. Considering that hypomania typically does not progress to mania, there is a very limited downside and a tremendous adaptive aspect. Even far removed from our hunting-gathering evolutionary context, the physical and mental over-activity provided by hypomania can be highly beneficial, reducing or eliminating the volitional inhibition inherent in a depressive episode. With maximal improvement in areas suffering the most from depressive inhibition, and enhanced social performance in many instances, hypomania is truly a leading edge reactive and proactive defense.

Role of the funding sources
Nothing declared.

Conflict of Interest
No conflict declared.

References

Akiskal, H.S., 1995. Toward a temperament-based approach to depression: implications for neurobiologic research. Adv. Biochem. Psychopharmacol. 49, 99–112.

Akiskal, H.S., 1996. The prevalent clinical spectrum of bipolar disorders: beyond DSM-IV. J. Clin. Psychopharmacol. 16, 4S–15S.

Akiskal, H.S., 2005. Searching for behavioral indicators of bipolar II in patients presenting with major depressive episodes: the "red sign," the "rule of three" and other biographic signs of temperamental extravagance, activation and hypomania. J. Affect. Disord. 84, 279–290.

Akiskal, H.S., Pinto, O., 1999. The evolving bipolar spectrum: prototypes I, II, III, and IV. Psychiatr. Clin. North Am. 22, 517–534.

Akiskal, K.K., Akiskal, H.S., 2005. The theoretical underpinnings of affective temperaments: implications for evolutionary foundations of bipolar disorder and human nature. J. Affect. Disord. 85, 231–239.

Akiskal, H.S., Khani, M.K., Scott-Strauss, A., 1979. Cyclothymic temperamental disorders. Psychiatr. Clin. North Am. 2, 527–554.

Akiskal, H.S., Masser, J.D., Zeller, P.J., Endicott, J., Coryell, W., Keller, M., Warshaw, M., Clayton, P., Goodwin, F., 1995. Switching from 'unipolar' to bipolar II: an 11-year prospective study of clinical and temperamental predictors in 559 patients. Arch. Gen. Psychiatry 52, 114–123.

Akiskal, H.S., Hantouche, E.G., Bourgeois, M.L., Azorin, J.M., Sechter, D., Allilaire, J.F., Lancrenon, S., Fraud, J.P., Liliane, C.D., 1998. Gender, temperament, and the clinical picture in dysphoric mixed mania: findings from a French national study (EPIMAN). J. Affect. Disord. 50, 175–186.

Akiskal, H.S., Hantouche, E.G., Allilaire, J.F., Sechter, D., Bourgeois, M.L., Azorin, J.M., Chatenet-Duchene, L., Lancrenon, S., 2003. Validating antidepressant-associated hypomania (bipolar III): a systematic comparison with spontaneous hypomania (bipolar II). J. Affect. Disord. 73, 65–74.

Angst, J., Marneros, A., 2001. Bipolarity from ancient to modern times: conception, birth and rebirth. J. Affect. Disord. 67, 3–19.

Bauer, M.S., Whybrow, P.C., Gyulai, L., Gonnel, J., Yeh, H.S., 1994. Testing definitions of dysphoric mania and hypomania: prevalence, clinical characteristics and inter-episode stability. J. Affect. Disord. 32, 201–211.

Benazzi, F., 2004. Factor Structure of Recalled DSM-IV Hypomanic symptoms of bipolar disorder. Compr. Psychiatry 45, 441–446.

Benazzi, F., 2006. Impact of temperamental mood lability on depressive mixed state. Psychopathology 39, 19–24.

Benazzi, F., 2007. Is overactivity the core feature of hypomania in bipolar II disorder? Psychopathology 40, 54–60.

Benazzi, F., Akiskal, H.S., 2001. Delineating bipolar II mixed states in the Ravenna-San Diego collaborative study: the relative prevalence and diagnostic significance of hypomanic features during major depressive episodes. J. Affect. Disord. 67, 115–122.

Benazzi, F., Akiskal, H.S., 2003. The dual factor structure of self-rated MDQ hypomania: energized-activity versus irritable-thought racing. J. Affect. Disord. 73, 59–64.

Biederman, J., Hirshfeld-Becker, D.R., Rosenbaum, J.F., Herot, C., Friedman, D., Snidman, N., Kagan, J., Faraone, S.V., 2001. Further evidence of association between behavioral inhibition and social anxiety in children. Am. J. Psychiatry 158, 1673–1679.

Bowins, B.E., 2004. Psychological defense mechanisms: a new perspective. Am. J. Psychoanal. 64, 1–26.

Bowins, B.E., 2006. How psychiatric treatments can enhance psychological defense mechanisms. Am. J. Psychoanal. 66, 173–194.

Calvin, W., 1994. The emergence of intelligence. Scientific American, pp. 101–107. Oct.

Campbell-Sills, L., Liverant, G.I., Brown, T.A., 2004. Psychometric evaluation of the behavioral inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. Psychol. Assess. 16, 244–254.

Carlson, G.A., Goodwin, F.K., 1973. The stages of mania: a longitudinal analysis of the manic episode. Arch. Gen. Psychiatry 28, 221–228.

Cassano, G.B., Akiskal, H.S., Savino, M., Musetti, L., Perugi, G., 1992. Proposed subtypes of bipolar II and related disorders: with hypomanic episodes (or cyclothymia) and with hyperthymic temperament. J. Affect. Disord. 26, 127–140.

Cassidy, F., Murry, E., Forest, K., Carroll, B.J., 1998. Signs and symptoms of mania in pure and mixed episodes. J. Affect. Disord. 50, 187–201.

Clore, G., Ortony, A., 2000. Cognition in emotion: Always, sometimes, or never? Cognitive Neuroscience of Emotion. Oxford University Press, New York, N.Y.

Cochran, G., Hardy, J., Harpending, H., 2006. Natural history of Ashkenazi intelligence. J. Biosoc. Sci. 38, 659–693.

Coryell, W., Endicott, J., Keller, M., Andreasen, N., Grove, W., Hirshfeld, R.M., Scheftner, W., 1989. Bipolar affective disorder and high achievement: a familial association. Am. J. Psychiatry 146, 983–988.

Cosmides, L., Tooby, J., 2000. Evolutionary psychology and the emotions. In: Lewis, M., Haviland-Jones, J. (Eds.), Handbook of Emotions (2nd Ed.). The Guilford Press, New York, N.Y., pp. 91–115.

Davis, C., Levitan, R., 2005. Seasonality and seasonal affective disorder (SAD): an evolutionary viewpoint tied to energy conservation and reproductive cycles. J. Affect. Disord. 87, 3–10.

Dimidjian, S., Hollon, S.D., Dobson, K.S., Schmaling, K.B., Kohlenberg, R.J., Addis, M.E., Gallop, R., McGlinchey, J.B., Markley, D.K., Gollan, J.K., Atkins, D.C., Dunner, D.L., Jacobson, N.S., 2006. Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. J. Consult. Clin. Psychol. 74, 658–670.

Draycott, S., Dabbs, A., 1998. Cognitive dissonance 1: an overview of the literature and its integration into theory and practice in clinical psychology. Br. J. Clin. Psychol. 37, 341–353.

Dufour, H., 1978. Depressive inhibition. Encephale 4, 431–471.
Eckblad, M., Chapman, L.J., 1986. Development and validation of a scale for hypomanic personality. J. Abnorm. Psychology 95, 214–222.
Fava, M., Rankin, M.A., Wright, E.C., 2000. Anxiety disorders in major depression. Compr. Psychiatry 41, 97–102.
Festinger, L., 1957. A Theory of Cognitive Dissonance. Stanford University Press, Stanford, California.
First, M.B., Frances, A., Pincus, H.A., 2002. DSM-IV-TR Handbook of Differential Diagnosis. American Psychiatric Publishing Inc., Washington, D.C.
Fowles, D.C., 1988. Psychophysiology and psychopathology: a motivational approach. Psychophysiology 25, 373–391.
Freeman, M.P., McElroy, S.L., 1999. Clinical picture and etiologic models of mixed states. Psychiatr. Clin. North Am. 22, 535–546.
Gardner, R., 1982. Mechanisms in manic-depressive disorder. Arch. Gen. Psychiatry 39, 1436–1441.
Gladstone, G.L., Parker, G.B., 2006. Is behavioral inhibition a risk factor for depression? J. Affect. Disord. 95, 85–94.
Gortner, E.T., Gollan, J.K., Dobson, K.S., Jacobson, N.S., 1998. Cognitive-behavioral treatment for depression: relapse prevention. J. Consult. Clin. Psychol. 66, 377–384.
Gray, J.A., 1987. Perspectives on anxiety and impulsivity: a commentary. J. Res. Pers. 21, 493–509.
Hayward, C., Killen, J.D., Kraemer, H.C., Taylor, C.B., 1998. Linking self-reported childhood behavioral inhibition to adolescent social phobia. J. Am. Acad. Child Adolesc. Psych. 37, 1308–1316.
Hebb, D., 1949. The Organization of Human Behavior. Wiley, New York, N.Y.
Henneberg, M., 1998. Evolution of the human brain: is bigger better? Clin. Exp. Pharmacol. Physiol. 25, 745–749.
Hess, U., Kirouac, G., 2000. Emotion expression in groups. In: Lewis, M., Haviland-Jones, J. (Eds.), Handbook of Emotions (2nd Ed.). The Guilford Press, New York, N.Y., pp. 368–381.
Himmelhoch, J.M., 1998. Social anxiety, hypomania and the bipolar spectrum: data, theory and clinical issues. J. Affect. Disord. 50, 203–213.
Hopko, D.R., Lejuez, C.W., Ruggiero, K.J., Eifert, G.H., 2003. Contemporary behavioral activation treatments for depression: procedures, principles, and progress. Clin. Psychol. Rev. 23, 699–717.
Jacobson, N.S., Dobson, K.S., Truax, P.A., Addis, M.E., Koerner, K., Gollan, J.K., Gortner, E., Prince, S.E., 1996. A component analysis of cognitive-behavioral treatment for depression. J. Consult. Clin. Psychol. 64, 295–304.
Jamison, K.R., Gerner, R.H., Hammen, C., Padesky, C., 1980. Clouds and silver linings: positive experiences associated with primary affective disorders. Am. J. Psychiatry 137, 198–202.
Johnson, S.L., Sandrow, D., Meyer, B., Winters, R., Miller, I., Solomon, D., Keitner, G., 2000. Increases in manic symptoms after life events involving goal attainment. J. Abnorm. Psychology 109, 721–727.
Kasch, K.L., Rottenberg, J., Arnow, B.A., Gotlib, I.H., 2002. Behavioral activation and inhibition systems and the severity and course of depression. J. Abnorm. Psychology 111, 589–597.
Klein, D.N., Lewinsohn, P.M., Seeley, J.R., 1996. Hypomanic personality traits in a community sample of adolescents. J. Affect. Disord. 38, 135–143.
Koukopoulos, A., Sani, G., Koukopoulos, A.E., Minnai, G.P., Girardi, P., Pani, L., Albert, M.J., Reginaldi, D., 2003. Duration and stability of the rapid-cycling course: a long-term personal follow-up of 109 patients. J. Affect. Disord. 73, 75–85.
Lang, R., 1996. Mental Darwinism and the evolution of the emotion-processing mind. Am. J. Psychother. 50, 103–124.
Lazarus, R., 1984. On the primacy of cognition. Am. Psychol. 39, 124–129.
Lazarus, R., 1991. Cognition and emotion in motivation. Am. Psychol. 46, 352–367.
Lejuez, C.W., Hopko, D.R., Hopko, S.D., 2001. A brief behavioral activation treatment for depression. Behav. Modif. 25, 255–286.
Marneros, A., 2001. Origin and development of concepts of bipolar mixed states. J. Affect. Disord. 67, 229–240.
McCrae, R., Costa, P., 1999. A five-factor theory of personality. In: Pervin, L., John, O. (Eds.), Handbook of Personality (2nd Ed.). The Guilford Press, New York, N.Y., pp. 139–153.
McElroy, S.L., Strakowski, S.M., Keck, P.E., Tugrul, K.L., West, S.A., 1995. Differences and similarities in mixed and pure mania. Compr. Psychiatry 36, 187–194.
McFarland, B.R., Shankman, S.A., Tenke, C.E., Bruder, G.E., Klein, D.N., 2006. Behavioral activation system deficits predict the six-month course of depression. J. Affect. Disord. 91, 229–234.
Meyer, B., Johnson, S.L., Carver, C.S., 1999. Exploring behavioral activation and inhibition sensitivities among college students at risk for bipolar spectrum symptomatology. J. Psychopathol. Behav. Assess. 21, 275–292.
Meyer, B., Johnson, S.L., Winters, R., 2001. Responsiveness to threat and incentive in bipolar disorder: relations of the BIS/BAS scales with symptoms. J. Psychopathol. Behav. Assess. 23, 133–143.
Mick, M.A., Telch, M.J., 1998. Social anxiety and history of behavioral inhibition in young adults. J. Anxiety Disord. 12, 1–20.
Mineka, S., Watson, D., Clark, L.A., 1998. Comorbidity of anxiety and unipolar mood disorders. Annu. Rev. Psychol. 49, 377–412.
Morwood, M., Sutikna, T., Roberts, R., 2005. World of the little people. National Geographic, pp. 2–15. April.
Nesse, R., 1998. Emotional disorders in evolutionary perspective. Br. J. Med. Psychol. 71, 397–415.
Perugi, G., Toni, C., Akiskal, H.S., 1999. Anxious-bipolar comorbidity diagnostic and treatment challenges. Psychiatr. Clin. North Am. 22, 565–583.
Placidi, G.F., Signoretta, S., Liguori, A., Gervasi, R., Maremmani, I., Akiskal, H.S., 1998. The semi-structured affective temperament interview (TEMPS-I): reliability and psychometric properties in 1010 14–26 year-old students. J. Affect. Disord. 47, 1–10.
Previc, F.H., 1999. Dopamine and the origins of human intelligence. Brain Cogn 41, 299–350.
Price, J., 1967. Hypothesis: the dominance hierarchy and the evolution of mental illness. Lancet 2, 243–246.
Schneier, F.R., Blanco, C., Antia, S.X., Liebowitz, M.R., 2002. The social anxiety spectrum. Psychiatr. Clin. North Am. 25, 757–774.
Scholten, M.R., van Honk, J., Aleman, A., Kahn, R.S., 2006. Behavioral Inhibition system (BIS) and the Behavioral Activation System (BAS) and schizophrenia: relationship with psychopathology and physiology. J. Psychiatr. Res. 40, 638–645.
Sitaram, N., Gillin, J., Bunney, W., 1978. The switch process in manic-depressive illness: circadian variation in time of switch and sleep and manic ratings before and after switch. Acta Psychiatr. Scand. 58, 267–278.
Vaillant, G., 1977. Adaptation To Life. Little Brown & Company, Boston.
Weissman, M.M., Myers, J.K., 1978. Affective disorders in a U.S. urban community. Arch. Gen. Psychiatry 35, 1304–1311.
Wicki, E.W., Angst, J., 1991. The Zurich study: hypomania in a 28- to 30-year-old cohort. Eur. Arch. Psychiatry Clin. Neurosci. 240, 339–348.