The social zeitgeber theory, circadian rhythms, and mood disorders:
Review and evaluation

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Abstract

The social zeitgeber theory [Ehlers, C. L., Frank, E., & Kupfer, D. J. (1988). Social zeitgebers and biological rhythms. Archives of General Psychiatry, 45, 948–952] offers an explanation of how life events trigger depressive episodes. According to this theory, life stress leads to mood episodes by causing disruptions in individuals’ social routines and, in turn, their biological circadian rhythms. In this article, we review the literature pertaining to the social zeitgeber theory, as well as evidence that this theory may be applied to (hypo)manic episodes. Given the limited data supporting the social zeitgeber theory to date, we also evaluate whether circadian rhythm disruptions are triggered by an internal mechanism, such as an abnormality in one’s pacemaker (the suprachiasmatic nucleus; SCN). We review these two theories in an attempt to understand the potential causes of circadian rhythm disruptions and affective episodes in individuals with unipolar and bipolar disorders. We also propose several areas of future research.

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1. Introduction

Ehlers, Frank, and Kupfer (1988) proposed that depressive episodes arise as a consequence of life events disturbing social zeitgebers (external cues that function to entrain biological rhythms) which, in turn, derail social and biological rhythms. According to this theory, disruptions in these rhythms influence somatic symptoms (e.g., sleep propensity) that in vulnerable individuals leads to a major depressive episode (see Fig. 1). This theory was, in part, derived from the substantial evidence that depressed individuals have irregular biological rhythms, such as sleep–wake cycles, temperature, melatonin, and cortisol rhythms (Howland & Thase, 1999). Recent evidence suggests that the social zeitgeber theory may also explain (hypo)manic episodes of individuals with bipolar disorder (Frank et al., 2005; Malkoff-Schwartz et al., 2000, 1998).

In this review, we elaborate on the associations proposed by the social zeitgeber theory, including its relevance to hypo(manic) episodes. Second, we focus on possible causes of circadian rhythm disruptions in unipolar and bipolar individuals. Consistent with the social zeitgeber theory, we will review studies suggesting that life events (external triggers) may trigger circadian rhythm disruptions and, consequently, affective episodes in vulnerable individuals (see Fig. 1). Alternatively, we will also discuss studies that suggest these circadian rhythm disruptions are due to a stable, trait-like, dysfunction in vulnerable individuals. We suggest that this stable dysfunction may be a biological abnormality in unipolar and bipolar individuals’ pacemakers. For reasons of parsimony, we term this theory the “internal trigger” theory, referring to an abnormality within the body, such as a genetic mutation. The “internal trigger” hypothesis is contrasted with the social zeitgeber theory, an “external trigger” hypothesis (see Fig. 1). In this review, we use the social zeitgeber theory interchangeably with the external trigger hypothesis.

Evidence for each proposed causal link of the social zeitgeber theory, or the five associations numbered in Fig. 1, is discussed in turn. First, we review the association of life events, social zeitgebers and rhythms, and mood (see Fig. 1). Next, we discuss the association of social and biological rhythm disruptions (pathway 3 in Fig. 1). We then review the

![Diagram](image-url)
association of biological rhythm disruptions and mood (pathways 4 and 5 in Fig. 1). In each section, we specifically discuss how the findings pertain to the internal or external trigger hypotheses, or both. We begin by providing necessary background on the definition and assessment of mood disorders, life events, social zeitgebers, social rhythms, and biological rhythms in this literature.

1.1. Mood disorders

A major depressive episode is diagnosed when an individual experiences five or more depressive symptoms for two weeks (for symptoms see Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition-Text Revision (DSM IV-TR), American Psychiatric Association [APA], 2000). It is also possible that individuals may experience less severe episodes, or minor depressive episodes, which require fewer symptoms as well as a shorter duration and less persistence of these symptoms (see Research Diagnostic Criteria; Spitzer, Endicott, & Robins, 1978). The majority of the studies reviewed only included individuals who had experienced major depressive episodes in their study groups; however, some studies did not make a distinction between participants who experienced minor or major depression. We will make this distinction whenever possible in reviewing the studies, as some studies did find differences between these subtypes (e.g., Brown et al., 1996). We also distinguish between individuals with clinical depression (e.g., individuals who have experienced a major or minor depressive episode) and individuals who have experienced only depressive symptoms. In short, we categorize individuals who have experienced a major or minor depressive episode (but not a manic or hypomanic episode) as unipolar depressed.

Individuals with bipolar disorder experience hypomanic or manic episodes as well as depressive episodes (although a depressive episode is not necessary for a bipolar diagnosis). Individuals diagnosed with bipolar I disorder experience at least one manic episode (and typically, at least one major depressive episode as well). Individuals diagnosed with bipolar II disorder experience at least one hypomanic episode, which is less severe and persistent than mania (see APA, 2000). In this review, bipolar disorder refers to individuals with bipolar I disorder unless otherwise specified.

Most studies utilize the Structured Clinical Interview for DSM version III-R (SCID; Spitzer, Williams, Gibbon, & First, 1990) or the Schedule for Affective Disorders and Schizophrenia-Lifetime version (SADS-L; Endicott & Spitzer, 1978) to diagnosis individuals with bipolar or unipolar disorder. Several studies have examined the reliability and validity of the SCID diagnoses (Segal, Kabacoif, Hersen, Van Hasselt, & Ryan, 1995; Skre, Onstad, Torgerson, & Kringlen, 1991; Williams et al., 1992; Zanarini & Frankenburg, 2001). In general, the kappas from these studies for diagnoses range from 0.5 to 1.0. In regards to its validity, several studies have used the SCID as the “gold standard” in determining the accuracy of clinical diagnoses (e.g., Shear et al., 2000; Steiner, Tebes, Sledge, & Walker, 1995). Studies also have found high reliability coefficients for the SADS-L interview, particularly for current and past diagnoses of mood disorders (Alloy & Abramson, 1999; Alloy et al., 2000; Hammen, 1991; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999).

1.2. Life events

Dohrenwend and Dohrenwend (1981) suggested that stressful life events are “those that are proximate to, rather than remote from, the onset of the disorder. For example, this category includes the recent death of a friend or relative but not the fact that an adult’s father died when he was a child. The latter event is not irrelevant to life stress but is subsumed under personal dispositions, since we assume that an early death of a parent can affect an adult’s behavior only insofar as its impact was internalized (p.131).” These authors make the point that events that occurred awhile ago, or when an individual was a young child, are not considered life events, because it is ‘life stress’ that is most likely incorporated into an individual’s personality style, cognitive vulnerability, or other trait-like characteristics. Thus, the ‘proximity’ of a life event to the onset of a disorder is vital in order to assume its association with the onset, and particularly, as the trigger of a mood episode.

Cobb (1974) and Brown and Harris (1978) pioneered the idea that one’s environment may influence the impact of a stressful event on an affective episode. Specifically, these investigators highlighted the need to understand an individual’s current life situation, the nature of social supports available to the person, personal characteristics, and the attitudes of peers when examining the impact of a life event. In support of such a contextual model, recent research has suggested that social support, personality, and other psychosocial variables may act as third variables in the relationship of life stress and affective disorders (Leskelä et al., 2004; Panzarella, Alloy, & Whitehouse, in press; Paykel, 2001).
Although such potential variables are not a focus of this review, it is important to acknowledge that contextual factors are important in understanding the complicated association of life events and mood episodes, as well as the impact of a life event.

Further, life events are different from chronic stress. Ezquiaga, Gutierrez, and López (1987) define life events as “circumstances punctually situated in time that induce stress and require the individual to use adaptation mechanisms” and chronic stress as “adverse circumstances that act uninterruptedly over a prolonged time (p. 136).” Thus, life events entail a change or readjustment by an individual. For this reason, many of the research designs in this review only examined events that had a moderate or severe impact on the participant.

The assessment of life events has improved substantially from earlier studies. One such improvement is a change from the use of self-report forms, or checklists of life events, to life event interviews conducted by highly trained interviewers based on the contextual model established by Brown and Harris (1978). For example, many studies utilize the Bedford Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1979), or variants of this interview (Alloy & Abramson, 1999; Hammen, Marks, Mayol, & DeMayo, 1985), particularly studies examining social rhythm disruption, life events, and mood (Frank, Anderson, Reynolds, Ritenour, & Kupfer, 1994; Malkoff-Schwartz et al., 1998, 2000). These interview-based assessments are similar in that they utilize the contextual threat methods of Brown and Harris and, thus, elicit objective impact ratings for each event (Brown & Harris, 1978). Further, these life event interviews utilize similar procedures of assessment and possess good psychometric properties (Alloy & Abramson, 1999; Daley et al., 1997; Hammen, 1991; Hammen, Henry, & Daley, 2000; Johnson & Miller, 1997; Malkoff-Schwartz et al., 2000; Simons, Angell, Monroe, & Thase, 1993).

1.3. Social zeitgebers and social rhythms

The term “zeitgeber,” German for “time-giver,” is used to describe environmental, or external, time cues that entrain human circadian rhythms. For example, research has found that in free running environments, circadian rhythms, such as the sleep–wake cycle, have a period of approximately 25 h (Reinberg and Menaker, 1992; Wehr et al., 1985; Weyer, 1975). Thus, circadian rhythms are likely entrained by zeitgebers in our environment in order to yield a period of almost exactly 24 h, even though the inherent, free running cycle is 25 h (Moore-Ede, Czeisler, & Richardson, 1983; Panda, Hogenesch, & Kay, 2002; Weyer, 1979). These data are consistent with the social zeitgeber theory; however, they do not rule out the possibility of an internal trigger as well. Moreover, do changes in zeitgebers cause severe enough disruptions to trigger affective episodes? We will review the evidence relevant to this question in the following sections.

Social zeitgebers are usually derived from social contact with other individuals, but solitary activities can also be social zeitgebers for the circadian clock. For example, many activities may occur alone or with others, such as, commuting to work, having meals, bedtimes, and watching television (Monk, Kupfer, Frank, & Ritenour, 1991). In Ehlers, Kupfer, Frank and Monk (1993) the social zeitgeber theory was amended to include physical zeitgebers in addition to social zeitgebers. For example, light is a physical zeitgeber (or “zeitstörer”) that can entrain and regulate the circadian rhythms of hormonal, metabolic, and physical activity, which has also been shown to have clinical implications for mood disorders (Panda et al., 2002; Weyer, 1979; Wirz-Justice, 2006). In this review, we focus specifically on the effects of social zeitgebers, but it is important to remember that other types of zeitgebers, or zeitstörers, affect one’s circadian rhythms and mood as well.

The Social Rhythm Metric (SRM) is intended to capture an individual’s “social rhythm,” or the frequency with which daily activities are performed and the level of regularity and social contact associated with these activities (Monk, Flaherty, Frank, Hoskinson, & Kupfer, 1990). The measure is completed in the evening before going to bed and tracks the timing of 15 specific and 2 write-in, or individualized, activities. The timing of these activities (i.e., waking up, eating meals) is thought to contribute to the stability of an individual’s daily routine or constitute their social rhythm. If the time an activity occurs on a given day is within 45 min of the average time, it is considered a “hit.” Social rhythm regularity is defined by the number of activities that had three or more hits in one week (possible range = 0–17 activities). Average frequency of activities also often is calculated by averaging the frequencies of all items that had been endorsed as regular (possible range = 3–7 times) (Monk et al., 1991). The SRM was found to be moderately consistent (i.e., $r=0.44, p<0.001$ between SRM scores in weeks 1 and 2) and valid (e.g., participants on vacation have considerably lower SRM scores) in a group of 50 healthy controls (Monk et al., 1990, 1991). Other evidence for the conceptual validity of the SRM is derived from studies that found SRM scores are correlated positively with other
indices of social rhythm stability (Brown et al., 1996; Monk et al., 1991; Monk, Petrie, Hayes, & Kupfer, 1994; Stetler, Dickerson, & Miller, 2004; Szuba, Yager, Guze, Allen, & Baxter, 1992).

Frank and colleagues developed the first standardized assessment of social rhythm disruption (SRD) events based on the LEDS interview (Frank, Malkoff-Schwartz, Sherrill, & Anderson, 1999). These ratings are determined “by a consensus panel and are guided by clearly delineated criteria and a ‘dictionary’ of examples (p. 1008, Malkoff-Schwartz et al., 2000).” The SRD rating scale relies on the degree of disruption in one’s sleep–wake cycle brought about by a life event (Frank, Malkoff-Schwartz et al., 1999; Malkoff-Schwartz et al., 2000, 1998). For example, a high SRD rating would be given to life events that contribute to sleep disruption (e.g., overseas travel) or to a substantial desynchronization in one’s routine that would likely change one’s sleep–wake pattern or promote sleep disruption (e.g., marital separation, ending or beginning a full-time job; Malkoff-Schwartz et al., 2000). One study conducted two reliability assessments of this rating system for social rhythm disruption events and found high inter-rater agreement (kappa = 0.94 and 0.87; Malkoff-Schwartz et al., 2000).

1.4. Biological rhythms

A biological process of the human body that repeats itself approximately every 24 h is considered to have a daily rhythm. Such rhythms are defined as “circadian” if they persist with the same period in the absence of external time cues, or are endogenously driven (Refinetti & Menaker, 1992). Circadian rhythms are described quantitatively as having a “mean,” or the level at which the oscillation takes place, a “period,” or the time to complete one cycle, an “amplitude,” or the distance between the mean and peak of the cycle’s oscillation, and an “acrophase” or time of the peak of the circadian rhythm’s phase (Minors & Waterhouse, 1985).

The suprachiasmatic nucleus (SCN), or internal pacemaker, in the anterior hypothalamus of the brain is suspected of regulating human biological rhythms. The SCN can function autonomously, without the need for external zeitgebers (“free running state”), but it can also be entrained by zeitgebers (“entrained state”) (Cermakian & Boivin, 2003; Wever, 1979). For example, the core of the SCN receives photic input from the retina, which triggers a response initiated by various genes and chromatin remodeling within the SCN neurons. The SCN also receives non-photic input from different parts of the brain (Cermakian & Boivin, 2003). The tightly packed neurons in the SCN are believed to integrate these photic and nonphotic zeitgebers and then produce an output signal that regulates the circadian rhythms in the body. These data suggest that a combination of internal and external triggers may affect individuals’ pacemakers, but it is unclear which source of cues, internal or external, predominantly influences affective episodes. Preliminary evidence in this area will be reviewed.

Studies that attempt to measure the free running, or endogenous, component of the SCN must remove or minimize potential external zeitgebers, such as noise, light, and clocks. These studies are interested in observing as many oscillations, or consecutive cycles, of the circadian rhythms as possible to understand the quantitative aspects of the rhythms. Participants in these studies are isolated from their normal environments for usually 10 to 30 d, although some studies may only isolate participants for a few days, particularly if a sleep manipulation is imposed (Minors & Waterhouse, 1985). Participants’ body temperature, sleep, and activity levels are usually monitored continuously with blood drawn approximately every hour to assess levels of plasma melatonin, cortisol, thyroid stimulating hormone (TSH), and other hormones suspected of having a circadian rhythm.

2. Life events, social rhythms, and mood

According to the social zeitgeber theory, life events begin the causal chain of processes that lead to major depressive episodes (Ehlers et al., 1988). Thus, we begin by very briefly reviewing the extensive evidence that life events are associated with affective episodes and symptomatology. It is important to note that there are several theories, other than the social zeitgeber theory, which also attempt to explain the association between life events and depression. These theories will not be discussed, as they are outside the scope of this review (see Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990; Hammen, 1991; Johnson & Roberts, 1995; Monroe & Handjiyannakis, 2002; Safford, Crossfield, Alloy, & Abramson, in press).

In this section, we will also review findings that may explain how life events influence mood. Specifically, we review the evidence that life events may be associated with mood because they affect social zeitgebers (i.e., meal times, exercise, clocks) (pathway 1 in Fig. 1), which then disrupt an individual’s social rhythm (i.e., daily activities, social
interactions) (pathway 2 in Fig. 1). We will also discuss the limitations of the literature and offer an alternative interpretation of the findings based on the internal trigger hypothesis.

2.1. Life events and mood

The social zeitgeber theory describes a potential pathway by which life stress may influence mood. This theory was based originally on the finding that life events are associated with depressive symptoms (Paykel, 2001), but is also supported by research suggesting that life events are associated with (hypo)manic symptoms (Dunner, Patrick, & Fieve, 1979), as well as precede bipolar episodes or relapse (Glassner & Haldipur, 1983; Joffe, MacDonald, & Kutcher, 1989; Kennedy, Thompson, Stancer, Roy, & Persad, 1983). However, it is important to note that some studies using structured life event interviews (e.g., LEDS) did not find a significant association of life events and (hypo)manic episodes (Chung, Langeluddecke, & Tennant, 1986; Sclare & Creed, 1990). These inconsistent findings in bipolar samples are likely due to these studies relying on long-term recall (e.g., 10 years) of events, using different time frames, utilizing different inclusion criteria, using hospital admission as the criterion for relapse (this is problematic given that events may occur between onset of episode and admission), and having insufficient power to detect meaningful group differences (see Alloy et al., 2005; Johnson, 2005; Johnson & Roberts, 1995).

In the last 15 years, the literature has improved considerably on these limitations and replicated earlier findings that life events are associated with bipolar episodes (Christensen et al., 2003; Ellicott et al., 1990; Hammen & Gitlin, 1997; Hunt, Jones, & Silverstone, 1992; Pardoen et al., 1996). For example, one particularly well-designed study found that life events are associated with the course of bipolar disorder or the time to recovery after a bipolar episode (Johnson & Miller, 1997). In short, extensive evidence suggests that life events precede depressive as well as (hypo)manic symptoms and episodes (Alloy et al., 2005; Johnson, 2005; Johnson & Roberts, 1995).

2.2. Life events, social zeitgebers and rhythms

The first study to examine whether life events are associated with social zeitgebers and rhythm disruption (pathways 1 and 2 in Fig. 1) assessed 87 individuals after the loss of a spouse (Flaherty, Frank, Hoskinson, Richman, & Kupfer, 1987). This study concluded that social rhythm disruptions were associated with the loss of a spouse (a severe negative, social rhythm disruption event), as well as with an increase in depressive symptomatology. In contrast, Prigerson et al. (1994) did not find a relationship between a spouse’s death and social rhythm disruption in an elderly (aged 60 to 80 years) sample. Perhaps these participants’ preservation of regular social rhythms, after the death of their spouse, buffered them from the development of bereavement-related depression. These studies are limited by their correlational design as well as their utilization of a community sample, which increases the generalizability of these results, but does not add to our understanding of how these events affect individuals with bipolar or unipolar mood disorders. Further, Prigerson et al. (1994) had a long time interval (e.g., 9 to 12 months) between the severe life event and assessment of social rhythm disruption and symptomatology. In short, we cannot determine from these studies whether social rhythm disruptions are due to the loss of a zeitgeber, the occurrence of an event, or a biological abnormality (e.g., in one’s pacemaker) in vulnerable individuals (see Fig. 1).

To improve upon these limitations, Brown et al. (1996) conducted a follow-up study to the Prigerson et al. (1994) study in which 43 elderly, spousally bereaved individuals and 15 elderly, nonbereaved individuals were added to their original sample. This study found that individuals currently in a major depressive episode, but not a minor depressive episode, had significantly lower social rhythm stability and activity levels than the nonbereaved controls (Brown et al., 1996). In the major depressive group, social rhythm stability was also inversely related to subjective sleep quality (i.e., sleep impairment increased as stability of social rhythm decreased) and severity of depression (i.e., less social rhythm regularity was associated with more depressive symptoms).

Similar to the early studies, this study had a cross-sectional design and, thus, we cannot determine whether it was the death, or loss of a zeitgeber, that impacted the individuals with major depression (external trigger hypothesis) or whether they had a more trait-like social rhythm irregularity present even prior to the spouse’s death (internal trigger hypothesis). The latter explanation is consistent with findings from the sleep literature that individuals with unipolar and bipolar disorders have disrupted sleep cycles, suggesting that they have a more permanent abnormality in their circadian rhythm pacemakers (Wehr & Wirz-Justice, 1982; Wu & Bunney, 1990). Future studies need to assess the temporal relationship of zeitgebers, circadian rhythm disruptions, and symptoms. However, this study did improve
upon the Prigerson et al. (1994) study by finding an association of social rhythm stability, sleep, and depressive symptoms in individuals experiencing a major depressive episode. Thus, the null findings from the Prigerson et al. (1994) study may be explained by their lumping of individuals with varying levels of vulnerability (i.e., intensity of depressive symptoms).

Two other studies have examined the association of life events and social rhythm regularity in a clinical sample (Malkoff-Schwartz et al., 2000, 1998). These studies utilized the LEDS to assess life events. As described above, the level of social rhythm disruption (SRD) of events was rated by a consensus panel and ranged from 1 (marked) to 4 (little/no disruption) (Frank, Malkoff-Schwartz et al., 1999). An event was considered disruptive if it received a score of 1, 2, or 3 (Malkoff-Schwartz et al., 1998). These studies primarily focused on the association of events and episode onsets; however, they also recognized that life events are associated with SRD (pathways 1 and 2 in Fig. 1) (Malkoff-Schwartz et al., 1998).

Malkoff-Schwartz et al. (1998) found that twice as many bipolar participants experienced a SRD event during an eight week pre-onset period compared to an eight week control period. Further, the proportion of participants with at least one SRD event was significantly greater during the pre-onset than control periods for manic participants, but not for depressed participants (55% versus 10%, respectively). Between-group comparisons illustrated that the association of SRD events and episode onsets was unique to manic episodes. It is possible that the eight week window in this study may have been ample time for SRD to trigger mania, but not depression (Malkoff-Schwartz et al., 1998). Alternatively, the group differences could be attributed to the small sample sizes or to manic participants experiencing more than twice as many total events than the depressed participants (81% and 38%, respectively), allowing for a greater chance for these events to be associated with SRD (Malkoff-Schwartz et al., 1998). Finally, it is possible that SRD events do not impact depressive symptoms as much because depression is manifested as a result of a more trait-like abnormality in the pacemaker (i.e., internal trigger hypothesis), whereas manic symptoms may be associated more with disruptions in zeitgebers (as proposed by the social zeitgeber theory) (see Fig. 1).

To examine these theories, the same authors conducted a similar study, but with 8- and 20-week pre-onset periods and episode-free control periods from the year prior to each participant’s mood episode onset (Malkoff-Schwartz et al., 2000). Participants with bipolar and unipolar mood disorders were divided into four groups: bipolar manic ($n=21$), bipolar depressed ($n=21$), bipolar rapid cycling ($n=24$), and unipolar depressed ($n=44$). Between-group comparisons illustrated that bipolar manic participants experienced more SRD events in the pre-onset periods than their control periods compared to the other three groups. The only significant within-subject comparison, as also found in the previous study, was that the manic participants experienced more SRD events in the 8-week pre-onset period compared to the 8-week control period. This supports Malkoff-Schwartz et al.’s (1998) earlier presumption that bipolar manic participants are especially susceptible to SRD events, particularly given the added comparison groups (i.e., unipolar depressed, bipolar cycling). Additionally, the null findings for the 20-week pre-onset period suggest that there were no delayed effects of SRD events on depressive episodes. Thus, bipolar individuals experiencing manic symptoms appear to be influenced by zeitgebers; although, it is still unclear whether these individuals also have trait-like abnormalities in their pacemakers as suggested by disruptions in their sleep (Sack, Nurnberger, Rosenthal, Ashburn, & Wehr, 1985; Wehr, Wirz-Justice, Goodwin, Duncan, & Gitlin, 1979) and the internal trigger hypothesis (see Fig. 1).

2.3. Summary

Research suggests that life events precede the onset of unipolar and bipolar episodes. Further, consistent with the social zeitgeber theory, there is some evidence that life events are associated with social rhythm disruptions which, in turn, may trigger affective symptoms (Malkoff-Schwartz et al., 2000, 1998). For example, life events that disrupt individuals’ social rhythms are significantly associated with onsets of manic episodes (Malkoff-Schwartz et al., 2000, 1998). However, this conclusion must be considered preliminary, given that these studies have small sample sizes and lack a non-psychiatric control group.

It is unclear from these studies whether social rhythm disruptions preceded depressive symptoms (Brown et al., 1996; Flaherty et al., 1987). Moreover, several studies did not find an association between depressive symptoms and socially disruptive events (Malkoff-Schwartz et al., 2000, 1998; Prigerson et al., 1994). The lack of an association between depressive symptoms and SRD events may suggest that individuals with mood disorders, particularly major depression, have trait-like social rhythm irregularities that contribute to their symptoms as suggested by the internal trigger theory (see Fig. 1) as opposed to disruptions due to changes in zeitgebers. Given the limitations of these
social and biological rhythms (pathways 2 and 3 in Fig. 1). According to the social zeitgeber theory, depressive symptoms are the result of changes in social zeitgebers disrupting that serve to entrain biological rhythms, such as temperature, hormone levels, and sleep (Monk et al., 1990, 1991). Thus, social zeitgebers, such as personal relationships, meals, exercise, or social demands (i.e., school, work) are tasks that serve to entrain biological rhythms, such as temperature, hormone levels, and sleep (Monk et al., 1990, 1991). According to the social zeitgeber theory, depressive symptoms are the result of changes in social zeitgebers disrupting social and biological rhythms (pathways 2 and 3 in Fig. 1).

3. Social zeitgebers and biological rhythms

Ehlers et al. (1988) proposed that the “primary path” of the social zeitgeber theory consisted of a “chain of events in which instability of social rhythms can lead to an instability in specific biological rhythms, particularly sleep (p. 950).” Thus, social zeitgebers, such as personal relationships, meals, exercise, or social demands (i.e., school, work) are tasks that serve to entrain biological rhythms, such as temperature, hormone levels, and sleep (Monk et al., 1990, 1991).

There has been very little research on the association of social zeitgebers and biological rhythms in patient populations (pathway 3 in Fig. 1); however, researchers have acknowledged for centuries that healthy humans are able to adapt their circadian rhythms to environmental zeitgebers (Elmore, Betrus, & Burr, 1994). For example, researchers have identified the entrainment effects of the light-dark cycle, social contact/interaction, time of meals, and ambient temperature on human circadian rhythms (Aschoff et al., 1971; Eastman, 1987; Sack, Lewy, Blood, Keith, & Nakagwa, 1992; Wever, 1989). More specifically, Aschoff et al. (1971) were among the first investigators to identify that social zeitgebers (i.e., meals, sleep propensity) can entrain the circadian rhythms of body temperature, cortisol, catecholamines, and sodium in individuals under constant darkness. Moreover, two small, but well designed studies found that the presence of others in a group was responsible for a significant phase shift in these rhythms and that the rate of cortisol phase shift was heavily influenced by social interactions (Vernikos-Danellis & Winget, 1979; Winget, Vernikos-Danellis, & Beljan, 1975).

More recently, a review concluded that changes in daily activity patterns and social contacts, whether natural or brought about by an experimental manipulation, a religious observance, or long-term travel, are associated with changes in biological rhythms (Stetler et al., 2004). Thus, preliminary research in community samples suggests that zeitgebers influence circadian rhythms (as suggested by the social zeitgeber theory); however, these studies are limited in that they do not utilize bipolar or unipolar samples, have small sample sizes, and do not ascertain the directionality of these relationships. It is unclear from these studies whether changes in external cues disrupt circadian rhythms to the extent that they may cause affective symptoms.

Stetler et al. (2004) examined social rhythm regularity and biological rhythms, specifically daily cortisol levels, in 50 unipolar depressed participants who were matched on age, sex, and ethnicity to 50 non-depressed, control participants. Similar to previous studies, this study found that control participants exhibited a decline in cortisol levels on days in which they performed more regular activities (Aschoff et al., 1971; Vernikos-Danellis & Winget, 1979; Winget et al., 1975). This relationship was not found for participants with unipolar depression (Stetler et al., 2004). Further, control participants had a significantly greater normative decline in cortisol levels on days in which they performed more regular activities with other people than on days in which they engaged in fewer regular activities with other people. The unipolar depressed group exhibited a similar, but nonsignificant, trend for regular activities conducted with others. Thus, regular activities carried out when other people are present may be an important factor in whether these activities influence the circadian rhythm of cortisol. This conclusion is supported by the finding that nonsocial, regular activities were not related to diurnal cortisol patterns in either group (Stetler et al., 2004).

This study is consistent with previous findings that healthy individuals’ social zeitgebers and rhythms are associated with their biological rhythms. The lack of such an association in unipolar depressed individuals may be due to the inability of social zeitgebers to entrain their biological rhythms. It is possible that a biological abnormality in depressed individuals’ pacemakers may make it difficult for them to entrain to their social environments, perhaps explaining the observed desynchrony of circadian rhythms in depressed individuals (Wehr & Wirz-Justice, 1982; Wu & Bunney,
This is supported by the finding that unipolar depressed individuals in Stetler et al.’s (2004) study experienced the same number of social zeitgebers as the normal controls. The absence of a social zeitgeber–circadian rhythm association in unipolar depressed individuals in the Stetler et al.’s (2004) study contradicts the social zeitgeber theory and some of the evidence previously reviewed illustrating that external zeitgebers (i.e., life events) do influence individuals’ lifestyle or social regularity. Specifically, the social zeitgeber theory suggests that depressed individuals’ social zeitgebers have the ability to entrain their biological rhythms. According to this theory, it is the irregularity of social zeitgebers (i.e., by loss or change in zeitgebers) that leads to a disruption in biological rhythms and eventually depressive symptoms (see Fig. 1) (Ehlers et al., 1988).

Another possible explanation for Stetler et al.’s (2004) findings is that unipolar depressed individuals may require stronger or more frequent social zeitgebers than nondepressed individuals to influence their biological rhythms, due to an abnormality in their circadian pacemakers or SCN. This “attenuation theory” suggests that depressive symptoms may be related to the strength of circadian rhythms, as measured by the amplitude of their phases, as well as the precision of the circadian rhythms (Czeisler, Kronauer, Mooney, Anderson, & Allan, 1987; Schulz & Lund, 1983). Preliminary evidence suggests that individuals with unipolar depression have weaker circadian rhythms compared to normal controls (Vogel, Vogel, McAbee, & Thurmond, 1980). This explanation is congruent with both theories proposed in Fig. 1 (represented by the dotted arrow in Fig. 1); the extent to which vulnerable individuals’ circadian rhythms are disrupted is a function of both (1) the zeitgeber’s strength in entraining the rhythm (the social zeitgeber or external trigger theory) and (2) a biological abnormality of the pacemaker causing it to be less responsive to its environment (the internal trigger theory).

Congruent with the attenuation theory, research has found that unipolar depressed individuals find their social interactions to be less enjoyable and less intimate compared to normal controls (Nezlek, Hampton, & Shean, 2000). Thus, it is possible that a higher frequency or a stronger intensity of social interactions are needed to act as zeitgebers for depressed individuals than are needed by normal controls. In sum, the impact of a zeitgeber on a biological rhythm can be “modulated by protective and vulnerability factors from both the psychosocial and the psychobiological spheres (p. 950, Ehlers et al., 1988),” perhaps a biological abnormality of the pacemaker. Thus, future research should examine the relationship of the type, quantity/intensity, and frequency of social zeitgebers and depression and (hypo)mania, and specifically, how individuals with these symptoms experience or interpret their social interactions.

Unfortunately, given the correlational nature of the Stetler et al. (2004) study, the causal direction of these associations cannot be determined. Thus, it is also possible that changes in cortisol levels could influence regularity and frequency of daily activities (the opposite directionality proposed by the social zeitgeber theory, see Fig. 1). Other limitations to this study include assessment of social rhythm regularity over only four days, in contrast to the typical two to six weeks (Monk et al., 1991). Yet, this study did report SRM scores that were quite stable across four days for both groups (for controls, $\alpha=.72$, for depressed, $\alpha=.84$) (Stetler et al., 2004). This study also only assessed one biological rhythm. Future research should address the relationship of social zeitgebers and other biological rhythms such as body temperature, sleep, and other hormones. Finally, this study was restricted to same-day analyses, given that the data collection days were not consecutive days. Thus, it is impossible to assess possible time lag effects of social rhythm regularity and cortisol levels.

3.1. Summary

There is preliminary evidence that social zeitgebers may influence circadian rhythms in community samples, supporting pathway 3 of the social zeitgeber theory (see Fig. 1). One study did not find such an association between social zeitgebers and the cortisol circadian rhythm in a sample of unipolar depressed individuals (Stetler et al., 2004). Although this finding contradicts the social zeitgeber theory, several possible explanations for these results were discussed. There are currently no studies that have addressed this relationship in a bipolar sample. Thus, future research is greatly needed in this area, particularly to give the social zeitgeber theory credence, as it is a necessary pathway in this model (or pathway 3 in Fig. 1).

Future research should also examine the critical components of social zeitgebers needed to entrain circadian rhythms. Specifically, particular types or intensities of social zeitgebers may be required to influence circadian rhythms as suggested by the attenuation theory. Likewise, as mentioned, an understanding of how the pacemaker (SCN) may interpret zeitgebers is needed. Alternatively, given the lack of evidence that social zeitgebers entrain biological rhythms in individuals with unipolar and bipolar disorder, it should be considered that a biological abnormality in the pacemaker...
is responsible for their symptoms as opposed to disruptions in external cues (see Fig. 1). In short, future work should address whether various characteristics of zeitgebers, such as the degree to which they entail social interactions, differentially impact circadian rhythms. Such research investigating how social zeitgebers influence biological rhythms is needed to gain further support for the external trigger hypothesis.

4. Circadian rhythms and mood

The last pathways of the social zeitgeber theory presume that circadian rhythm disruption creates the somatic symptoms of depression, leading to a major depressive episode in vulnerable individuals (see Fig. 1) (Ehlers et al., 1988). Again, we also review evidence pertinent to (hypo)manic symptoms. First, we review the association of bipolar and unipolar individuals’ social rhythm regularity and mood episodes. Next, we review preliminary evidence from treatment studies that suggests efforts to regularize, or improve social rhythm regularity, are associated with improved mood. The effectiveness of these psychotherapies, although questionable, yields preliminary support for the social zeitgeber theory.

4.1. Social rhythms and mood

The most discouraging results for the social zeitgeber theory are from an early study that found no differences in SRM scores between 20 individuals with unipolar depression and 15 day-working normal controls over a 12-week period (Monk et al., 1990). However, the depressed individuals had more intrasubject variability in their SRM scores than controls and required more weeks of sampling to achieve a stable measure of their social rhythm regularity (Monk et al., 1991). These results suggest that individuals with unipolar depression have more difficulty maintaining social rhythm regularity from day to day; however, this may be a result of either an external force acting on depressed individuals (e.g., life events acting as social rhythm disrupters as postulated by the social zeitgeber theory) or an internal component of the depressed individuals (e.g., cognitive vulnerability, personality style, abnormal circadian pacemaker) (see Fig. 1). Further, these results, as most other studies conducted with patient populations, are limited by the unstandardized medication regimes of participants with mood disorders, given that medications have been shown to affect circadian rhythms (Bendetti et al., 2001).

Given that participants in the Monk et al. (1991) study were in remission, it is possible that the lack of group differences was due to the patient group not being currently depressed. As a result, Szuba et al. (1992) examined the daily SRM scores of individuals with bipolar and unipolar depression\(^1\) \((n=19)\) in an inpatient psychiatric hospital compared to a control group of full-time, day-shift employees of the hospital. In contrast to Monk et al. (1991), this study found that less social rhythm regularity was correlated with a higher report of depressive symptoms and the depressed group had lower overall SRM scores (less regular social rhythms) than the control group (Szuba et al., 1992). Thus, even in highly structured environments, there was enough variability in SRM scores to yield a significant difference between groups. However, this study is limited in that pre-hospitalization social rhythm and activity levels were not assessed. Similar to Monk et al.’s (1991) study, it is unclear from these data why depressed individuals have less regular schedules (i.e., life events, abnormal pacemakers), but it appears that their schedules are associated with their depressive symptoms.

More recently, two other studies have obtained the finding that bipolar individuals have significantly lower social rhythm regularity scores (as measured by the SRM) compared to sex- and age-matched control participants (Ashman et al., 1999; Jones, Hare, & Evershed, 2005). Unfortunately for the social zeitgeber theory, these studies also found that lower social rhythm regularity scores did not vary systematically with mood state; however, a power analysis revealed that these studies did not have enough participants to demonstrate a significant effect (Ashman et al., 1999). Nonetheless, these results suggest that circadian rhythm regularity may be a trait marker (internal trigger theory) as opposed to a consequence of clinical state (external trigger theory) (see Fig. 1). This is supported by post-hoc analyses in the Ashman et al. (1999) study that morning activities (but not evening activities) were significantly phase delayed (i.e., occurred later) during periods of depressed mood compared to (hypo)manic and euthymic mood states, suggesting that there may be a biological abnormality with these individuals’ pacemakers. Such an abnormality may create a trait-
like phase delay of their social rhythms which, in turn, could trigger their symptoms (i.e., internal trigger hypothesis). However, without assessing these individuals’ daily exposure to zeitgebers, we cannot rule out that these seemingly, trait-like social rhythm disruptions could also be due to external events as suggested by the social zeitgeber theory and indicated by the dotted line in Fig. 1.

Congruent with this theory, a recent study found that individuals with bipolar spectrum disorders had lower trait-like social rhythm regularity scores (as defined by a modified version of the SRM) than demographically matched normal controls (Chang, Alloy, & Abramson, 2003). Moreover, further analyses with a larger sample from the same study showed that lower regularity scores prospectively predicted depressive and (hypo)manic episodes (Shen, Alloy, Abramson, & Grandin, submitted for publication). These data are consistent with the theory that individuals with affective disorders have less regular daily routines as a result of a more stable trait, such as an abnormality in their pacemakers. However, zeitgebers may also affect the pacemakers’ rhythm or worsen these individuals’ lifestyle regularity (as indicated by the dotted line in Fig. 1).

4.2. Psychotherapy and mood: Implications for the social zeitgeber theory

Interpersonal and Social Rhythm Therapy (IPSRT) was specifically designed to maintain regular daily rhythms as well as identify and manage potential precipitants of rhythm dysregulation (Frank, Holly, & Kupfer, 2000). The central tenet of IPSRT, similar to the social zeitgeber theory, is that circadian rhythm disruptions are the primary underlying mechanism in bipolar disorder. Thus, IPSRT is presumed to alter the underlying neuronal circuitry that is associated with bipolar symptomatology by regularizing circadian rhythms, particularly the sleep-wake cycle (Frank et al., 2000).

One longitudinal study has addressed the effectiveness of IPSRT for bipolar individuals. Specifically, bipolar participants with an acute depressive or (hypo)manic episode were randomized to either IPSRT (n=18) or to a standard medication clinic treatment condition, Clinical Status and Symptom Review Treatment (CSSRT; n=20) (Frank et al., 1997). Participants completed the SRM everyday and symptom measures every few weeks for up to 52 weeks (M=24.8±13.8 and M=26.7±15.0 for the IPSRT and CSSRT groups, respectively). In the first publication of their findings, the authors reported that the IPSRT group increased their number of regular activities over time faster than the CSSRT group, but the two groups did not differ in their number of bipolar symptoms (Frank et al., 1997). These results did not provide support for the social zeitgeber theory in that increasing bipolar individuals’ social rhythm regularity did not appear to improve their mood.

These findings were replicated in a second publication, as the authors found no difference in the outcomes between four, randomized groups after two years of follow up: (1) acute IPSRT followed by preventative IPSRT, (2) acute IPSRT followed by preventative clinical management (CM), (3) acute CM followed by preventative CM, or (4) acute CM followed by preventative IPSRT (Frank, Swartz et al., 1999). However, the most recent findings from this longitudinal study, indicated that participants treated with IPSRT, as opposed to CM, in the acute phase experienced longer episode-free periods and were more likely to remain well in the two year preventive maintenance phase (Frank et al., 2005). Further, this association was mediated by increased social rhythm regularity for participants receiving IPSRT. These data suggest that social rhythm disruptions may cause affective episodes. These findings provide stronger support for the social zeitgeber theory than for the internal trigger theory, given that stable, trait-like rhythms probably would be less likely to show such improvements or changes over a relatively short time period than rhythms triggered by external events (see Fig. 1).

4.3. Summary

Research is needed to determine whether disruptions in social rhythms are associated with bipolar episodes. Although preliminary findings suggest this association exists, the results are mixed and, thus, require replication. In addition, several of the studies examining this association have been limited by small sample sizes, short assessment

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2 Bipolar spectrum disorders are considered categories of bipolar disorder and include bipolar II, cyclothymic, and bipolar not otherwise specified (NOS) diagnoses. Cyclothymia involves at least two years of numerous hypomanic and depressive episodes that do not meet criteria for mania or major depression and occur without a symptom-free period of two months (APA, 2000). A diagnosis of bipolar NOS entails recurrent hypomanic episodes without depressive episodes, or a cyclothymic pattern but with hypomanic and depressive periods that do not meet minimum duration criteria for hypomanic and depressive episodes, or hypomanic and depressive periods that were too infrequent to qualify for a cyclothymic diagnosis.
periods, heterogeneous patient populations, and not assessing the direction of their findings. Further, the SRM may not adequately capture social rhythm regularity, given that it relies on participants’ ability to accurately assess the timing of activities that they often do habitually. Alternatively, given the high frequency of these activities, participants are likely to be accurate reporters of their timing, which is supported by the sound psychometric properties of the SRM.

It is possible that subtypes of bipolar disorder may respond differently to particular interventions (Jones, 2004). There are very few studies examining the manipulation of social rhythm regularity, but there have been none to date that have attempted such an intervention in bipolar NOS, cyclothymic, or bipolar II individuals. This is particularly important because reduced social rhythm regularity in bipolar spectrum individuals prospectively predicted time to the onset of their (hypo)manic and depressive episodes (Shen et al., submitted for publication). Further, given the preliminary, but promising, findings for IPSRT, future research should address the effects on mood of manipulating social rhythm regularity in a bipolar sample, particularly for individuals with specific subtypes of the disorder.

These findings also offer preliminary evidence that circadian rhythm regularity is trait-like as opposed to a consequence of symptomatology in bipolar samples (Ashman et al., 1999; Chang et al., 2003; Jones et al., 2005). Given that even remitted bipolar individuals are likely to experience symptoms, future research should examine a trait hypothesis in at-risk healthy participants (e.g., offspring of bipolar parents). Another consideration includes examining differences between bipolar and control groups that may confound the interpretation of social rhythm and circadian rhythm differences between the groups. As mentioned, bipolar samples differ from normal controls in their medication regimes. Specifically, lithium, a popular mood stabilizer for bipolar individuals, has been shown to have circadian-stabilizing effects (Bendetti et al., 2001). Further, studies have found lifestyle differences between bipolar samples and control groups that are not accounted for in their study design, such as employment, marital status, and personality characteristics (Jones et al., 2005; Swendsen, Hammen, Heller, & Gitlin, 1995). Future studies should be wary of these individual and group differences and how they may affect the study design and interpretation of results.

Taken together, the studies reviewed in this section illustrate that vulnerable individuals have less social rhythm stability and that this may lead to their affective episodes (Chang et al., 2003; Shen et al., submitted for publication). Further, this research indicates that regularizing social rhythms in bipolar samples could be an effective intervention (Frank et al., 2005).

5. Summary of limitations and directions for future research

This review highlighted several limitations of the social zeitgeber theory, as well as the studies conducted to test this theory to date. A general comment is that there is not enough prospective, longitudinal research, particularly with unipolar and bipolar samples, to fully evaluate the merits of the social zeitgeber theory. At best, this review highlighted several associations that suggest circadian rhythm disruptions may contribute to the symptoms experienced by individuals with bipolar and unipolar mood disorders. It is still unclear whether external cues (the social zeitgeber theory), internal cues (the internal trigger theory), or both influence circadian rhythm disruptions and whether these disruptions are associated with changes in mood (see Fig. 1). These clarifications are necessary to determine the validity of the social zeitgeber theory. Consequently, we briefly highlight specific limitations and suggestions for future research.

Evidence suggests that life events precede affective symptoms. Thus, it is likely that events trigger mood symptoms, consistent with the external trigger, or social zeitgeber theory, illustrated in Fig. 1. However, it is unclear whether bipolar and unipolar individuals are more vulnerable to life events or zeitgebers because they have an abnormally functioning pacemaker. It is also ambiguous whether social rhythm disruptions act as the mechanism by which events affect symptoms. For example, many studies did not assess social rhythms prior to life events, assess life events and social rhythms in close proximity to one another, and group individuals with potentially different vulnerabilities (i.e., bipolar vs. unipolar individuals). Longitudinal studies to determine the temporal association of circadian rhythm regularity, life events, and symptoms would eliminate several of these limitations. Further, standardized interviews (i.e., SADS-L, SCID) should be utilized to identify diagnostic groups and current mood state, or level of vulnerability, in community samples. This is particularly important as research suggests that social rhythm disruption events may be more likely to precipitate mania than depression (Malkoff-Schwartz et al., 2000, 1998). Utilizing larger sample sizes will also become important if comparing several groups. Such studies would allow a detailed examination of the consequences of life events, such as disruptions of social rhythms, and whether...
such consequences vary depending on the mood state (i.e., depressed, manic, mixed) and/or the diagnosis (i.e., unipolar, bipolar) of an individual (Chang et al., 2003).

Several studies found that social and physical zeitgebers (i.e., people, meals, noise, room temperature, light) affect biological rhythms (i.e., cortisol, body temperature, sodium, sleep, heart rate) in community samples; however, there has been only one study conducted with a unipolar depressed sample and none with a bipolar sample. The lack of research in this area may be because it is time consuming and expensive research to conduct. For example, it is very difficult to assess biological rhythms, such as body temperature and hormone levels, outside of a laboratory. Further, such methodology interrupts participants’ lifestyles and, thus, is difficult to do for more than a few days and requires considerable compensation to participants. Recent advances in noninvasive methods of measuring biological rhythms in one’s natural environment, such as with actigraphy, may further research in this area (Jones et al., 2005). Specifically, future research should examine the effects of various zeitgebers, and the characteristics of these cues (i.e., intensity, frequency), on biological rhythms in bipolar and unipolar samples to determine whether social zeitgebers are responsible for biological circadian rhythm disruptions. If the data fail to support this link, it would undermine the social zeitgeber theory and suggest that further attention needs to be paid to the influence of the pacemaker on these rhythms (see Fig. 1).

This review also highlighted the finding that bipolar and unipolar samples may have less social rhythm regularity, but that this was not associated with their current mood state; however, therapy regularizing social rhythms did improve mood in a bipolar sample (Frank et al., 2005). Additional research is needed to determine whether social and circadian rhythm regularity are prospectively predictive of bipolar episodes to lend credence to both theories in Fig. 1. Improvements upon earlier studies would include utilizing larger and more homogenous samples, assessing social and circadian rhythm regularity and mood daily for at least two weeks, and utilizing objective measures of circadian activity (e.g., actigraphy). Preferably, future research would also stabilize participants’ medications prior to assessing circadian rhythm regularity, as medications can alter circadian activity.

In summary, the social zeitgeber theory is an attractive explanation of affective episodes, given that it attempts to understand how external triggers (e.g., social zeitgebers) may affect mood. However, it is unclear from the literature whether it accurately reflects the chain of events that culminate in affective episodes. Although the social zeitgeber theory is an excellent working model, we encourage other investigators to consider and test alternative explanations, such as the internal trigger theory, or more specifically, vulnerability linked to genetic factors (i.e., serotonin transporter gene) (for review, see Alda, 2001). Investigators should also consider other explanatory theories of affective episodes, such as the influence of individual or personality traits (Swendsen et al., 1995), behavioral sensitization to stress (i.e., kindling model; Post & Weiss, 1997), for example, due to early trauma (Benes, 1994; Pine, 2003), and dysregulation due to one’s behavioral activation systems (for review, see Johnson, 2005; Johnson & Roberts, 1995; Power, 2005).

The importance of this research is highlighted by the numerous implications of elucidating an accurate model of affective disorders. For example, it would give treatment teams, families, and patients a framework in which to understand the mood disorders, as well as aid in their prevention and intervention. Thus, we hope that the issues raised in this review will stimulate future research that may ultimately lead to a more complete understanding of a causal pathway to unipolar and bipolar mood episodes.

References


