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Is there evidence for social rhythm instability in people at risk for affective disorders?

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Abstract

Social rhythm disruptions are thought to be related to the etiology of affective symptoms. 'Hypomanic personality' and 'rigidity' are hypothesized to be risk factors for affective disorders. We examined whether people scoring high on such scales would demonstrate instability of social rhythms and sleep. In a short-term prospective diary study with one group factor, the following three groups were selected from a non-university student sample: 'bipolar risk' (scoring high on the 'Hypomanic Personality Scale'; $n=56$); 'Unipolar risk' (scoring high on the 'Rigidity Scale'; $n=37$); and a control group (scoring low on both scales; $n=48$). The participants completed ratings of their activities and sleep for 28 days. People at risk for bipolar disorders showed a lower regularity of daily activities than controls. Their sleeping pattern was not characterized by fewer but by more variable hours of sleep. The unipolar risk group did not differ from the control group at all. Despite some limitations, there is partial evidence for social rhythm and sleep irregularities in people putatively at risk for bipolar disorders. Further research is, however, needed to replicate and extend these results.

Keywords: Hypomanic personality; Rigidity; Bipolar disorder; Circadian rhythm

1. Introduction

In DSM-IV, bipolar disorders are classified as affective disorders (American Psychiatric Association, 1994). However, the issue of whether a dimensional approach or a categorical classification of affective disorders is more appropriate remains controversial. As an alternative to strict diagnostic categorization, several researchers have suggested a spectrum concept where affective symptoms are located on a continuum. Whether there is a single spectrum of affective disorders encompassing bipolar and unipolar conditions is still under discussion (e.g. Angst, 1978; Goodwin and Jamison, 1990; Akiskal, 2003; Angst et al., 2003a,b). Of more relevance in our context is

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that according to such a spectrum concept, manic as well as depressive symptoms can differ in their intensities. Such a spectrum model with its dimensional point of view defines subsyndromal affective manifestations as part of the affective symptom spectrum, opening up the field for research in high-risk paradigms.

There are multiple factors that increase the probability of bipolar disorders. The influence of genetic transmisson (Nurnberger and Gershon, 1992; McGuffin et al., 2003), the effects of stressful life events and daily hassles (Healy, 1987; Wehr et al., 1987; Ehlers et al., 1988; Johnson and Roberts, 1995), and temperamental and trait-like factors such as the 'manic' and 'melancholic' types or attributional styles (Tellenbach, 1961; Akiskal and Akiskal, 1992; von Zerssen, 1996; Alloy et al., 1999) can be brought together in the model of a 'final common pathway' (Akiskal and McKinney, 1975). In this vulnerability-stress model, it is argued that different risk factors or their combinations lead to biochemical dysregulations and thereby cause affective symptoms.

From a theoretical as well as an empirical point of view, there is reason to assume that circadian rhythms play a central role in the origin and course of bipolar disorders. Research focused on the sleep-wake cycle suggests that circadian rhythms – primarily the sleep-wake rhythm - play a role in the origin and course of bipolar disorders. The sleep-wake cycle is clearly implicated in the pathophysiology of bipolar disorder by the fact that sleep deprivation has antidepressant and manicogenic effects (e.g. Wehr and Goodwin, 1983; Ehlers et al., 1988; Healy and Williams, 1989; Wehr, 1990; Leibenluft et al., 1996; Malkoff-Schwartz et al., 1998; Jones, 2001). Theoretical models emphasize that there might be an interplay of circadian rhythms with the modern world, and that there is a social rhythm in addition to purely circadian and biological rhythms (e.g. Ehlers et al., 1988; Frank, 2005). This suggests that factors such as the work schedule, regular activities (e.g. going to the gym, having family dinners) influence and stabilize or destabilize biological rhythms, especially if there is vulnerability for unstable rhythms. Ehlers et al. (1988) term such factors that characterize our daily schedule and also influence biological rhythms 'social zeitgebers'. They argue that disruptions in the social rhythm cause a dysregulation of biological patterns that in turn can cause affective symptoms in vulnerable individuals. Whether an irregular social rhythm causes biological dysregulations itself or influences biological patterns by influencing the sleep-wake cycle, which in turn causes a biological dysregulation, is yet unclear. Using the "Social Rhythm Metric" (SRM; Monk et al., 1990) to quantify social daily rhythms, several studies have demonstrated an association between social rhythm disruptions and affective symptoms. Monk et al. (1991) found that patterns of social rhythm in patients with unipolar depression demonstrated more intraindividual variability compared with a healthy control group. Although the focus of the theory of social zeitgebers (Ehlers et al., 1988) was originally on depression, disruptions of social and biological rhythms may be even more relevant for the course of bipolar disorders (e.g. Frank et al., 1999; Jones, 2001; Paykel, 2003).

In patients with bipolar disorders life events which cause a social rhythm disruption were more frequently observed prior to (hypo-)manic episodes than prior to control periods without affective symptoms (e.g. Malkoff-Schwartz et al., 1998, 2000). Similarly Ashman et al. (1999) found a significantly reduced social rhythm in patients with rapid-cycling bipolar disorder. Therefore there is evidence that for individuals with the diagnosis of an affective disorder, social rhythm disruptions appear more frequently than in healthy individuals and might increase the risk for relapses. We do not know yet, however, if such social rhythm disruptions precede the onset of affective disorders. There are no data available confirming the hypotheses that disruptions in the social rhythm can be considered as a causal risk factor for the occurrence of affective disorders. Nevertheless, the data indicate an association between irregular social rhythms and affective symptoms and can be integrated into a concept of reciprocal interaction where affective symptoms and social rhythm irregularities might reinforce one another.

In a recent study, Chang et al. (2003) found a reduced social rhythm both in a group of adolescents at high risk for developing bipolar disorders and in a sample of bipolar II patients than in a control group. Additionally, after a follow-up period of 20 weeks, a reduced social rhythm correlated with a higher probability for a hypomanic or a depressive episode. It should be noted that the SRM (Monk et al., 1990) was used retrospectively to assess the social rhythm,