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

Thomas D. Meyer\(^a\) \* & Silke Maier\(^a, b\)

\(^a\) Department of Clinical and Physiological Psychology; Eberhard Karls University; Psychological Institute; Christophstrasse 2; 72072 Tübingen, Germany

\(^b\) Universidade Federal do Rio Grande do Sul, Instituto de Psicologia, Porto Alegre, Brasil

Published in ‘Psychiatry Research’

Please keep in mind that there might be minor changes between this and the finally published version.

\*Corresponding Author. Tel.: +49 70 71 29 782 94; fax: +49 70 71 29 52 19; E-mail: th.meyer@uni-tuebingen.de
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 if people scoring high on such scales will demonstrate less stability of social rhythms and sleep. Short-term prospective diary study with one group factor. 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); 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 were showing lower regularity of daily activities compared to controls. Their sleeping pattern was not characterized by less but 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

Following DSM IV- classifications, bipolar disorders are part of the affective disorders (American Psychiatric Association, 1994). However, the discussion of whether a dimensional approach or a categorical classification of affective disorders is more appropriate remains controversial. As an alternative to the strict diagnostic categorization, several researchers suggest a spectrum concept where affective symptoms are located on a continuum. If one assumes one single spectrum of affective disorders encompassing bipolar and unipolar conditions or better distinguishes between a unipolar and bipolar spectrum is still discussed (e.g. Angst, 1978; Goodwin and Jamison, 1990; Akiskal, 2003; Angst et al., 2003a,b), Of more relevance in our context ist 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, which increase the probability of bipolar disorders. The influence of genetic transmission (Nurnberger and Gershon, 1992; McGuffin et al., 2003), the effects of stressful live events and daily hassles (Healy, 1987; Wehr et al., 1987; Ehlers, 1988; Johnson and Roberts, 1995) as well as temperaments and trait-like factors such as the “manic” and “melancholic” types or attributional style (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 Mc Kinney, 1975). In this vulnerability-stress-model it is argued that different risk factors or their combinations lead to the typical dysregulations in the biochemical system 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 and could show that this circadian rhythm – primarily the sleep-wake rhythm - plays 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 & 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 rhythm with the modern world, and that there is also a social rhythm in addition to purely circadian and biological rhythms (e.g. Ehlers et al., 1988; Frank, 2005). This means that factors such as a work schedule, regular activities (e.g. going to the gym, having family dinners) are thought to influence and stabilize or destabilize biological rhythms, especially if there is vulnerability for unstable rhythms. Ehlers et al. (1988) call 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 which in turn can cause affective symptoms in vulnerable individuals. Whether an irregular social rhythm causes biological dysregulations itself or influences biological patterns through 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, an association between social rhythm disruptions and affective symptoms has been demonstrated in several studies. Monk et al. (1991) has shown different patterns of social rhythm for patients with unipolar depression demonstrating more intraindividual variability compared to a healthy control group. Although the focus of theory of social zeitgebers (Ehlers et al., 1988) was originally on depression, the relevance of disruptions of social and biological rhythms is recently even more emphasized 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 who received the diagnosis of an affective disorder, social rhythm disruptions appear more frequently compared to 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. Still there is 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 indicates an association between irregular social rhythms and affective symptoms and can be integrated in 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 high-risk group of adolescents for developing bipolar disorders and in a sample of bipolar II patients compared to 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. Although this confirms their hypothesis, the SRM (Monk et al., 1990) was used retrospectively to assess the social rhythm, i.e. asking the participants to describe their typical rhythm for the month prior to the interview. The SRM is usually used to assess the daily times for different activities and to empirically define the stability of the social rhythm. Furthermore, Chang et al. (2003) did not actually investigate if changes in regularity (i.e. social rhythm disruptions) – as hypothesized – influence symptom status but whether self-reported social rhythm irregularities are associated with symptoms. Their results, however, suggest that people who are considered within the bipolar spectrum (cyclothymia, bipolar II, bipolar NOS) report fewer regular activities for the last month.

Using such as measure such as the SRM to retrospectively assess social rhythm stability might lead to distorted results due to memory bias. Therefore we wanted to know what happens if people at risk for affective disorders, especially bipolar disorders, complete the SRM on a daily basis. The social zeitgeber model was originally proposed for depression,
therefore it seemed important to consider the risk for unipolar and bipolar affective disorder. To define vulnerability, we adopted a behavioral or psychometric high-risk approach (e.g. Depue et al., 1989; Klein and Anderson, 1995; Meyer and Hautzinger, 2001). Using a psychometric approach, the vulnerability is operationalized by scores on a test or psychometric instrument (e.g. personality inventory) instead of being a first-degree relative of a patient. Compared to the biological-genetic high-risk approach, the main advantages of the behavioral or psychometric approach are that large samples can easily be screened and that the risk status of the individual is not defined by the biological relationship to an index patient. Since we do not know much yet about possible different pathways for familial and non-familial forms of the disorder, the results of the psychometric approach are at least not limited to the familial form.

In Germany there is a long tradition to see the so-called Typus melancholicus as a premorbid personality style for depression (Tellenbach, 1961; von Zerssen, 1996). The Typus melancholicus is characterized by obsessive-compulsive, perfectionistic and rigid traits with the latter one being a central characteristic. Several studies showed that relatives of patients with affective disorders have elevated scores on Rigidity (e.g. Maier et al., 1992, 1995; Lauer et al., 1997), and these authors consider rigidity as a risk factor for affective disorders, especially unipolar depression (e.g. Mundt et al., 1999). Instead of using attributional styles or dysfunctional beliefs as others (e.g. Hammen and Goodman, 1990; Alloy et al., 1997, 1999), we decided to use von Zerssen´s model (1996) as a guideline to select risk indicators and therefore chose one of the core features of the Typus melancholius - the Rigidity Scale of the Munich Personality Scale (von Zerssen et al., 1988) - as a risk indicator for unipolar depression. Unfortunately the Rigidity Scale itself has not yet been tested in other studies longitudinally if it does predict depression. A concept that is related to rigidity – perfectionism – has, however, been shown to predict depression (e.g. Hewitt et al., 1996; Kenney-Benson and Pomerantz, 2005). In his model, von Zerssen also described a premorbid
personality or temperament associated with mania, the Typus manicus. Characteristics of this Typus manicus are high self confidence, unconventionality, and inconsistency. Sometimes they are upbeat, gregarious, and very energetic people. We chose the “Hypomanic Personality Scale” (HSP, Eckblad and Chapman, 1986) as an indicator for this Typus manicus and therefore risk for bipolar disorders, because conceptually the HPS fits von Zerssen’s description best, and there is growing evidence that the HPS is associated with affective symptoms (e.g. Klein et al., 1996; Meyer, 2002a; Meyer and Hautzinger, 2003; Blechert and Meyer, 2004). People scoring high on the HPS also show similar aberrations in processing of emotional information as do bipolar patients (e.g. French et al., 1996; Lyon et al., 1999), and there is evidence for familial aggregation of HPS scores (Meyer and Hautzinger, 2001). Most compelling, however, is that the HPS predicted bipolar disorders in a 13 year follow-up study (Kwapil et al., 2000). Therefore it seems justified to use the HPS as a risk factor for bipolarity.

One hypothesis was that there is an association between vulnerability for affective disorders and irregularity of the social rhythm. People putatively at risk for bipolar disorders – as defined by the HPS – were expected to show an unstable social rhythm over time, reflecting the core vulnerability for bipolar disorders: the instability of circadian-biological rhythms (e.g. Goodwin and Jamison, 1990). Individuals hypothesized to be at risk for unipolar depression, however, were expected to have a very stable social rhythm due to their rigidity. Social rhythm disruptions are seen as risk factors for the occurrence of depressive symptoms (e.g. Ehlers et al., 1988), but at baseline it is assumed that rigidity is associated with a very stable daily rhythm. In a premorbid status a stable social rhythm might protect people with high rigidity against depression. Due to their high rigidity, however, they display a very low level of flexibility in their life-styles and life-rhythms. According to von Zerssen (1996) they might be at risk not to be able to deal with rhythm disruptions in a healthy, adaptive way. In the long run their risk of developing affective symptoms increases by their non-adaptive
(rigid) need for rhythmicity, and any kind of dysregulations may lead to strong affective reactions. Participants who are neither at risk for bipolar disorders nor at risk for depression were thought to have an intermediate position between the two high-risk groups concerning the stability of social rhythm. The rational for this was that their social rhythm is on one hand more flexible and responsive to situational requirements, but returns quickly to their individual (stable) baseline. In addition to this main hypothesis we also tested if the notion is true that people with a hypomanic-hyperthymic temperament habitually sleep less than others (Eckblad and Chapman, 1986; Akiskal, 1996). Although they might report this as a trait for themselves (which is considered highly socially desirable, implying being more productive and energetic), nobody has ever tested if this reflects reality if one looks at sleep reports on a daily basis. Therefore using a diary we want to see if they truly sleep less than control subjects. In viewing an instability of biological-circadian rhythms as the core vulnerability for bipolar disorders (e.g. Goodwin and Jamison, 1990) we predict, however, a greater intraindividual variability of sleep for the bipolar risk group compared to others.

2. Methods

2.1 Participants

In 1998/1999 6000 students who either still attended college or high-school or went to vocational schools receiving a specific training for a job (e.g. cosmetology, mail services, banking, etc.) completed a questionnaire package including the German version of the Hypomanic Personality Scale (HPS; Eckblad and Chapman, 1986; Meyer, 2002b; Meyer and Hautzinger, 2003) and the subscale Rigidity of the Munich Personality Test (MPT, von Zerssen et al., 1988). The study was approved by the ethical committee of the German Psychological Association (DGPs), and subjects provided written consent prior to enrolling the study. The questionnaires were completed in the classrooms. The original sample consisted of 6000 students. Non-native German students were excluded (see Chmielewski et
al., 1995). Additionally, we measured an Infrequency score (Chapman et al., 1976) by which inadequate response patterns can be identified. A score higher than 3 indicates unusual and untypical responses and identifies those who tend to distort their answers. Individuals were excluded who scored 3 or higher. Those whose score on the Lie scale of the EPI was 5 or higher (Eysenck and Eysenck, 1964) were also excluded from the study.

Then we selected three groups that corresponded to different vulnerability status for affective disorders based on their HPS and Rigidity scores: (1) Group with vulnerability for bipolar disorders (bipolar): Students whose scores were in the upper 10% of the score distribution of the original cohort in the HPS; (2) Group with vulnerability for unipolar depression (unipolar): Students whose score were in the upper 10% of the score distribution of the original cohort in the Rigidity Scale, (3) Control group (control): Subjects whose scores did not exceed $M + 1/2 SD$ in neither the HPS nor the Rigidity Scale. The control group was matched for sex and age to the two risk groups as to limit sample size (for details about the study: Krumm-Merabet and Meyer, 2003; Meyer and Keller, 2003; Blechert and Meyer, 2005; Meyer and Krumm-Merabet, 2005).

Persons who met the criteria for one of the three groups were invited to an interview ($n = 357$). Two-hundred-forty-seven (69.2 %) participated. There was no evidence for selective attrition concerning risk status, $\chi^2 (2, n = 357) = 2.20, n.s.$, or age, $t (355) = 0.78, n.s.$, but women were more willing to further participate (74.3 % [159/214]) than men (61.5 % [88/143]), $\chi^2 (1, n = 357) = 6.55, P = 0.01$. Furthermore, all participants were asked to take part in a prospective study for four weeks completing a diary every day. This diary contained the Social Rhythm Metric (see below). To be included in the analyses, the participants had to provide us with continuous data from the diary, i.e. completing at least 21 days. Ninety-nine female and 41 male participants ($n = 141$) agreed and completed the diary continuously every day (predominantly female with 70.7 %). The mean age was 18.18 ($SD = 2.14$; range 15-23). Thirteen students (9.2 %) had a high level of education (13 years of education, “Abitur”), the
majority with 54.6 % (n = 77) of students attended school for 10 years, representing an intermediate level of education (“Mittlere Reife”), and 10.6 % (n = 15) of the students had either a low level of education (9 years, “Hauptschule”). Additional 9.9 % (n = 14) were attending vocational schools and 14.2 % (n = 20) had not yet completed school at the time of the study. Two persons did not provide this kind of information (1.4 %)

The final group sizes were therefore (1) a group with vulnerability for bipolar disorders (bipolar): n = 56 (39.7 %), (2) a group with vulnerability for unipolar depression (unipolar): n = 37 (26.2 %), and (3) a control group (control): n = 48 (34 %) (Table 1).

2.2 Materials

2.2.1 Hypomanic Personality Scale (HPS; Eckblad and Chapman, 1986):

The HSP contains 48 items and proved to be reliable and sufficiently stable (e.g. Eckblad and Chapman, 1986; Klein et al., 1996). The German version achieved an internal consistency of .87 to .89 (Cronbach’s Alpha) and a retest-correlation of $r_{tt} = 0.77$ (n = 74; interval: 7 months) (Meyer, 2002b; Meyer et al., 2000). Different studies have shown its validity for the bipolar spectrum (e.g. French et al., 1996; Kwapil et al., 2000; Meyer, 2002b; Meyer and Hautzinger, 2003).

2.2.2 MPT- “Rigidity- Scale”( von Zerssen, et al., 1988):

The Rigidity- Scale is one of the six scales of the Munich Personality Inventory (von Zerssen, 1977). It contains eight items and was validated with a Cronbach’s $\alpha$ between 0.68 and 0.84. After a 12-month test-retest interval, it reached a retest-correlation of $r_{tt} = 0.70$. Different studies showed its association to unipolar depression (e.g. Maier et al., 1992; Heerlein et al. 1996; Mundt et al., 1999).

2.2.3 Structured Clinical Interview for DSM-IV Axis I disorders (SCID, First et al., 1996).

Participants were interviewed using the Structured Clinical Interview for DSM IV (SCID, First et al., 1996; German version: Wittchen et al., 1997) to assess symptoms of affective
disorders. This interview was chosen on the basis of its accepted and widespread international use. Interviews were conducted by graduate psychology students trained extensively for this purpose. Specifically, this training consisted of two full weekends and additional supervised training interviews. Furthermore, all interviews were videotaped to permit a consensus decision for all diagnoses.

2.2.4 Social Rhythm Metric (SRM; Monk et al., 1990):

The Social Rhythm Metric quantifies the stability of an individual’s daily routine. It lists 17 activities which have been identified as central in everyday life (Rehm, 1978; Kanner et al., 1981) and leaves space for two more optional activities for the individual. The SRM is meant to be completed every day, writing down the exact time of the beginning of an activity. A short version of the SRM was proposed by Monk et al. (2002) where only five activities (get out of bed, first communication, start work, have dinner, go to bed) are considered. Using the SRM-5 short form, we investigated these five central activities. As two of the five activities are related to sleep-wake-patterns (“get out of bed” and “go to bed”) this short version of the SRM emphasizes the sleep-wake-cycle as a major anchor in the daily social rhythm. The score of the SRM rises with the regularity of daily activities. For this purpose one calculates the average time for each activity and counts how often these took place within a certain predetermined time frame, i.e. average time +/- 45min. Then one divides these hits through the number of activities performed more than three times a week (see for details: Monk et al., 1990). The SRM has a moderate to high retest-reliability. Monk et al. (1990) reports a highly significant correlation ($\rho = 0.44; P < 0.001$) between week 1 and week 2. The SRM has been described as a valid instrument in different studies (e.g. Monk et al. 1990; Frank et al. 1994, 1997, 1999; Chang et al., 2003).

2.3 Procedure
Following the procedure used by Alloy et al. (1997) and by Lovejoy and Steuerwald (1997), participants were asked to complete a diary every day. The diary consisted of 28 sheets, each for one day. Each sheet included the SRM (e.g. time going to bed, falling asleep, starting work, doing sport). In order to obtain the same stability and consistency, each participant was instructed thoroughly about how to fill out the sheets daily and to send them back daily. For this purpose they all received 28 pre-paid envelopes. If the sheet for a specific day was not returned to us within 2 days (except weekends), the subjects were called and asked if they have forgotten to fill out (leading to a drop out) or if there was a mailing problem. Subjects were paid 80 DM (about 40 US $) for their participation.

3. Results

3.1 Dropout analyses and distribution of affective disorders

Comparing the ones who continuously completed the diary \( (n = 141) \) with those that did not, there was no difference regarding risk status, \( \chi^2 (2, n = 247) = 2.74, \text{n.s., or age, } t (245) = -0.54, \text{n.s.} \). However, more women (100/159) than men (41/88) provided us with complete data from the diaries, \( \chi^2 (1, n = 247) = 6.15, P = 0.01 \). Completion of the diary was not associated with the presence or absence of affective disorders, \( \chi^2 (1, n = 247) = 2.04, \text{n.s.} \).

In Table 1 the distribution of bipolar disorder and major depression disorder is displayed for the three groups. Although bipolar disorders were only diagnosed in the two risk groups, with 5.4 % in the bipolar risk group and 2.7 % in the unipolar risk group, there were no significant group differences in this sample of adolescents and young adults, \( \chi^2 (2, n = 141) = 2.69, \text{n.s.} \). Episodes of major depression were found in all three groups but did also not differ between groups, \( \chi^2 (2, n = 141) = 1.53, \text{n.s.} \).

3.2 Social rhythm: means

Comparing all three groups concerning the Social Rhythm Score (SRS), a one-way ANOVA resulted in a significant effect, \( F (2, 141) = 4.61, P = 0.01 \). Running planned simple
contrasts comparing the risk groups with the control group, the unipolar risk group did not differ significantly from the control group ($P = 0.18$) but the bipolar risk group did significantly differ from the control group ($P = 0.01$) indicating a lower regularity of daily activities in people hypothesized to be at risk for bipolar disorders (see Table 2).

To see if these results are due to people suffering from affective disorders, we also ran the analysis for the sample excluding the participants with a bipolar disorder ($n = 4$) and those with a major depression disorder ($n = 18$). The ANOVA did not approach significance any more, but the same trend was still seen, $F (2, 119) = 2.55, P = 0.08$.

### 3.3 Social rhythm: stability

To test the stability of the SRS over time, we calculated the retest-correlation of SRS for each week with the following weeks. Table 3 shows the retest-correlations for the sample as a whole. The range of the test-retest correlation is 0.20 to 0.41, all being significant at least at $P < 0.05$. There is therefore some indication for stability of the SRS score over time but the correlations are fairly low.

We also calculated the retest correlations separate for all three groups (see Table 3). In the control group we find more significant retest correlations than in the unipolar and bipolar risk group. Testing for possible significant differences in stability of the SRS over time, we used Fisher’s $Z$ transformation for the averaged retest correlations for all weeks. However, none of the comparisons reached significance, i.e. despite different levels of social rhythm regularity the three groups were similar regarding the stability of the SRS scores ($z < 0.3$, all $n.s.$)

### 3.4 Sleep duration

Hypomanic temperament is thought to be associated with less sleep but we did not find the expected differences between groups concerning means of sleep duration using the diary, $F (2, 138) = 0.10, n.s.$ Regardless of risk status the average duration of sleep was comparable over four weeks, and this was also the case if we excluded people suffering from major
affective disorders, $F (2, 116) = 0.17, n.s.$ Table 2 displays the means and standard deviations for the total sample.

Additionally, we assumed that the bipolar risk group shows a less stable sleep pattern compared to both the unipolar risk group and the control group. To test this prediction, we calculated the intraindividual standard deviations in sleep duration over the four weeks and averaged them for the three groups (Table 2). A one-way ANOVA indicated a highly significant effect, $F (2, 134) = 6.19; P < 0.003$. Planned contrasts revealed - as expected - that the unipolar risk group did not differ from controls ($P = 0.98$), but the bipolar risk group showed significantly more variability in their sleep pattern ($P < 0.01$). This result remained significant even after excluding all participants with bipolar and unipolar affective disorders, $F (2, 114) = 5.53, P < 0.005$.

4. Discussion

We examined whether there is evidence of social rhythm instabilities in people at risk for affective disorders. We actually found evidence for reduced social rhythm scores measured by the Social Rhythm Metric (SRM; Monk et al, 1990) for participants of the bipolar risk group compared to the control group. This was not the case for people from the unipolar risk group, defined by high rigidity scores. When excluding participants who already were diagnosed with bipolar or unipolar disorders, this effect is no longer significant but a trend remains that people with high HYP scores show reduced social rhythms. Correlations of the Social Rhythm Score between the four weeks become all but one significant. However, correlations are fairly low ($0.20 \geq r \leq 0.41$). Looking at the sleeping pattern we found in contrast to descriptions of the hypomanic temperament (e.g. Eckblad and Chapman, 1986; Akiskal, 1996) that people with high HYP scores did not sleep less averaged over 28 days compared with others. However, although sleeping the same amount on average, their daily sleep pattern
shows much more fluctuations over time than the one of controls or people hypothesized at risk for unipolar depression.

Before discussing the other results, it seems important to address the issue why the stability of social rhythm seems fairly low in our sample. Looking at retest-reliability for week 1 and week 2 Monk et al. (1990) reported a retest-correlation of $r = 0.44$. All participants were intensively instructed how to complete the SRM. One reason for the lower stability might be that our subjects were asked to send back the forms daily. It seems that Monk et al. (1990) collected the booklets containing the SRM only at the end of the two-week period. Furthermore our sample consisted only of adolescents and young adults. Although this is highly speculative it seems plausible to assume that they tend to have a lower regularity in their life-styles than adults. These factors might explain that the retest-correlations of the SRM scores are lower than might have been expected.

The background for our study was that it is assumed that depression and bipolar disorders are related to social rhythm disruptions. Caused by stress or other conditions (e.g. divorce, birth of a child, traveling over time zones) such disruptions in social rhythms are thought to lead to circadian rhythm disruptions. The latter ones cause physical symptoms that interact with biological and/or psychological vulnerabilities. The interaction of these factors is hypothesized to lead to affective symptoms in people who are at risk (e.g. Ehlers et al. 1988; Monk et al., 1990; Frank et al. 1994). There is evidence that circadian rhythm disruptions, mostly defined by changes in sleep or the SRM, are associated with depression and mania (e.g. Wehr et al., 1982; Wehr, 1990; Brown et al., 1996; Malkoff-Schwartz et al., 1998, 2000; Bukhari et al., 2003; Chang et al., 2003).

All studies focused so far on patients diagnosed with either unipolar or bipolar affective disorders. Theoretically social rhythm disruptions are always seen as an independent factor interacting with the vulnerability. Chang et al. (2003) examined if social rhythm regularity is different in people diagnosed with cyclothymia and bipolar II disorder compared to controls
and found evidence for this assumption. Furthermore low social rhythm regularity predicted occurrence of hypomanic and major depressive episodes. Our data does not allow predictions of onset of affective episodes because we were not able to follow-up the participants long enough. Nevertheless assessing social rhythm and sleep prospectively over four weeks on a daily basis the results show that social rhythm irregularity and instability of sleeping pattern is only pronounced in people putatively at risk for bipolar disorders and not in people hypothesized at risk for unipolar depression.

Overall the results support the available evidence for the Hypomanic Personality scale as an indicator for vulnerability of bipolar disorders (e.g. French et al., 1996; Kwapil et al. 2000; Meyer, 2001; Meyer and Hautzinger, 2003; Blechert and Meyer, 2004). Looking at our results one might question, however, the accuracy of criteria for or descriptions of the hyperthymic-hypomanic temperament assuming “habitual less sleep” (e.g. Eckblad and Chapman, 1986; Akiskal, 1996). It is not clear if this notion of less need for sleep or actually less sleep is a recall bias by people characterized by a hypomanic temperament when asked for their sleeping pattern or if this idea is more derived by the clinical picture of hypomania. Our data suggest that these people scoring high on the HYP scale are better characterized by an unstable sleeping pattern resulting in switching between hyposomnia and hypersomnia. This is consistent with the idea that hyperthymia is part or special form of cyclothymia (e.g. Akisskal, 1996) and that the core vulnerability for bipolar disorders is instability of biological rhythms (e.g. Goodwin and Jamison, 1990; Frank, 2005). Another consideration focuses on the fact that the groups also might differ in their sleep efficiency. Sleeping time is measured by the time between the two activities “go to bed” and “get out of bed”. Differentiating between actual sleep and time spent in bed one can argue that groups do not actually differ in the amount of time spent in bed, but in the amount of sleep. Our study does not help resolve this question. Better controlled studies in sleep laboratories are needed to clarify such issues.
Looking at risk for depression, people scoring high on Rigidity (von Zerssen et al., 1988) did not seem to show any evidence for deviations in social rhythms and sleep. On one hand there is evidence for an association between melancholia and rigidity (e.g. Maier et al., 1992; Lauer et al., 1997; Mundt et al., 1999), but on the other the only existing longitudinal study stems from our own laboratory. We did not find evidence that rigidity is predicting depression over a two-year period (Blechert and Meyer, 2005). Further studies are needed to resolve the question if rigidity is a valid indicator of risk for depression at all. Related concepts, however, seem to predict depression (e.g. Hewitt et al., 1996).

Before drawing final conclusions some limitations of the study should be mentioned: First, we had a selective dropout of male participants so that the majority of our sample was female. Chang et al. (2003) reported that women endorsed significantly more activities as ‘regular’ than males, but they did not assess the Social Rhythm Metric on a daily basis so comparisons are difficult. On a theoretical level there seems, however, to be no reason to assume that social rhythm disruptions themselves have differential effects for women and men when it comes to affective disorders. Second, although we had a prospective design this only covered 28 days. Our primary goal was to assess the stability of sleep and social rhythm and not the occurrence of affective episodes. However, this means that we cannot say anything about the relationship between social rhythm disruptions and the onset of mania or depression. One might also raise the question if the group differences are due to any potential confounding variables (e.g. season, life events). The subjects started at different points of time completing the diary (over a 9 month period) and we did actually not control for such variables. Nevertheless we think that a lot of potential confounding variables are randomly distributed over the groups. However, it seems reasonable to assume that people at risk experience more (perhaps even self-inflicted) stress, so it could be that group differences (e.g. unstable sleep pattern) are indeed reflecting different stress levels. Last but not least – and related to the former limitation - it has to be emphasized that we cannot disentangle the question if social rhythm
disruptions or changes in sleep are of more concern because sleep parameters were also used to estimate the social rhythm. More research is definitely needed to see if social rhythm disruptions themselves lead to changes in symptoms or even onset of episodes in people at risk for affective disorders or if these are only mediated by changes in sleep.

Despite these limitations, our conclusion is that we found evidence for reduced social rhythms in young people hypothesized to be at risk for bipolar disorders, referring to daily routines as well as to sleep. Since instability of the social rhythm and sleep are seen as potential risk factors for the onset and occurrence of mania and depression, further studies are needed to examine the interplay of vulnerability, social rhythm (disruptions) and symptoms in people at risk for affective disorders. Of special relevance would be to investigate if social rhythm disruptions are mediated by sleep disruptions.
Acknowledgements:

This research was supported by a grant from the Deutsche Forschungsgemeinschaft to T. D. Meyer (DFG Me 1681/7-1). We would like to thank our team of graduate students and research assistants for their assistance.
5. References

Akiskal, H.S., 1996. The prevalent clinical spectrum of bipolar disorders: Beyond the DSM-IV. Journal of Clinical Psychopharmacology 16 (Suppl.1), 4S-14S.


Table 1:
Sample description and Distribution of affective disorders depending on risk group status

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unipolar Risk Group</th>
<th>Bipolar Risk Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>48</td>
<td>37</td>
<td>56</td>
</tr>
<tr>
<td>N women (%)</td>
<td>34 (70.8 %)</td>
<td>27 (73.0 %)</td>
<td>39 (69.6 %)</td>
</tr>
<tr>
<td>Affective Disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bipolar</td>
<td>0</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Major Depression</td>
<td>8</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Mean  (SD)</td>
<td>Age</td>
<td>HPS score</td>
<td>Rigidity score</td>
</tr>
<tr>
<td></td>
<td>18.33 (2.10)</td>
<td>16.19 (4.55)</td>
<td>1.85 (1.13)</td>
</tr>
<tr>
<td></td>
<td>18.32 (2.11)</td>
<td>15.81 (4.88)</td>
<td>5.45 (0.57)</td>
</tr>
<tr>
<td></td>
<td>17.95 (2.20)</td>
<td>33.78 (3.91)</td>
<td>3.13 (2.19)</td>
</tr>
</tbody>
</table>

Notes. Unipolar Risk = High scores on the Rigidity Scale; Bipolar Risk = High scores on the HPS; HPS = Hypomanic Personality Scale (Eckblad & Chapman, 1986); Rigidity = Rigidity scale from the MPI (Zerssen et al., 1988). Diagnoses were assessed with SKID I (First et al., 1995).
Table 2:
Means and Standard Deviation of Social Rhythm Score, intraindividual Stability of Social Rhythm Score over four weeks, Sleep Duration and Stability of Sleep over four weeks

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unipolar Risk Group</th>
<th>Bipolar Risk Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 48</td>
<td>N = 37</td>
<td>N = 56</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SRS</td>
<td>2.47 0.81</td>
<td>2.71 0.94</td>
<td>2.19 0.75</td>
</tr>
<tr>
<td>F (2, 141)</td>
<td>4.61, 0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>469.04 47.56</td>
<td>468.60 54.33</td>
<td>473.10 62.92</td>
</tr>
<tr>
<td>F (2, 138)</td>
<td>0.01, n.s.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stability of Sleep</td>
<td>88.57 30.39</td>
<td>89.13 27.47</td>
<td>145.43 142.58</td>
</tr>
<tr>
<td>F (2, 134)</td>
<td>5.53, &lt; 0.005</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes. Unipolar Risk = High scores on the Rigidity Scale; Bipolar Risk = High scores on the HPS; HPS = Hypomanic Personality Scale.

SRS = Score of the Social Rhythm Metric (Monk et al., 1990); Sleep Duration = minutes of sleep per night; Stability of Sleep = Intraindividual Variation of the sleeping duration over four weeks.
Table 3:
Retest- Correlations of the Social Rhythm Scores for each week with the following

<table>
<thead>
<tr>
<th></th>
<th>Total (N= 141)</th>
<th>Control (N = 48)</th>
<th>Unipolar Risk Group (N= 37)</th>
<th>Bipolar Risk Group (N= 56)</th>
</tr>
</thead>
<tbody>
<tr>
<td>week 1 * week 2</td>
<td>0.17*</td>
<td>0.04</td>
<td>0.25</td>
<td>0.23</td>
</tr>
<tr>
<td>week 1 * week 3</td>
<td>0.30**</td>
<td>0.13</td>
<td>0.41**</td>
<td>0.15</td>
</tr>
<tr>
<td>week 1 * week 4</td>
<td>0.32**</td>
<td>0.30*</td>
<td>0.31</td>
<td>0.25</td>
</tr>
<tr>
<td>week 2 * week 3</td>
<td>0.24**</td>
<td>0.20</td>
<td>0.26</td>
<td>0.14</td>
</tr>
<tr>
<td>week 2 * week 4</td>
<td>0.27**</td>
<td>0.37**</td>
<td>0.14</td>
<td>0.23</td>
</tr>
<tr>
<td>week 3 * week 4</td>
<td>0.43**</td>
<td>0.37**</td>
<td>0.36*</td>
<td>0.40**</td>
</tr>
<tr>
<td>Estimated Mean Correlation</td>
<td>0.29</td>
<td>0.25</td>
<td>0.29</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Notes: There were no significant differences in retest-correlations between groups if one averaged the coefficients over the weeks (all z < 0.3, n.s.); Mean correlation were obtained through Fisher’s Z transformation.

* P < 0.05
** P < 0.01