Sleep-EEG in borderline patients without concomitant major depression: a comparison with major depressives and normal control subjects

José Manuel De la Fuente\textsuperscript{a,b,c,*}, Julio Bobes\textsuperscript{b}, Coro Vizuete\textsuperscript{c}, Julien Mendlewicz\textsuperscript{a}

\textsuperscript{a}Department of Psychiatry, Erasme Hospital, Free University of Brussels, 808 route de Lennik, B-1070 Brussels, Belgium
\textsuperscript{b}Faculty of Medicine, Oviedo University, Julian Clavería 6, 33006 Oviedo, Spain
\textsuperscript{c}Hôpital Psychiatrique de Lannemezan, Route de Toulouse, F-65300 Lannemezan, France

Received 17 May 2000; received in revised form 5 October 2000; accepted 30 November 2000

Abstract

The link between borderline personality disorder (BPD) and the affective disorders remains controversial. The aim of this study was to examine the relationships between BPD and major depression (MD) from the perspective of sleep parameters and to contribute to the characterisation of the sleep-EEG in BPD. We compared 20 off-medication BPD in-patients without co-existing MD with 20 sex- and age-matched MD patients without BPD and 20 sex- and age-matched control subjects. BPD patients had a greater prevalence of drug or alcohol abuse and suicide attempts than MD patients. MD patients had higher scores on the Hamilton Depression Rating Scale (HDRS). Both BPD and MD patients had less total sleep time, more prolonged sleep onset latency, and a greater percentage of wakefulness than control subjects. BPD patients and control subjects had more stage 2 sleep than MD patients. BPD patients had a longer duration of rapid eye movement (REM) sleep, and less stage 3, stage 4 and slow wave sleep than MD patients and control subjects. REM latency did not differentiate the three groups. BPD and MD patients shared sleep-continuity characteristics, but sleep architecture differentiated the two groups. BPD patients with a past history of MD had more wakefulness and less slow wave sleep than BPD patients without a history of MD; other sleep parameters, age, sex and HDRS scores were not statistically different in the two BPD subgroups. Although BPD and MD may coexist, the present study offers more arguments favouring the concept that they are not biologically linked and that BPD patients with depressive symptoms often experience an affective syndrome different from that in MD patients without BPD, in terms of quality and duration of symptoms and of the biological substrate.

Keywords: Borderline; Personality; Biological substrate; Distinct affective syndrome; Polysomnography

* Corresponding author. Hôpital Psychiatrique de Lannemezan, Route de Toulouse, F-65300 Lannemezan, France. Tel.: +33-5-6299-5525; fax: +33-5-6299-5305.

E-mail address: jdlf@ch-lannemezan.fr (J.M. De la Fuente).

0165-1781/01/$ - see front matter © 2001 Elsevier Science Ireland Ltd. All rights reserved.

PII: S 0 1 6 5 - 1 7 8 1 ( 0 1 ) 0 0 3 3 0 - 4
1. Introduction

Borderline personality disorder (BPD) manifests a wide variety of symptoms. BPD has been associated with several pathological conditions such as epilepsy, affective disorders, schizophrenia, schizoaffective disorder, and atypical psychoses (Andrulonis et al., 1982; Post et al., 1982; Schulz et al., 1989). Although the hypothesis of a relationship with epilepsy is difficult to support, there is evidence of an excess of electroencephalographic (EEG) slow activity, which can indicate brain dysfunction, in patients with BPD (De la Fuente et al., 1998). Other methodologies, such as positron emission tomography, have also shown the existence of brain abnormalities in BPD (De la Fuente et al., 1997).

The link between BPD and the affective disorders is controversial (Korzekwa et al., 1993; Coid, 1993; Rogers et al., 1995). Arguments for a link between BPD and affective disorders (Schulz et al., 1989) are contrasted with those that suggest the two disorders to be independent (Soloff et al., 1989; Benson et al., 1990). Gunderson and Phillips (1991) conclude that the two disorders may coexist, but are not etiologically related. Some authors have suggested that depression associated with BPD is distinguished from depression without BPD by the quality and duration of symptoms (Westen et al., 1992; Coid, 1993; Rogers et al., 1995). Moreover, other authors have proposed that the depressed symptoms in BPD might have a biological substrate that is distinct from that in depressive illness without concomitant BPD characteristics (Krishnan et al., 1984; Korzekwa et al., 1993; Kavoussi et al., 1993; De la Fuente and Mendlewicz, 1996). Pharmacological studies have shown differences in medication response between BPD and MD patients. Conventional antidepressants have been reported to lead to poor response and even worsening in BPD (Cole et al., 1984; Soloff et al., 1989).

The dexamethasone suppression test (DST) and the thyrotropin-releasing hormone stimulation test (TRH-ST), which have proved valuable in the study of affective illness, have also been studied in BPD (Korzekwa et al., 1991; De la Fuente and Mendlewicz, 1996). In some of these studies, positive findings with the DST and TRH-ST seem to be more related to an Axis I diagnosis of MD than to BPD itself (Soloff et al., 1991). The only study evaluating the DST and the TRH-ST in BPD patients without a co-existing diagnosis of MD (De la Fuente and Mendlewicz, 1996) provided no evidence for a biological link between BPD and the MD.

Another approach to the putative affective nature of BPD is the study of sleep-electroencephalography (EEG), which has been useful in the biological characterisation of depressive and other psychiatric syndromes (Benca et al., 1992). To our knowledge, there have been at least eight studies on the sleep-EEG in BPD. Most have disregarded or not taken into account the existence of a concomitant Axis I MD in the BPD patients. Bell et al., (1983) described the ‘characteristic short REM latency reported in MD’ in a group of eight Research Diagnosis Criteria (RDC)-primary major depressive patients with borderline symptoms and 11 primary depressive patients without borderline symptoms. McNamara et al. (1984) found sleep continuity disturbances, greater rapid eye movement (REM) activity and shortened REM latency in 10 depressed borderline women and MD patients compared with controls. Reynolds et al. (1985) compared 20 retrospective and prospective BPD (MD was not an exclusion criterion and most patients had RDC diagnoses of depression) to 10 MD patients and to controls; they found similar REM latency values in both patient groups. King et al. (1987) compared 18 BPD (eight with current or past history of MD) patients with 16 MD patients and controls; BPD patients could not be distinguished from controls, whereas MD patients had shortened REM latencies compared with the other two groups. Lahmeyer et al. (1988) found similar EEG-sleep abnormalities in 17 BPD patients (eight also had RDC diagnoses of MD) and 20 MD patients; stage 1 sleep was lower and sleep efficiency was higher in the BPD group. In these five studies, the sleep-EEG perturbations could be more directly linked to a concomitant MD than to BPD itself (Lahmeyer et al., 1989).

Only three studies have clearly considered the existence of MD in their BPD patients. Akiskal et al. (1985) compared 24 non-MD BPD patients (not diagnosed with the Diagnostic Interview of
Borderlines) to 30 non-BPD MD patients, 16 patients with other personality disorders, and 14 controls (the non-MD BPD patients had significantly higher scores on the Beck Depression Inventory than the major depressives); they found similar REM latencies in the BPD and MD groups. In the study of Benson et al. (1990), 18 BPD patients with and without a present or past history of affective disorder were compared to each other and to controls. The three groups could not be distinguished in terms of REM latency; the BPD group had less total and delta sleep and more time awake after sleep onset than controls. Battaglia et al. (1993) studied 10 ‘never-depressed’ BPD patients and controls; they found reduced REM latency in the BPD group.

Taken together, the phenomenology and duration of the depressive episodes in BPD, along with their poor response to conventional antidepressants and the findings of some TRH-ST, DST and EEG sleep studies, provide suggestive evidence against the existence of a biological link between BPD and MD. Furthermore, the depressive symptoms observed in BPD patients might not have the same biological substrates as those found in MD patients.

The aim of our without Major Depression study was to further examine the relationships between BPD present and MD patients from the perspective of sleep-EEG parameters and to contribute to the characterisation of the sleep-EEG in BPD patients without Major Depression. Specifically, we wanted to test the hypothesis that, as already proposed by us in studies with the DST and the TRH-ST, sleep-EEG patterns in BPD without Major Depression patients and MD patients would be different, therefore not giving arguments favouring the existence of a biological link between BPD and the MD nor between the substrates of the depressive symptoms in BPD and non-borderline MD.

2. Methods

2.1. Subjects

Twenty in-patients (14 women and six men, mean age = 32.40; range 21–45) who met DSM-III-R criteria for BPD (American Psychiatric Association, 1987) as established in a clinical interview and with a score of at least 7 on the Diagnostic Interview for Borderlines (DIB; Gunderson et al., 1981) were recruited. All the patients were also assessed with the Schedule for Affective Disorders and Schizophrenia, lifetime version (SADS-L; Spitzer and Endicott, 1978). The reasons for their hospitalisation were as follows: self-mutilation (n = 6); chronic feelings of dysphoria (n = 5); suicide attempts with chemical compounds (n = 3); acute poisoning with chemical compounds without suicidal intent (n = 3); acute psychotic episode (n = 2), and pathological gambling (n = 1).

Twenty in-patients with MD, who were matched to the BPD patients for age and sex, were also recruited (15 women and five men, mean age = 35.85; range 26–43). Diagnoses of depression in this group were made with DSM-III-R criteria (clinical interview) and Research Diagnostic Criteria (SADS-L interview). All patients in this group met both DSM-III-R and Research Diagnostic Criteria for MD.

Exclusion criteria were as follows: other current DSM-III-R Axis I disorders (clinical interview) in both groups with special attention paid to the presence of MD in the BPD group (also assessed with the SADS-L); a DSM-III-R and DIB diagnosis of BPD in the MD group; presence of general medical or neurological disorders as evidenced by medical history, physical examination, standard laboratory blood tests, thyroid panel baseline TSH, T3, and T4, electrocardiogram; inability to give up consumption of alcohol or psychoactive substances before the study; and suspected poor treatment compliance.

Twenty age-matched control subjects (14 women and six men, mean age = 35.45; range 24–44) were free of somatic and psychiatric pathology as assessed by the SADS-L.

The 40 patients underwent a psychotropic drug washout period for at least 10 days before the sleep recordings were made (15 days for tricyclic antidepressants and monoamine oxidase inhibitor agents). Eighteen BPD patients were withdrawn from benzodiazepines (n = 6); amitriptyline (n =
phenelzine ($n = 1$); alcohol ($n = 3$); alcohol plus benzodiazepines ($n = 4$); and alcohol plus benzodiazepines plus barbiturates ($n = 2$). No BPD patient had taken neuroleptics in the 2 months before the study. The washout period was strictly controlled by regular plasma monitoring of drugs. All 40 patients were assessed with the 24-item Hamilton Depression Rating Scale (HDRS) on the same day as the sleep recordings. After complete description of the study to all subjects, informed consent was obtained.

2.2. Sleep laboratory

Each subject spent at least two consecutive nights in the sleep laboratory. The first recording night was used for adaptation to the laboratory procedures and environment. Sleep data analyses relate to the last recording night. The procedure for sleep recording and the definition of sleep variables are as described by Kerkhofs et al. (1985). REM latency denotes the minutes elapsed from the first stage 1 period until the first REM epoch. REM activity and density were not recorded. Percentage of wakefulness is the ratio of waking after the onset of sleep to the sleep period time. The presence of a primary sleep disorder such as sleep apnea or myoclonus was investigated (oxymetry, plethysmography and multi-electrode placement for evaluation of muscular activity).

Sleep-EEG measures, HDRS, lifetime diagnoses of major depression, drug and/or alcohol abuse, and suicide attempts were compared between the groups. Within the BPD group, a further analysis comparing the sleep-EEG variables in the BPD patients with ($n = 9$) versus the BPD patients without ($n = 11$) a lifetime history of major depression was also performed.

Between-group comparisons involving continuous data were calculated using parametric one-way analysis of variance (ANOVA) and post hoc two-tailed Student’s $t$-test for independent samples. Between-group comparisons involving categorical data were estimated using a chi-square ($\chi^2$) test.

3. Results

Tables 1–3 present clinical and EEG-sleep characteristics of the sample. All the patients in the three groups were free of primary sleep disorder. Two MD patients had past histories of BPD not confirmed by the DIB or by DSM-III-R. BPD patients showed a greater lifetime prevalence of drug or alcohol abuse and of suicide attempts than MD patients. MD patients had significantly higher scores on the HDRS. The lifetime prevalence of major depression was not statistically different in the two groups of patients. The clinically observed depressive symptoms in the BPD group differed from those in the MD group. Emptiness, loneliness, desperation in rela-

### Table 1

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>BPD</th>
<th>MD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>32.4 ± 6.90</td>
<td>35.85 ± 4.39</td>
</tr>
<tr>
<td>Sex (women)</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>HDRS</td>
<td>28.35 ± 8.36</td>
<td>33.75 ± 4.25*</td>
</tr>
<tr>
<td>Lifetime prevalence of major depression ($n =$)</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Lifetime prevalence of drug/alcohol abuse ($n =$)</td>
<td>11**</td>
<td>1</td>
</tr>
<tr>
<td>Lifetime prevalence of suicide attempts ($n =$)</td>
<td>15***</td>
<td>6</td>
</tr>
</tbody>
</table>

BPD, borderline personality disorder; MD, major depression; HDRS, Hamilton Depression Rating Scale.

*MD > BPD, two-tailed t-test for independent samples, $P = 0.014$.

**BPD > MD, $\chi^2$ test = 0.1, $P = 0.001$.

***BPD > MD, $\chi^2$ test = 6.4, $P = 0.01$. 
4. Discussion

Several differences in sleep-EEG patterns were

Table 3
Selected clinical and sleep EEG measures in 20 BPD with (+) and without (−) a lifetime history of MD

<table>
<thead>
<tr>
<th></th>
<th>BPD (+) n = 9</th>
<th>BPD (−) n = 11</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.66 ± 6.48</td>
<td>30.54 ± 6.96</td>
</tr>
<tr>
<td>Sex (women)</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>HDRS</td>
<td>25.55 ± 9.93</td>
<td>30.63 ± 6.42</td>
</tr>
<tr>
<td>Lifetime prevalence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Of drug/alcohol abuse</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>372.89 ± 71.12</td>
<td>347.99 ± 51.08</td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>29.14 ± 30.80</td>
<td>41.51 ± 42.55</td>
</tr>
<tr>
<td>Wakefulness (%)</td>
<td>18.53 ± 10.60</td>
<td>8.88 ± 4.72</td>
</tr>
<tr>
<td>Stage 1 (%)</td>
<td>6.94 ± 2.90</td>
<td>61.41 ± 31.91</td>
</tr>
<tr>
<td>Stage 2 (%)</td>
<td>48.32 ± 6.86</td>
<td>46.59 ± 12.13</td>
</tr>
<tr>
<td>Stage 3 (%)</td>
<td>2.24 ± 2.87</td>
<td>4.11 ± 2.14</td>
</tr>
<tr>
<td>Stage 4 (%)</td>
<td>0.61 ± 1.59</td>
<td>3.70 ± 4.88</td>
</tr>
<tr>
<td>Slow wave sleep (%)</td>
<td>2.85 ± 4.18</td>
<td>9.29 ± 7.00</td>
</tr>
<tr>
<td>REM duration (%)</td>
<td>23.33 ± 7.70</td>
<td>27.63 ± 10.95</td>
</tr>
<tr>
<td>REM latency (min)</td>
<td>63.96 ± 28.12</td>
<td>61.41 ± 31.91</td>
</tr>
</tbody>
</table>

Two-tailed t-test for independent samples (mean ± S.D.). BPD, borderline personality disorder; MD, major depression; BPD (+), patients with a lifetime prevalence of major depression; BPD (−), patients without a lifetime prevalence of major depression; HDRS, Hamilton Depression Rating Scale; min, minutes. Significant between-group differences were: *P < 0.05, **P < 0.01, ***P ≤ 0.001. Significant (P < 0.05) post hoc pairwise t-tests for independent samples were: a, BPD ≠ MD; b, BPD ≠ CTRL; c, MD ≠ CTRL.

Table 2
Selected sleep EEG measures in 20 age- and sex-matched BPD, MD and control subjects

<table>
<thead>
<tr>
<th></th>
<th>BPD</th>
<th>MD</th>
<th>CTRL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time (min)</td>
<td>359.20 ± 60.53</td>
<td>343.55 ± 99.03</td>
<td>407.20 ± 29.11</td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>35.95 ± 37.31</td>
<td>28.45 ± 25.81</td>
<td>12.81 ± 10.63</td>
</tr>
<tr>
<td>Wakefulness (%)</td>
<td>13.22 ± 9.12</td>
<td>17.79 ± 16.41</td>
<td>7.95 ± 5.31</td>
</tr>
<tr>
<td>Stage 1 (%)</td>
<td>7.55 ± 3.37</td>
<td>10.16 ± 4.86</td>
<td>9.02 ± 4.82</td>
</tr>
<tr>
<td>Stage 2 (%)</td>
<td>47.41 ± 9.76</td>
<td>38.73 ± 11.45</td>
<td>48.50 ± 9.02</td>
</tr>
<tr>
<td>Stage 3 (%)</td>
<td>3.23 ± 2.62</td>
<td>7.66 ± 4.79</td>
<td>8.06 ± 3.75</td>
</tr>
<tr>
<td>Stage 4 (%)</td>
<td>2.23 ± 3.94</td>
<td>6.81 ± 7.66</td>
<td>6.99 ± 7.02</td>
</tr>
<tr>
<td>Slow wave sleep (%)</td>
<td>6.39 ± 6.63</td>
<td>15.35 ± 11.20</td>
<td>15.33 ± 9.20</td>
</tr>
<tr>
<td>REM duration (%)</td>
<td>25.69 ± 9.64</td>
<td>18.97 ± 8.10</td>
<td>19.64 ± 4.98</td>
</tr>
<tr>
<td>REM latency (min)</td>
<td>62.58 ± 29.50</td>
<td>58.16 ± 56.11</td>
<td>72.59 ± 34.04</td>
</tr>
</tbody>
</table>

Parametric one-way ANOVA and post hoc two-tailed t-test for independent samples (mean ± S.D.). BPD, borderline personality disorder; MD, major depression; CTRL, controls; min, minutes. Significant between-group differences were: *P < 0.05, **P < 0.01, ***P ≤ 0.001. Significant (P < 0.05) post hoc pairwise t-tests for independent samples were: a, BPD ≠ MD; b, BPD ≠ CTRL; c, MD ≠ CTRL.

Other sleep parameters, age, sex and HDRS scores showed no statistically significant differences between the two BPD subgroups.
found between non-depressed BPD patients and MD patients. The parameters of continuity TST, SOL, and percentage of wakefulness differentiated both BPD and MD patients from controls. Sleep-architecture parameters clearly differentiated BPD from MD patients. BPD patients and controls had more stage 2 than MD patients. BPD patients had less SWS and longer REM sleep duration than MD patients and controls. The differences in delta sleep between BPD and MD patients are of interest because reduced delta sleep is thought to be a biological feature of MD (Mendels and Hawkins, 1967; Kupfer et al., 1985; Reynolds et al., 1990). The diminished SWS in BPD without Major Depression patients is not an artifact due to age or sex, since our patients were relatively young and the three groups were age- and sex-matched. Conversely, this finding could be related to the significantly higher prevalence of alcohol abuse in the BPD group, since alcohol abuse has been associated with decreased SWS (Lester et al., 1973), even 2 years after detoxification (Adamson and Burdick, 1973). A diminution in SWS has also been described in schizophrenia (Caldwell and Domino, 1967; Feinberg et al., 1969). An alternative possibility to explain the diminution in SWS in our BPD patients is that it could parallel BPD psychotic symptoms, as one of the classic hypotheses on the pathophysiology of BPD involves schizophrenia, schizoaffective psychoses, and atypical psychoses (Gunderson, 1979; Schulz et al., 1989).

REM latency did not differentiate the three groups. The small sample sizes and group variability in REM latencies do not allow us to exclude a type II error here. We must note that the mean REM latency in our BPD and MD patients was less than 65 min compared with a mean of 73 min in controls. The limit of 65 min has been reported to have a specificity of 90–93% and a sensitivity of 35–68% in differentiating MD patients from controls (Giles et al., 1987). If we assume these data to be correct, our BPD patients would belong to the 7–10% of no Major Depression persons with a false-positive finding of shortened REM latency. The increased REM duration that we found in our BPD patients has also been reported in MD (Kupfer et al., 1978; Kerkhofs et al., 1985).

When a further analysis comparing sleep-EEG variables in the group of BPD patients with \( n = 9 \) versus the group of BPD patients without \( n = 11 \) a lifetime prevalence of MD was performed (Table 3), the BPD patients with a history of MD had a greater percentage of wakefulness and less SWS than the BPD patients without such a history; shortened REM latency did not differentiate the two subgroups, however. This was not the case in the study by Benson et al. (1990) in which present or past affective and non-affective BPD patients had similar delta sleep values. In our groups, lifetime prevalence of drug/alcohol abuse and sex did not account for the differences in SWS. The BPD patients with a history of MD were slightly older \( 34.66 \pm 6.48 \) vs. \( 30.54 \pm 6.96 \) years). This small difference in age might have accounted for the SWS difference. Alternatively, the lower SWS in BPD patients with a history of MD could be an intrinsic characteristic of this subgroup of BPD patients.

Our data are in contrast with earlier reported findings of shortened REM latency in without Major Depression BPD patients versus controls and with those reporting persistently shortened REM latency in remitted patients after recovery from MD (Cartwright, 1983; Rush et al., 1991). The present data are in accord with those of Benson et al. (1990), who showed the REM latencies of BPD patients to be similar to those of controls, and the REM latencies of BPD patients with and without a history of affective disorder to be indistinguishable. Like Benson et al., we also found diminished TST and SWS in BPD versus controls.

As we have already reported using the DST and the TRH-ST (De la Fuente and Mendlewicz, 1996), our results do not offer support for a biological link between BPD and MD on the basis of the tests now studied, even if our BPD patients had high HDRS scores. The depressive symptoms experienced by our BPD patients are different in quality from, and may not share the biological substrate of, those found in non-BPD MD. The present study offers more arguments favouring the earlier suggested concept (Westen et al., 1992;
Korzekwa et al., 1991; Rogers et al., 1984; Siever et al., 1986; Korzekwa et al., 1991, 1993; Kavoussi et al., 1993; De la Fuente and Mendlewicz 1996) that BPD patients with depressive symptoms often suffer from an affective syndrome different from non-borderline MD not only in terms of quality and duration of symptoms but also in terms of biological substrate. This distinct affective syndrome is neither characterised by the endocrine alterations that accompany MD nor by the reduced REM latency. Perhaps it is accompanied by other alterations, such as shortened TST and SWS, increased SOL and percentage of wakefulness, increased REM duration with normal REM latency, and normal DST and TRH-ST, which could be more intrinsically related to the BPD syndrome. Also, reduced function of the central serotonergic system could accompany BPD (Coccaro et al., 1994) and parallel the more chronic symptoms such as self-destructiveness, impulsiveness and disturbed personal relationships.

On the other hand, and apart from the distinctive form of affective disorder often presented by BPD patients, it is clear from clinical practice that BPD patients can have MD. In this regard, Gunderson and Phillips (1991) have proposed that the two disorders may coexist but are not etiologically related.

Although our BPD patients did not have current MD and their HDRS scores were significantly lower than those of MD patients, their mean HDRS score was nevertheless somewhat elevated (28.35 ± 8.36). This fact may have several interpretations: BPD patients often present with subjective dysphoric complaints and relatively few vegetative symptoms to meet criteria for MD (Korzekwa et al., 1991); they could exaggerate their depressive complaints (Korzekwa et al., 1993); and BPD-related depressive symptoms are often atypical, not qualifying for a diagnosis of MD but leading to high scores on depression scales (Rogers et al., 1995). Moreover, the most frequently used tools for depression evaluation have been suggested not to be satisfactory to assess depression in BPD (Soloff et al., 1987; Korzekwa et al., 1991).

In 12 of our BPD patients, a diagnosis of recurrent brief depression (RBD) (Angst et al., 1990) could be established. If BPD patients do not share the biological alterations of MD and frequently display an affective syndrome distinct from that characteristic of non-borderline MD, it might be hypothesized that this alternative form of affective disorder could be RBD, as proposed by Montgomery (1991). However, patients with recurrent brief depression have findings similar to those reported for MD patients on the DST, TRH-ST and sleep-EEG (Staner et al., 1992), and BPD patients have shown biological similarities with MD patients in some studies (Akiskal et al., 1985; Battaglia et al., 1993) but not in others (Soloff et al., 1982; Nathan et al., 1986; Siever et al., 1986; Lahmeyer et al., 1988; Benson et al., 1990; Korzekwa et al., 1991; De la Fuente and Mendlewicz 1996). These contradictory results on the phenomenological and biological relations between BPD and recurrent brief depression, along with the consolidation and characterisation of the biological alterations in non-MD BPD patients, warrant further research.

References


Rush, A.J., Gullion, C.M., Armitage, R., Roffwarg, H.P., 1991. The polysomnogram as a vulnerability marker for depres-