Critical Evaluation of the Effect of Valerian Extract on Sleep Structure and Sleep Quality

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A carefully designed study assessed the short-term (single dose) and long-term (14 days with multiple dosage) effects of a valerian extract on both objective and subjective sleep parameters. The investigation was performed as a randomised, double-blind, placebo-controlled, cross-over study. Sixteen patients (4 male, 12 female) with previously established psychophysiological insomnia (ICSD-code 1.A.1), and with a median age of 49 (range: 22 to 55), were included in the study. The main inclusion criteria were reported primary insomnia according to ICSD criteria, which was confirmed by polysomnographic recording, and the absence of acute diseases. During the study, the patients underwent 8 polysomnographic recordings: i.e., 2 recordings (baseline and study night) at each time point at which the short and long-term effects of placebo and valerian were tested. The target variable of the study was sleep efficiency. Other parameters describing objective sleep structure were the usual features of sleep-stage analysis, based on the rules of Rechtschaffen and Kales (1968), and the arousal index (scored according to ASDA criteria, 1992) as a sleep microstructure parameter. Subjective parameters such as sleep quality, morning feeling, daytime performance, subjectively perceived duration of sleep latency, and sleep period time were assessed by means of questionnaires. After a single dose of valerian, no effects on sleep structure and subjective sleep assessment were observed. After multiple-dose treatment, sleep efficiency showed a significant increase for both the placebo and the valerian condition in comparison with baseline polysomnography. We confirmed significant differences between valerian and placebo for parameters describing slow-wave sleep. In comparison with the placebo, slow-wave sleep latency was reduced after administration of valerian (21.3 vs. 13.5 min respectively, p<0.05). The SWS percentage of time in bed (TIB) was increased after long-term valerian treatment, in comparison to baseline (9.8 vs. 8.1% respectively, p<0.05). At the same time point, a tendency for shorter subjective sleep latency, as well as a higher correlation coefficient between subjective and objective sleep latencies, were observed under valerian treatment. Other improvements in sleep structure – such as an increase in REM percentage and a decrease in NREM1 percentage – took place simultaneously under placebo and valerian treatment. A remarkable finding of the study was the extremely low number of adverse events during the valerian treatment periods (3 vs. 18 in the placebo period). In conclusion, treatment with a herbal extract of radix valerianae demonstrated positive effects on sleep structure and sleep perception of insomnia patients, and can therefore be recommended for the treatment of patients with mild psychophysiological insomnia.

Introduction

The prevalence of sleep disturbances is between 20 and 40% of the population in the Western European countries; it increases with age. About 40% of these patients use hypnotics occasionally, and 4 to 6% of the patients receive daily doses (Hajak and Rüther, 1995). The effect of hypnotics, however, is not stable: epidemiological investigations have shown that sleep disturbances persist in 40 to 50% of the treated patients (Holzrichter et al., 1994). The most widely used substances for the treatment of sleep disturbances are benzodiazepines and benzodiazepine receptor agonists, such as zopiclone and zolpideme. An advantage of these substances is their fast and reliable induction of sleep. However, they do not restore the normal sleep macrostructure: studies have determined reduced SWS sleep, often accompanied by REM sleep reduction, under treatment with benzodiazepines (Borbély, 1986). The chief disadvantages of benzodiazepines are the appearance of hangover effects, drug tolerance, rebound insomnia after withdrawal, and the risk of addiction (Laux, 1995). Although benzodiazepine-receptor agonists have shorter half-lives than most benzodiazepines, similar side effects have likewise been observed in these substances (Noble et al., 1998). Long-term treatment of sleep disturbances with benzodiazepines and benzodiazepine receptor agonists is therefore inadvisable due to these side effects.

Other drugs suitable for the treatment of sleep disorders are antidepressants, antihistamines, and low-potency neuroleptics with a sedative effect. Together with varying negative effects on the proportion of REM and NREM sleep stages, they may give rise to many typical side effects. The indications to administer these drugs are therefore mainly sleep disturbances accompanied by corresponding psychiatric symptoms or parasomnia.

Natural remedies prepared from plants – such as extracts and teas from valerian, melissa, hops, and St. John’s wort – are
The doses of valerian root used in these studies ranged from 400 mg/die with a dose/extract ratio (DER) of 3:1 (Leathwood et al., 1982, Balderer and Borbély, 1985) to 1215 mg/die with a DER of 5 to 6:1 (Schulz et al., 1994). Although these studies disclosed moderate changes under valerian treatment, the effects could well have proved more clearly developed if treatment had been studied for sleep-disturbed patients with application of doses toward the upper limit of this dosage range.

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The aim of the present study was therefore to evaluate the short and long-term effects of valerian on the sleep structure and subjective sleep quality of insomniacs in a placebo-controlled study design. In addition to the usual polysomnographic parameters of sleep macrostructure, we established parameters for sleep microstructure – such as the number of arousals and arousal index – and analysed them over the course of the treatment periods.

**Methods**

**Patients**

The study included sixteen patients – 12 female and 4 male – suffering from psychophysiological insomnia (ICSD code I.A.1.). All patients signed written consent to participate in the study. The diagnosis of insomnia was established on the base of subjective complaints, medical history, medical examination, and the results from control polysomnography throughout one night. The patients’ subjective sleep disturbances had lasted from three months to several years. Four patients suffered from sleep-onset difficulties, 6 patients reported difficulties maintaining sleep, and 6 patients showed both subjective sleep-onset and sleep-maintaining problems. None of the patients enrolled in the study demonstrated an RDI > 5/h, periodic limb movements, or restless-legs syndrome during the control night.

The patients manifested normal states of health appropriate to their age and did not suffer from organic or psychiatric diseases which could cause sleep disturbances. Such diseases were excluded on the base of their actual clinical status and corresponding laboratory investigations. Patients were not allowed to take any drugs influencing sleep structure and daytime vigilance from a period beginning 14 days before the start of the study until its conclusion. All demonstrated negative screening results for psychotropic drugs, including alcohol, cocaine, benzodiazepines, barbiturates, etc.

Median patient age was 49 (22 to 56), and only 4 patients were younger than 40. The control night revealed the following objective parameters (given in median and quartiles):

- Sleep efficiency (TST/TIB) = 77.8 % (24.3 to 90.4)
- Waking during the sleep period = 15.4 % (5.2 to 68.5)
- Sleep-onset latency = 27.5 min (7.5 to 71).

**Study protocol**

The study was designed with a randomised, double-blind, placebo-controlled, cross-over protocol. The patients underwent nine polysomnographic nights (Fig. 1): 1 control night before and 8 nights during the study. On the basis of the control night, patients with sleep apnoea syndrome (RDI > 5/h), periodic limb movements, or restless-legs syndrome were excluded from the study. The 8 study nights were scheduled in two trial periods, separated by a washout period of 13 days.

![Fig. 1](image-url)  
**Fig. 1** Study design. B 1 – B 4: baseline/adaptation PSG; S 1/S 3: short-term treatment PSG; S 2/S 4: long-term treatment PSG.
Each trial period lasted 15 days, including 4 polysomnographic nights:

Day 1: baseline PSG under placebo
Day 2: short-term treatment PSG,
Days 3 to 13: treatment at home
Day 14: baseline PSG under treatment
Day 15: long-term treatment PSG

The conduct of baseline polysomnographic recordings served to adapt patients to sleep laboratory conditions; therefore, the data from these nights were not used as references to correct for the effect of time. The patients received placebo or valerian in the first trial period (day 2 to day 15) in a randomised double-blind procedure, and subsequently switched to the alternative treatment in the second trial period.

Drug

According to the study design, the patients received either valerian (as Sedonium®) or the placebo. A single coated tablet of Sedonium® contains 300 mg dry extract of radix valerianae with a drug/extract ratio (DER) of 5:1. During the trial periods, the patients were instructed to take two pills 1 hour before bedtime, which is equivalent to a daily dose of 600 mg valerian root extract. Since the recommended doses of natural valerian drug for the treatment of sleep disturbances lie between 1 g and 3 g/die (Bundesanzeiger, 1995), the DER involved in the present study meant that the treatment chosen here represents the high end of the dose range (3 g/die).

Objective parameters of sleep structure

Polysomnographic recordings included 4 EEG, 2 EOG, 2 EMG, and 1 ECG leads during the trial periods and were performed from 10:00 p.m. until 6:00 a.m. Only during the control night were respiratory parameters recorded such as snoring, nasal flow, oxygen saturation, and thoracic and abdominal efforts. Sleep stages were visually scored by one qualified rater according to the criteria of Rechtschaffen and Kales (1968). In addition, visual scoring of arousals based on the ASDA report criteria (1992) took place. Ratings were supervised and randomly verified by a qualified assistant.

On the base of these data, the following parameters were used to describe objective changes in sleep structure: sleep efficiency (total sleep time/time in bed × 100); sleep period time (SPT); sleep-onset latency (SOL); SWS and REM latency; percentages of NREM 1, NREM 2, REM, and SWS sleep stages on the basis of total time in bed (TIB); and arousal index (ARI, as number of arousals per hour of the total sleep time).

Subjective parameters of sleep quality

To assess the subjective quality of sleep, a structured questionnaire was filled in by the patients in the evening and the morning, before and after each polysomnographic recording. It contained visual analogue scales (VAS) for the following factors: sleep quality, morning feeling, and daytime performance. The questionnaire also asked for entry of the subjectively perceived duration of sleep latency and sleep-period time. These items were included as subjective parameters in the study.

Apart from that, the questionnaire included questions about dreams; subjective sleep disturbances; daytime activities; intake of drugs, caffeine, and nicotine during the day; and the time of consumption of these substances. These data were used for assessment of patients’ compliance, and as criteria for exclusion of volunteers from the study.

Statistics

The target variable of the study was objective sleep efficiency. The absence of differences between the sleep efficiencies of the placebo and the valerian condition after long-term treatment on day 15 (long-term treatment PSG) was postulated as null hypothesis, at an α-level = 0.05, two-sided. The distributions of all objective and subjective parameters were described with median, first, and third quartiles for all nights, respectively. The Wilcoxon test for paired samples was used to check on the differences between valerian and placebo effects separately, for singular and multiple dose treatments. To describe both placebo and valerian effects, the data from the long-term treatment nights were compared with the values from the first baseline night during the study, also on the basis of the Wilcoxon test for paired samples. To exclude an effect from the cross-over study design, the differences of the valerian and placebo values (valerian minus placebo) from the two cross-over groups were subjected to the Mann-Whitney-U test.

No α-adjustment was performed, with the result that all tests except the target variable were required to be interpreted on a descriptive level.

The investigation was approved by the local Ethics Committee of the Charité University Medical Centre.

Results

Effect of time and repeated measurements

Upon comparison of the baseline placebo nights of the two trial periods (B1 and B3, see Fig. 1), none of the objective parameters showed a significant difference. Sleep efficiency amounted to 80.6% (71.7 to 87.7) in the first and 86.2% (79.2 to 89.3) in the second trial period, while objective sleep onset time was 23.5 (13.9 to 36.3) min in the first and 18.0 (12.1 to 33.5) min in the second trial period. Among the subjective parameters, only subjective sleep latency was significantly prolonged (p < 0.05), and amounted to 60 (30 to 90) min in the first study period versus 85 (55 to 128) min in the second period.

The two cross-over groups demonstrated significant differences in the main parameters of sleep structure. The differences between the valerian and placebo treatment effects of the two cross-over groups (scheduled valerian-placebo versus scheduled placebo-valerian) were therefore tested for every objective and subjective parameter. None of these measured data revealed significant changes as a result of the schedule of administering valerian and placebo (Fig. 4).

Valerian versus placebo

The median, first, and third quartile of all objective and subjective parameters are listed in Table 1 for the baseline of the first study period, for short and long-term treatment nights.
After a single dose of valerian, no significant effect on these parameters was observed.

Sleep efficiency as target parameter of the study showed a continuous increase from baseline PSG – throughout short-term treatment to long-term treatment – with results similar for valerian and for placebo. Comparison of baseline PSG and long-term treatment disclosed that sleep efficiency was significantly higher in both groups at the end of the trial periods. However, sleep efficiency did not show any significant differences between placebo and verum groups after long-term treatment.

Significant changes were confirmed for slow-wave sleep (NREM 3 + 4, SWS). SWS latency was reduced in the verum group after long-term treatment (Table 1, p < 0.05). The median of SWS percentage under long-term treatment was higher in the verum group than in the placebo group, and significant increase in the SWS percentage in comparison with baseline was apparent only for the verum condition.

Certain trends, similar for placebo and valerian treatments, became apparent upon comparison of the baseline of the first trial period with the results after 2 weeks of treatment. SPT and REM sleep percentage increased, while NREM 1 percentage decreased (Fig. 2).

The arousal index as a parameter of sleep microstructure amounted to 15.3 (10.8 to 17.8) per hour of sleep time (/h) and did not change after long-term placebo application – 13.7 (12.6 to 19.6)/h – or after verum treatment – 14.9 (13.6 to 19.9)/h.

A tendency towards reduced subjective sleep latency after long-term verum application became apparent (Table 1). Ten of the 16 patients had a lower subjective sleep latency under valerian treatment than under placebo, despite the fact that the objective sleep-onset latency under verum was not different from the latency under placebo. An analysis of the relationship between the objective and subjective sleep latencies revealed differences between valerian and placebo effects. Whereas a correlation coefficient of $r = 0.56$ (p < 0.05) was calculated for the values under placebo treatment, the coefficient after valerian treatment amounted to $r = 0.75$ (p < 0.001). See Fig. 3 for this relationship.

The other subjective parameters – subjective sleep time, sleep quality, morning feeling and daytime performance – remained unchanged during this study.

### Adverse events and side effects

During the study, lasting 44 days for each of the 16 patients, only 21 adverse effects with a duration of 1 to 3 days were observed. The majority of these adverse events occurred under placebo or during washout after placebo (Table 2). Only one attack of a previously known migraine, one episode of gastrointestinal complaints, and one complaint as a consequence of polysomnography (an accident) were seen under valerian treatment. No adverse event was observed during the washout period after valerian.

### Table 1  Objective and subjective sleep parameters in the first baseline and treatment nights. Median, first, and third quartiles are listed for each parameter

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Baseline</th>
<th>Placebo short-term</th>
<th>Valerian short-term</th>
<th>Placebo long-term</th>
<th>Valerian long-term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep efficiency (%)</td>
<td>80.6 (71.7–87.7)</td>
<td>87.5 (82.5–90.9)</td>
<td>85.2 (80.3–90.5)</td>
<td>88.4 (80.7–93.1)</td>
<td>89.6 (79.7–93.5)</td>
</tr>
<tr>
<td>Sleep period time (min)</td>
<td>442.3 (406.0–457.4)</td>
<td>443.0 (430.1–459.4)</td>
<td>450.5 (437.3–458.0)</td>
<td>457.3 (437.4–468.6)</td>
<td>457.8 (430.5–467.9)</td>
</tr>
<tr>
<td>NREM 1 (%)</td>
<td>8.2 (5.4–10.2)</td>
<td>8.6 (6.5–11.1)</td>
<td>9.4 (6.8–13.7)</td>
<td>7.6 (5.8–9.4)</td>
<td>7.8 (5.8–10.7)</td>
</tr>
<tr>
<td>NREM 2 (%)</td>
<td>45.4 (40.6–54.5)</td>
<td>51.4 (44.1–55.1)</td>
<td>48.6 (45.8–54.3)</td>
<td>49.6 (46.1–54.8)</td>
<td>49.2 (44.2–53.6)</td>
</tr>
<tr>
<td>SWS (%)</td>
<td>8.1 (5.0–9.5)</td>
<td>11.0 (7.4–13.8)</td>
<td>9.6 (6.4–10.2)</td>
<td>9.2 (8.0–12.4)</td>
<td>9.8 (6.2–14.0)</td>
</tr>
<tr>
<td>REM (%)</td>
<td>16.9 (13.5–18.3)</td>
<td>17.5 (14.8–21.3)</td>
<td>16.9 (15.7–19.0)</td>
<td>18.9 (15.5–21.0)</td>
<td>19.0 (14.4–22.1)</td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>23.5 (13.9–36.3)</td>
<td>19.0 (13.3–22.1)</td>
<td>20.3 (12.5–25.8)</td>
<td>12.8 (7.1–34.0)</td>
<td>13.3 (6.9–25.0)</td>
</tr>
<tr>
<td>SWS latency (min)</td>
<td>15.5 (11.8–28.8)</td>
<td>12.5 (7.6–10.3)</td>
<td>16.0 (12.3–20.8)</td>
<td>21.3 (11.5–26.9)</td>
<td>13.5 (11.1–20.8)</td>
</tr>
<tr>
<td>REM latency (min)</td>
<td>80.3 (59.6–109.8)</td>
<td>71.8 (54.5–97.8)</td>
<td>61.3 (45.9–74.1)</td>
<td>73.6 (63.1–93.4)</td>
<td>69.5 (53.6–85.0)</td>
</tr>
<tr>
<td>Arousal index (arousal/h)</td>
<td>15.3 (10.8–17.8)</td>
<td>13.7 (12.6–19.6)</td>
<td>14.9 (13.6–19.9)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Subjective sleep latency (min)</td>
<td>60.0 (30.0–90.0)</td>
<td>52.5 (30.0–78.8)</td>
<td>37.5 (21.3–105.0)</td>
<td>60.0 (30.0–105.0)</td>
<td>45.0 (17.5–75.0)</td>
</tr>
<tr>
<td>Subjective sleep quality (%)</td>
<td>33.0 (26.8–44.0)</td>
<td>41.0 (21.0–62.0)</td>
<td>29.5 (19.8–56.5)</td>
<td>47.5 (36.0–73.3)</td>
<td>43.0 (28.8–70.8)</td>
</tr>
<tr>
<td>Morning feeling (%)</td>
<td>47.2 (43.6–56.5)</td>
<td>60.8 (36.9–68.3)</td>
<td>47.5 (30.4–81.9)</td>
<td>53.5 (46.3–71.0)</td>
<td>55.8 (42.0–70.4)</td>
</tr>
<tr>
<td>Daytime performance (%)</td>
<td>58.0 (41.5–67.5)</td>
<td>47.0 (35.8–69.3)</td>
<td>48.5 (37.0–71.0)</td>
<td>65.0 (34.5–79.0)</td>
<td>65.0 (45.0–68.5)</td>
</tr>
</tbody>
</table>
Discussion

The population of patients with psychophysiological insomnia is very heterogeneous with respect to objective polysomnographic parameters (Reite et al., 1995). The main explanations for the interindividual differences in recorded sleep parameters are the various forms (sleep-onset or sleep-maintaining difficulties) and the various extents of sleep disturbance found in patients with psychophysiological insomnia. In the present study, these differences became apparent in the great ranges of the main sleep parameters for the baseline night (Table 1). They did not correlate with age, gender, or duration of sleep disturbance. To minimise the influence of this considerable interindividual variance of sleep parameters on test statistics, it was necessary to carry out the study according to a meticulous placebo-controlled, cross-over design. Although the cross-over design required a long study-time interval (6 weeks and 9 polysomnographic recordings for each patient), it was possible to exclude effects caused by time or repeated measurements: no differences between the two patient groups with different medication schedule were found on comparing the differences between valerian and placebo values. No significant improvement in any objective or subjective parameter, and only a slight prolongation of subjective sleep latency, became apparent in comparison to the baseline nights of the two trial periods (which were separated by 4 weeks and 3 other PSG recordings). This result demonstrates the intraindividual stability of our patients’ sleep parameters and complaints during periods without treatment.

Upon analysis of the results for the target variable of sleep efficiency, the primary finding of the study initially appears to be the absence of a difference between the placebo and valerian treatment. However, sleep efficiency and a number of other parameters - such as sleep-period time, subjective sleep latency, NREM 1, and REM percentage - changed simultaneously under placebo and valerian after 2 weeks of treatment and marked an improvement of sleep structure compared to
We may thus assume a positive effect of valerian on slow-wave sleep for insomnia patients. Despite almost identical objective sleep parameters such as baseline PSG. Since there was no statistically confirmed influence of the course of time or repeated polysomnograms on the sleep parameters, these trends must be interpreted as placebo effects. These placebo effects consist not only of the influence of assumed drug intake but also of influences in the form of attention paid to the patients in such a study design. Especially in patients with psychophysiological insomnia, every instruction regarding a regular sleep schedule, daytime sleep restriction, and caffeine abstinence represents an important factor supporting the therapy of the sleep disorder. The effect of participation in the study as a patient undergoing non-pharmacological treatment might accordingly have been greater than the effect of the drug treatment, and the existing differences may have been masked by the effect of non-pharmacological therapy. Similar results have been shown by Morin et al. (1998), who found no significant improvement of sleep efficiency comparing placebo and temazepam in a controlled clinical trial with patients suffering from primary insomnia.

In comparison with placebo administration, various effects of valerian have been observed primarily after long-term treatment. Slow-wave sleep latency was significantly shorter after valerian than after placebo treatment, with the result that SWS was shifted to the beginning of the sleep period. Simultaneously, the SWS percentage increased slightly under long-term valerian treatment compared to baseline. These two parameters may indicate a reconstruction of slow-wave sleep, with a shift of slow-wave-sleep back to its proper physiological place in the sleep profile. This result supports the observations of Schulz et al. (1994), who determined a slight increase in SWS percentage after 7 days of valerian treatment.

We may thus assume a positive effect of valerian on slow-wave sleep. Since slow-wave sleep plays an essential role in physical recovery (Reite et al., 1995), this finding could prove important for insomnia patients. Despite almost identical objective sleep-onset latencies, subjective sleep latencies for the majority of the patients were lower under valerian than under placebo treatment, and the correlation between the subjective and objective sleep latencies was greater after long-term treatment with valerian. These data indicate a reduction in the misperception of sleep and wake phases which often results in an overestimation of wake phases in patients with psychophysiological insomnia. The more realistic estimation of sleep latency is conceivably associated with reduced SWS latency, and may lead to shorter subjective sleep latency in patients who show a disturbed SWS profile before the treatment.

Short-term treatment with valerian did not reveal any effect on sleep structure and subjective parameters: the values were within the normal range of night-to-night variability found among insomniacs (Reite et al., 1995). The same was observed by Schulz et al. (1994) in poor sleepers after one single dose of 405 mg valerian extract. With regard to subjective parameters, Leathwood and co-workers (1982, 1985) determined an improvement in subjective sleep latencies and subjective sleep quality in 43% and 54% of poor sleepers after a single dose of 400 mg valerian extract, respectively. These data confirm that the majority of poor sleepers and insomniacs will not demonstrate an immediate effect of valerian treatment.

The mechanism of the mildly sedative effect of valerian after multiple dose application is still not clear. Some authors found changes in the activity of GABA neurons (Santos et al., 1994), while others discuss the possibility of an effect on melatonin secretion (Rödemeck et al., 1998). The clinical effect of valerian possibly results owing to the combined action of several substances in valerian root extract on different receptors. The absence of a short-term effect could be explained by the pharmacokinetic distribution characteristics of the active substances. The following may be supposed: slow distribution to the effector site and a slow increase in concentration at this site, with the expected effect only after a lengthy period. Another explanation for the observed delay in the valerian effect could be adaptation processes at the receptor site which modulate the signal transduction of neurotransmitters.

For the first time, this study establishes and analyses the number of arousals and the arousal index in patients with psychophysiological insomnia during the course of valerian treatment. A baseline level of 15.3 (10.8 to 17.8) arousals per hour of total sleep time was observed, which did not change significantly throughout the course of the study. Mathur and Douglas (1995) established a median arousal index of 21/h (CI 95% 7 to 36/h) for healthy volunteers of varying ages (20 to 70) in a first-night polysomnographic investigation, while Boselli et al. (1998) determined a mean arousal index of 17.8/h in a group of middle-aged (40 to 59) healthy volunteers. Comparison of these results with our data indicates that the extent of arousal among middle-aged patients with psychophysiological insomnia is apparently the same as in normal sleepers. Since, furthermore, the extent of arousal did not change after the 14 days of treatment, this finding may lead to the conclusion that the arousal index is a distinguishing feature of individual sleep microstructure rather than a marker of disturbed sleep in insomniacs.

In comparison to the immediate effects of other sleep-inducing substances such as benzodiazepines, the influence of valerian on sleep is slight and delayed. Valerian will therefore hardly prove effective for patients with acute, reactive sleep disturbances who need rapid recovery from their complaints. In treatment of patients with chronic insomnia, however, it is important to change the patient’s attitude to his sleep. For this process, active participation by the patient in non-pharmacological measures such as sleep hygiene, group therapy, psychotherapy, and training with relaxation exercises is indispensable. Slow and gradual development of the valerian effect can promote and maintain such an active contribution. Therapy based on a combination of non-pharmacological and herbal drug therapies will reduce to a minimum withdrawal effects of the nature of those caused by benzodiazepines. Schmitz and Jäckel (1998) have confirmed this possibility by comparing the withdrawal effects of a preparation based on valerian and hops with those of bromazepam in patients suffering from short-term insomnia. Since the valerian-root extract elicited no specific side effects – and since nonspecific side effects even diminished during the study – long-term application of valerian is evidently harmless. Moreover, the reduction of nonspecific complaints such as headaches and gastrointestinal complaints could well contribute to the overall benefits of valerian treatment.
In conclusion, treatment with a herbal extract of radix valerianae at relatively high dose levels demonstrated a number of positive effects on the sleep structure and sleep perception of insomniac patients. It can consequently be recommended as adjuvant therapy in the treatment of patients suffering from chronic psychophysiological insomnia.

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