

## Cognitive behavioral therapy for chronic insomnia

Małgorzata Fornal-Pawłowska, Waldemar Szelenberger

Medical University of Warsaw, Department of Psychiatry  
Head of Chair: prof. dr hab. med. Marcin Wojnar

### Summary

**Objectives.** To evaluate the efficacy of cognitive behavioral therapy (CBT-I) for chronic insomnia treatment.

**Method.** The 236 patients with ICD-10 nonorganic insomnia were assigned to group CBT-I (6 sessions, 6-10 patients). From this pool, 72 participants with no history of other psychiatric or sleep disorders conditions were selected. Eventually, 51 patients (40f, mean age: 54.6±13.9y, mean insomnia duration: 7±6.3y) and 51 matched healthy controls (mean age: 55.4±14.3y) completed the study. Outcomes in the insomnia group at baseline and post-treatment were compared to control group. Subjects underwent sleep diary, the Athens Insomnia Scale (AIS), the Beck Depression Inventory (BDI), the Ford Insomnia Response to Stress Test (FIRST), the SF-36 questionnaire and the State-Trait Anxiety Inventory (STAI).

**Results.** At baseline, groups differed significantly in most dependent variables. At post-treatment, a substantial improvement in all sleep parameters was observed in insomnia group: sleep latency, number of awakenings, wake time after sleep onset, sleep time, sleep efficiency, sleep quality and frequency of hypnotic use. These outcomes were accompanied by lower AIS and FIRST scores, reductions of depression and anxiety symptoms, and improved energy and social functioning ratings. All changes were maintained during the 3-month follow-up. Only 10/51 patients had no clinically meaningful improvement at any post-treatment time points. After the therapy, patients did not differ significantly from good sleepers in number of awakenings, sleep quality, feeling in the morning, depression and anxiety symptoms, and quality of life related to mental health.

**Conclusions.** The CBT-I produced a sustained, clinically meaningful improvement in nocturnal sleep and daytime functioning.

**Key words:** chronic insomnia, cognitive behavioral therapy, daytime functioning

### Introduction

Chronic insomnia – a disorder of unsatisfactory quantity and/or quality of sleep and daytime impairment related to it [1] - is a serious health problem affecting about 6% of the adult population [2]. It is associated with reduced quality of life [3], increased absenteeism at work and enhanced healthcare costs [4]. When not treated it increases the risk of depression [5] and cardiovascular disorders [6]. The mechanisms underlying the disorder are not fully known, but numerous studies have shown physiological hyperactivity during sleep and wakefulness in insomniacs [7, 8].

Using cognitive behavioral techniques for insomnia treatment comes from the assumption that maladaptive coping strategies with sleep difficulties play a crucial role in the maintenance of this disorder [9]. These strategies may perpetuate hyperarousal. The main behavioral perpetuating factor is the association between bed and wakefulness [10]. Dysfunctional beliefs about sleep and insomnia have been indicated as the main cognitive factor [11]. Techniques aimed to reduce the perpetuating mechanisms are: stimulus control therapy, sleep restriction, relaxation, sleep hygiene education, paradoxical intention and cognitive restructuring of dysfunctional beliefs about sleep [12]. These techniques are usually used together as a cognitive behavioral therapy for insomnia (CBT-I). Previous studies have shown that CBT-I results in improved sleep parameters in 70-80% of patients with insomnia [13]. This treatment is effective for both primary and comorbid insomnia [12] and produces more durable sleep improvements compared to benzodiazepine receptor agonists therapy [14]. There is still few information about the impact of CBT-I on daytime functioning and patients' quality of life [12]. There are also few studies using normal sleepers as a control group to assess clinical significance of CBT-I outcomes.

The aim of the study was to evaluate the effects of CBT-I on sleep parameters and daytime functioning in patients with persistent insomnia compared to good sleepers.

### Materials

The study group was recruited from 236 consecutive patients of Sleep Disorders Clinic of Department of Psychiatry of Medical University of Warsaw, assigned to cognitive behavioral therapy in years 2006-2010. All therapy participants met the criteria of non-organic insomnia according to ICD-10 [1]. From this pool, patients with no history of other psychiatric or sleep disorders conditions, substance dependence, introduction of CNS affecting treatment during the participation in the study and current involvement in psychotherapy were selected ( $n = 72$ ). Eventually, 51 insomniacs and 51 matched healthy, self-defined good sleepers (control group) completed the study.

### Methods

The study design included: two pre-treatment assessment points (before first sleep clinic visit and about 12 weeks later – just before the therapy) and two post-treatment assessment points (after the therapy and three months after the treatment completion). At the last study phase, the control group was recruited. Controls were asked to completed all questionnaires used in insomnia sample once.

Patients filled in sleep diary, the Athens Insomnia Scale (AIS) [15], the Ford Insomnia Response to Stress Test (FIRST) [16], the Beck Depression Inventory (BDI) [17], the State-Trait Anxiety Inventory (STAI) [18] and the quality of life questionnaire – SF-36 [19]. At baseline (before the first diagnostic visit in the Sleep Disorder Clinic) patients completed the AIS, the FIRST, the BDI and the SF-36. At further assessment phases, in addition to above questionnaires, the sleep diary monitoring and the STAI were introduced. In sleep diaries, completed daily for two weeks at each time point,

participants reported their sleep parameters from previous night and sleep medication usage.

The treatment comprised six weekly sessions of 1,5 hour each and one follow-up meeting three months after the therapy. The sessions were implemented in groups of 6-10 patients. During each meeting behavioral and cognitive techniques were introduced successively following a standardized format (Table 1).

Table 1. **Outline of cognitive behavioral therapy for insomnia protocol**

Number and description of session
1. Education about insomnia perpetuating mechanisms and sleep hygiene.
2. Education about physiology of sleep and sleep restriction therapy. This therapy consists of establishing regular bed- and rising times, curtailed nearly to the patient's mean sleep time from previous week (sleep diary). The aim is a deeper and more consolidated sleep.
3. Stimulus control therapy introduction. According to this technique a patient is required to eliminate from sleep environment activities associated with wakefulness and to leave the bed whenever he/she cannot quickly (within 15-20 minutes) fall asleep or return to sleep.
4. Relaxation training (progressive muscle relaxation with elements of imagery training)
5-6. Restructuring dysfunctional sleep and insomnia cognitions.

Repeated measures ANOVA was computed to assess the effects of therapy on sleep and daytime functioning parameters. Post-hoc analyses were performed with Bonferroni corrections. At the next analysis stage the clinical significance of changes due to the treatment was assessed. Based on the criteria from previous studies [12, 13], it has been estimated how many of the treated individuals reached: 1) the clinical improvement defined as a above 50% reduction on the main insomnia symptoms (mean sleep onset latency and/or mean wake time after sleep onset) plus were not taking hypnotics or had reduced a frequency of hypnotic usage; and 2) were not taking hypnotics and reported "normal" sleep parameters defined as: a) both mean sleep onset latency and mean wake time after sleep onset  $\leq 30$  minutes or b) mean sleep efficiency [(mean total sleep time/mean time spent in bed)  $\times 100\%$ ]  $\geq 85\%$ . The clinical significance of the therapy was also assessed by comparing the mean values of dependent variables in patients before and after CBT-I and in good sleepers. The Student's t-test and the Mann-Whitney U test were used. All analyses were conducted using SPSS for Windows version 17.0 statistical software.

## Results

Fifty-one insomnia subjects and 51 healthy controls participated in the study. Each group consisted of 40 women and 10 men. The mean age was  $54.6 \pm 13.9$  years for patients and  $55.4 \pm 14.3$  years for healthy controls ( $p = ns$ ). Most participants in each group had higher level of education ( $n = 30$  and  $n = 26$ ) and were married ( $n = 34$  and  $n = 36$ ), 26 subjects in each group were retired. The mean insomnia duration of patients was  $7 \pm 6.3$  years. Thirty-two patients completed all therapy sessions, 16

subjects missed one session and the rest 3 participants attended 4 sessions. There were no significant differences between the final insomnia group ( $n = 51$ ) and dropouts ( $n = 21$ ) in any demographic or pre-treatment clinical variables.

At baseline, the insomniacs differed significantly from the controls in most dependent variables, except for time in bed and quality of life dimensions related to physical health. Baseline and pre-treatment comparisons in insomnia group revealed a reduction on AIS scores ( $14.8 \pm 3.5$  points versus  $13 \pm 3.4$  points;  $d = 0.53$ ) and no changes of BDI, FIRST and SF-36 scores while awaiting therapy.

After CBT-I, a substantial improvement in all sleep parameters was observed in insomnia group. Patients fell asleep on average by about 28 minutes faster ( $53.6 \pm 40.1$  minutes versus  $25.8 \pm 17.5$  minutes;  $p < 0.001$ ;  $d = 0.76$ ), had less nighttime awakenings ( $2.1 \pm 1$  versus  $1.2 \pm 0.9$ ;  $p < 0.001$ ;  $d = 1.21$ ) and reported reduced wake time after sleep onset from  $86.6 \pm 58.1$  to  $29 \pm 28.1$  minutes ( $p < 0.001$ ;  $d = 1.02$ ). Total sleep time increased from  $5.5 \pm 1.3$  to  $6 \pm 0.9$  hours ( $p = 0.005$ ;  $d = 0.47$ ) and sleep efficiency from  $70 \pm 15\%$  to  $87 \pm 10\%$  ( $p < 0.001$ ;  $d = 1.13$ ). Patients rated higher their sleep quality ( $3 \pm 0.6$  points versus  $3.5 \pm 0.6$  points;  $p < 0.001$ ;  $d = 1.33$ ) and feeling in the morning ( $3.1 \pm 0.5$  points versus  $3.5 \pm 0.6$  points;  $p < 0.001$ ;  $d = 1.06$ ), used hypnotics less frequently ( $28.6 \pm 35.4\%$  versus  $8.2 \pm 20.6\%$ ;  $p < 0.001$ ;  $d = 0.60$ ), reported a significant reduction in insomnia symptoms assessed by the AIS ( $13 \pm 3.4$  points versus  $7.2 \pm 3.5$  points;  $p < 0.001$ ;  $d = 1.49$ ) and reported their vulnerability to stress-related sleep disturbances as lower (FIRST:  $27.4 \pm 5.6$  points versus  $25.3 \pm 6.4$  points;  $p = 0.009$ ;  $d = 0.49$ ).

After the therapy, a substantial improvement in daytime functioning was also observed. Patients reported a reduction of depression symptoms, lower trait and state (before sleep) anxiety and improved energy and social functioning ratings (Table 2).

All changes in sleep parameters and daytime functioning achieved during the therapy were maintained at the 3-month follow-up, with additional improvement, as compared to post-treatment assessment, in total sleep time ( $6 \pm 0.9$  h versus  $6.3 \pm 1$  h;  $p = 0.019$ ), and better functioning, as compared to pre-treatment assessment, in two quality of life dimensions: role limitations due to emotional problems and emotional well-being (Table 2 – next page).

Among 51 participants, 25 subjects met all criteria for clinically significant improvement both at post-treatment and three months after the therapy. There were no significant differences between this group and the rest of the patients ( $n = 26$ ) in demographic variables, pre-treatment symptoms intensity or number of completed therapy sessions. After the therapy 22 subjects reported normal sleep parameters and 16 of them maintain this results after three months. Only 10 patients had no clinically meaningful improvement at any post-treatment time points. In this group most patients were retired and took hypnotics at baseline. Four subject were widowed, and in the whole study sample 6 patients were widowed.

Table 3 – next page shows variables in which patients no longer significantly differed, at both post-treatment time points, from healthy subjects.

Table 2. Changes in daytime functioning after the therapy (ANOVA)

VARIABLES	Pre-treatment		Post-treatment		F	p	Post hoc analysis Significance of differences		
	BT1	BT2	PT1	PT2			BT2-PT2	BT2-PT1	PT1-PT2
	M	SD	M	SD					
Depression symptoms (BDI)	10.2	9.3	6.5	5.7	24.7	0.000	0.001	0.000	1.000
	5.2	5.6	4.9	4.4					
Anxiety symptoms (STAI)									
trait anxiety	-	43.4	40.9	41.1	7.9	0.001	0.012	0.002	1.000
	-	7.9	6.9	6.1					
state anxiety (before sleep)	-	40.6	37.1	36.4	11.8	0.000	0.002	0.000	1.000
	-	8.1	8.5	7.2					
Quality of life (SF-36)									
energy	49.1	50.7	59.3	59.9	13.5	0.000	0.000	0.005	1.000
	20.0	15.2	14.3	13.9					
social functioning	67.2	71.1	78.9	79.7	19.8*	0.000	0.004	0.011	0.826
	22.2	23.3	21.3	21.8					
role limitation due to emotional problems	69.3	69.3	84.0	87.3	16.0*	0.001	0.021	0.010	0.484
	38.0	38.6	30.3	27.7					
emotional well-being	58.7	63.4	68.9	71.7	14.3	0.000	0.068	0.013	0.928
	17.6	16.8	15.5	14.2					

BT – before treatment; PT – post-treatment; M – mean; SD – standard deviation; p – statistical significance (test of within-subjects effects); \* – Friedman test (Chi<sup>2</sup>)

Table 3. Not significantly different variables in patients three months after the therapy and in healthy subjects

VARIABLES	Insomnia patients		Healthy controls		T-test for equality of means	
	M	SD	M	SD	t	p
Sleep parameters (sleep diary)						
number of awakenings	1.3	0.9	1.1	0.8	1.1	0.278
sleep quality (1-5 pts)	3.6	0.7	3.7	0.7	-1.0	0.327
feeling in the morning (1-5 pts)	3.6	0.7	3.8	0.6	-1.5	0.137
Depression symptoms (BDI)	5.7	4.4	5.8	6.0	-0.1	0.926
Anxiety symptoms (STAI)						
trait anxiety	41.1	6.1	38.8	8.5	1.6	0.113
state anxiety (before sleep)	36.4	7.2	35.0	7.7	1.0	0.343
Quality of life (SF-36)						
energy	59.9	13.9	65.1	16.2	-1.6	0.112
social functioning	79.7	21.8	84.1	20.8	1150*	0.293
role limitations due to emotional problems	87.3	27.7	85.0	31.5	1268*	0.754

M – mean; SD – standard deviation; p – statistical significance; \*Mann-Whitney U test

## Discussion

The purpose of the present study was to assess the efficacy of cognitive behavioral therapy for insomnia – the treatment aimed to reduce insomnia perpetuating factors. The results indicate that after CBT-I patients achieve self-reported improvements in both sleep and daytime functioning.

Changes obtained in sleep parameters are similar to those reported in meta-analytic studies [12, 13]. These meta-analyses show that CBT-I produces a reduction on insomnia nighttime symptoms by about 50%: sleep-onset latency is reduced from an average of 60-65 to 35 minutes, number of awakening decreases from two to about one, duration of wake time after sleep onset is reduced from an average of 70 to 38 minutes and total sleep time is increased by about 30 minutes (from 6 to 6.5 hours). The proportion of patients with clinically meaningful improvement in the present study is also similar to this reported in previous studies. According to meta-analytic findings 50% of individuals reach such improvement after CBT-I and 1/3 subjects become good sleepers. In the present study 25/51 patients reported a clinically significant reduction on insomnia symptoms both after the therapy and at the three month follow-up. Sixteen of them achieved sleep parameters within a normative level at both post-treatment time points.

Despite the fact that impaired daytime functioning is a necessary diagnostic criterion for insomnia [1], few studies on the efficacy of CBT-I have targeted this area for outcome assessment [12]. Results of these studies are not consistent and only partially confirm the positive changes after therapy [20-22]. Single trials have shown that the improvement obtained during CBT-I is reflected in better quality of life ratings [23], but these findings were not confirmed in other investigations [22]. The results of the present study demonstrate that after CBT-I patients experience less depression and anxiety symptoms and better quality of life related to mental health. These outcomes are particularly important in the context of studies showing a higher risk of depression in insomnia subjects [5] and suggest that such therapy may have a preventive value. The improvement in psychological well-being may also reflect a better self-estimated ability to cope with insomnia after therapy. This may be confirmed by lower post-treatment FIRST scores, indicating lower subjects' vulnerability to stress-induced sleep disturbance.

The important aim of the present study was to evaluate the clinical significance of the therapy outcomes by comparing sleep and daytime functioning of patients after CBT-I and of healthy subjects. It has been shown that at post-treatment patients stopped to differ significantly from the control group in number of awakenings, sleep quality, feeling in the morning, depression and anxiety symptoms, and quality of life dimensions related to mental health: energy, social functioning, role limitations due to emotional problems and emotional well-being. Significant approximation of the results obtained in treated patients to those obtained in healthy population is identified as the most convincing evidence of treatment efficacy [24]. According to the authors' knowledge, there has been only one study in which individuals after CBT-I were compared to normal sleepers [25]. This study showed that after this therapy sleep-onset insomniacs no longer differed from control group in self-estimated sleep efficiency and

objectively measured (in polysomnography) sleep onset latency and sleep efficiency, and in reported depression and anxiety symptoms.

The present study design has some methodological limitations which need to be discussed. The study was conducted using a quasi-experimental design (without randomization), therefore the control of variables which, apart from the CBT-I participation, might contribute to patients' improvements was limited. Two pre-therapy assessments allowed to conclude about changes occurring without treatment. It was shown that the study group already reported some improvement in symptoms measured by the AIS during the CBT-I waiting period. These results might be influenced mainly by factors associated with a first doctor visit in the clinic - a contact with a specialist and basic sleep hygiene recommendations, which were allowed during this visit. A substantial reduction in the AIS mean score, below the threshold considered to be diagnostic for insomnia [15], was observed in the study group only after the therapy completion. Thus, it seems that the observed improvement was related primarily to the participation in CBT-I. This may be confirmed by the absence of significant changes in the average pre-treatment scores of other instruments applied during the waiting period.

Data on adherence to treatment recommendations were not collected in the present study, and that might influence the outcomes. The finding that the group with a sustained improvement did not differ from the rest of participants in the number of completed sessions may presumably be explained by the fact that patients with non-full attendance were given the materials from the missed sessions. Moreover, most of them did not complete one of six sessions, therefore they had an opportunity to make up the missing content at further meetings. Based on the characteristics of group with no clinically meaningful improvement at post-treatment, it may be assumed that occupational inactivity, state of widowhood and hypnotic usage predict poor CBT-I response. Because of the small size of this group ( $n = 10$ ) and the lack of similar data from other studies, [26] these results should be interpreted with caution. Further research should attempt to identify predictors of CBT-I efficacy. Positive CBT-I outcome may also be influenced by other variables associated with psychotherapy process. In case of sessions delivered in a group format, meeting other people with similar problem may be an important healing factor.

## Conclusions

These findings support the previous data on the important role of techniques aimed to reduce perpetuating factors in successful treatment of chronic insomnia. The cognitive behavioral group therapy produced a sustained self-reported improvement in nocturnal sleep and daytime functioning, and was effective in bringing patients' sleep quality and mental health ratings within the distribution of good sleepers.

## References

1. *Międzynarodowa Statystyczna Klasyfikacja Chorób i Problemów Zdrowotnych, rewizja dziesiąta. Klasyfikacja zaburzeń psychicznych i zaburzeń zachowania w ICD-10. Opisy kliniczne i wskaźniki diagnostyczne.* Kraków-Warszawa: Uniwersyteckie Wydawnictwo Medyczne „Vesalius”, Instytut Psychiatrii i Neurologii; 2000.

2. Ohayon MM. *Epidemiology of insomnia: what we know and what we still need to learn*. Sleep Med. Rev. 2002; 6: 97–111.
3. LeBlanc M, Beaulieu-Bonneau S, Mérette C, Savard J, Ivers H, Morin CM. *Psychological and health-related quality of life factors associated with insomnia in a population-based sample*. J. Psychosom. Res. 2007; 63: 157–166.
4. Godet-Cayré V, Pelletier-Fleury N, Le Vaillant M, Dinét J, Massuel MA, Léger D. *Insomnia and absenteeism at work. Who pays the cost?* Sleep 2006; 29: 179–184.
5. Baglioni C, Battagliese G, Feige B, Spiegelhalder K, Nissen C, Voderholzer U, Lombardo C, Riemann D. *Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies*. J. Affect. Disord. 2011, epub.
6. Phillips B, Mannino DM. *Do insomnia complaints cause hypertension or cardiovascular disease?* J. Clin. Sleep. Med. 2007; 3: 489–494.
7. Bonnet MH, Arand DL. *24-hour metabolic rate in insomniacs and matched normal sleepers*. Sleep 1995; 18: 581–588.
8. Wołyńczyk-Gmaj D, Szelenberger W. *Waking EEG in primary insomnia*. Acta Neurobiol. Exp. 2011; 71: 387–392.
9. Spielman AJ, Glovinsky PB. *The varied nature of insomnia*. W: Hauri PJ red. *Case Studies in Insomnia*. New York: Plenum Medical Book Co; 1991. s.1–15.
10. Bootzin RR, Epstein D, Wood JM. *Stimulus control instructions*. W: Hauri PJ red. *Case Studies in Insomnia*. New York: Plenum Medical Book Co; 1991. s.19–28.
11. Morin CM. *Insomnia: Psychological assessment and management*. New York: The Guilford Press; 1993.
12. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. *Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004)*. Sleep 2006; 29: 1398–1414.
13. Morin CM, Hauri PJ, Espie CA, Spielman AJ, Buysse DJ, Bootzin RR. *Nonpharmacologic treatment of chronic insomnia*. Sleep 1999; 22: 1–23.
14. Riemann D, Perlis ML. *The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies*. Sleep Med. Rev. 2009; 13: 205-214.
15. Fornal-Pawłowska M, Wołyńczyk-Gmaj D, Szelenberger W. *Walidacja Ateńskiej Skali Bezsenności*. Psychiatr. Pol. 2011; 45: 211–221.
16. Fornal-Pawłowska M, Skalski M, Szelenberger W. *Badanie predyspozycji do bezsenności za pomocą skali FIRST*. Sen 2007; 7: 104–109.
17. Parnowski T, Jernajczyk W. *Inwentarz Depresji Becka w ocenie nastroju osób zdrowych i chorych na choroby afektywne*. Psychiatr. Pol. 1977; 11: 417–425.
18. Sosnowski T, Wrześniewski K, Jaworowska A, Fecenec D. *Inwentarz Stanu i Cechy Lęku STAI. Polska adaptacja STAI*. Podręcznik, wyd. III, rozszerzone. Warszawa: Pracownia Testów Psychologicznych Polskiego Towarzystwa Psychologicznego; 2006.
19. Marcinowicz L, Sienkiewicz J. *Badanie trafności i rzetelności polskiej wersji kwestionariusza SF-36: wyniki wstępne*. Przegl. Lek. 2003; 60: 103-106.
20. Backhaus J, Hohagen F, Voderholzer U, Riemann D. *Long-term effectiveness of a short-term cognitive-behavioral group treatment for primary insomnia*. Eur. Arch. Psychiatry Clin. Neurosci. 2001; 251: 35–41.
21. Espie CA, Inglis SJ, Tessier S, Harvey L. *The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice*. Behav. Res. Ther. 2001; 39: 45–60.
22. Omvik S, Sivertsen B, Pallesen S, Bjorvatn B, Havik OE, Nordhus IH. *Daytime functioning in older patients suffering from chronic insomnia: Treatment outcome in a randomized controlled trial comparing CBT with zopiclone*. Behav. Res. Ther. 2008; 46: 623–641.

23. Espie CA, MacMahon KM, Kelly HL, Broomfield NM, Douglas NJ, Engleman HM, McKinstry B, Morin CM, Walker A, Wilson P. *Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice*. *Sleep* 2007; 30: 574–584.
24. Rakowska JM. *Skuteczność psychoterapii. Przegląd badań*. Warszawa: Wydawnictwo Naukowe SCHOLAR; 2005.
25. Jacobs GD, Benson H, Friedman R. *Home-based central nervous system assessment of a multifactor behavioral intervention for chronic sleep-onset insomnia*. *Behav. Ther.* 1993; 24: 159-174.
26. Edinger JD, Carney CE, Wohlgenuth WK. *Pretherapy cognitive dispositions and treatment outcome in cognitive behavior therapy for insomnia*. *Behav. Ther.* 2008; 39: 406–416.

*The study has not been sponsored.*

**Correspondence address:** Małgorzata Fornal-Pawłowska  
Medical University of Warsaw, Department of Psychiatry  
Nowowiejska Street 27, 00-665 Warszawa,