






# RELATIONSHIP BETWEEN INSOMNIA AND TOBACCO SMOKING IN ALCOHOL-DEPENDENT PATIENTS

## ZWIĄZEK BEZSENNOŚCI I PALENIA TYTONIU U PACJENTÓW UZALEŻNIONYCH OD ALKOHOLU

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### Abstract

**Introduction:** Insomnia is a risk factor for relapse in alcohol-dependence. Many conditions contribute to the severity of insomnia, including smoking. The aim of this study was to assess the association between parameters of tobacco smoking and the severity of insomnia after controlling for other correlates of insomnia in alcohol-dependent patients.

**Material and methods:** In the study included 386 patient participants. Standardised tools were used to assess tobacco dependence (FTND), sleep (AIS and SDQ-7), alcohol dependence and consumption (MAST, TFLB), psychiatric symptoms (BSI), social support and impulsivity.

### Streszczenie

**Wprowadzenie:** Bezsenność jest czynnikiem ryzyka nawrotu picia w uzależnieniu od alkoholu. Wiele czynników wpływa na nasilenie zaburzeń snu, m.in. palenie tytoniu. Celem badania było określenie związku pomiędzy paleniem a bezsennością u osób uzależnionych od alkoholu.

**Materiał i metody:** Do badania włączono 386 pacjentów. Zastosowano narzędzia do oceny nasilenia uzależnienia od tytoniu (FTND), snu (AIS i SDQ), nasilenia uzależnienia od alkoholu i jego konsumpcji (MAST, TFLB), objawów zaburzeń psychicznych (BSI), oparcia społecznego i impulsywności.

**Wyniki:** Osoby palące stanowiły 79% przebadanych pacjentów uzależnionych od alkoholu; większość

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**Results:** Current smokers constituted 79% of alcohol dependent patients. Most of them were tobacco-dependent (mean  $\pm$  SD FTND score: 6.12  $\pm$  2.09); 62% of participants reported insomnia (AIS). The multivariate regression analysis revealed that the severity of psychiatric symptoms (BSI, beta = 0.422,  $p < 0.0005$ ), severity of tobacco dependence (FTND, beta = 0.140,  $p = 0.013$ ) and intensity of drinking (TLFB, beta = 0.123,  $p = 0.034$ ) predicted the severity of insomnia in alcohol-dependent subjects.

**Discussion:** Our findings suggest that smoking is significantly associated with insomnia severity in alcohol-dependent individuals. Some alcohol dependence treatment programmes include tobacco-dependence interventions, but they are not obligatory. Tobacco dependence as well as the severity of psychiatric symptoms and recent drinking consumption were independent predictors of insomnia severity in alcohol-dependent patients, which should be taken into consideration in determining addiction treatment goals.

**Conclusions:** Our results may implicate that addressing the issue of nicotine use during alcohol-dependence programmes might have positive influence on alcohol treatment outcomes.

**Keywords:** Alcohol, Insomnia, Nicotine

z nich stanowiły osoby uzależnione od nikotyny (średni FTND  $\pm$  SD: 6,12  $\pm$  2,09); 62% badanych zgłosiło bezsenność (AIS). Wieloczynnikowa regresja logistyczna wykazała, że większe nasilenie objawów zaburzeń psychicznych (BSI, beta = 0,422,  $p < 0,0005$ ), uzależnienia od tytoniu (FTND, beta = 0,140,  $p = 0,013$ ) oraz duża intensywność picia alkoholu (TLFB, beta = 0,123,  $p = 0,034$ ) są predyktorami bezsenności u pacjentów uzależnionych od alkoholu.

**Omówienie:** Bezsenność wpływa negatywnie na jakość życia i jest związana ze współwystępowaniem objawów psychopatologicznych. Niniejsze badanie wskazuje, że palenie tytoniu jest w istotny sposób związane z nasileniem bezsenności u osób uzależnionych od alkoholu. Niektóre programy terapeutyczne dla pacjentów uzależnionych od alkoholu zawierają interwencje dotyczące palenia tytoniu, ale zajęcia te nie są obowiązkowe. Uzależnienie od tytoniu, stopień nasilenia objawów zaburzeń psychicznych i niedawne spożywanie alkoholu były niezależnymi predyktorami nasilenia bezsenności u osób uzależnionych od alkoholu, co powinno zostać uwzględnione w planowaniu leczenia odwykowego.

**Wnioski:** Niniejsze badanie dowodzi, że uwzględnienie problematyki palenia tytoniu w programach odwykowych może mieć korzystny wpływ na wyniki leczenia.

**Słowa kluczowe:** alkohol, bezsenność, nikotyna

## ■ INTRODUCTION

One of the most common problems observed in alcohol-dependent population is insomnia, which manifests as difficulties with falling asleep, frequent rousing at night or too early awakenings in the morning. It may occur as a single sleep disorder or as a manifestation of other comorbid mental disorders such as anxiety, depression or addictive behaviours. In alcohol-dependent individuals, insomnia is specifically intensive in the early abstinence period and its severity often determines addiction treatment outcomes, because it is a serious risk factor for relapse [1]. As recent research indicates, insomnia also increases the risk for suicidal behaviour [2, 3] which is

highly prevalent in alcohol-dependent subjects. In the general population, the prevalence of chronic insomnia is estimated about 16% [4], but among alcohol-dependent individuals it is from 36% to 72% [1, 5]. The effects of alcohol on insomnia are well documented [5]. However, it is important to remember that sleep problems may occur as a result of both chronic ethanol effects on the central nervous system (CNS) and other factors independent of alcohol use. Research on factors that may influence insomnia is clinically important because it could help in modifying treatment programmes and reducing relapse.

Another common disorder that coexists with alcohol dependence is tobacco dependence (TD). It is estimated that from 70% to 90% of patients en-

tering alcohol treatment programmes smoke cigarettes as compared to 20-30% of current smokers in the general population [6-8]. Analysis of the data gathered in the Project MATCH (Matching Alcoholism Treatment to Client Heterogeneity) study suggests that smoking is a risk factor for poor alcohol treatment outcomes [9]. Some studies show that nicotine enhances drinking [10] due to the biological interactions between nicotine and ethanol impacting the reward system [11, 12]. The relationships between ethanol and nicotine are reciprocal, such that drinking stimulates smoking urges as well. Furthermore, tobacco and ethanol act synergistically generating negative health consequences. The chronic use of both substances multiplies the risk for many somatic disorders as compared to the use of only one substance [13]. The consequences of tobacco smoking are often underestimated, although tobacco-related diseases are the major cause of deaths in the alcohol-dependent population [14]. Tobacco smoking is also associated with depression and anxiety. At first, nicotine in single doses alleviates symptoms in accordance with the self-medication hypothesis, but chronic smoking contributes to both anxiety and depression [15-18].

Nicotine is known to have an adverse impact on sleep. In healthy non-smoking individuals, researchers observed dose-dependent sleep disorders after administration of nicotine, specifically reduced REM-sleep latency and low subjective sleep quality ratings [19]. These changes were similar to ethanol-related effects. Previous polysomnography (PSG) studies showed that current smokers fell asleep later and experienced more sleep apneas, arousals and leg movements during sleep. In addition, the architecture of sleep phases changes, which is demonstrated by an increased number of REM sleep phases [20]. Wallander *et al.* indicated that smoking and increased alcohol consumption are the two major causes of sleep disorders beyond factors like comorbid mental disorders and somatic dysfunction among UK General Practice patients [21].

The aim of this study was to evaluate the relationship between tobacco dependence (TD) and insomnia in alcohol-dependent patients admitted for addiction treatment. Previous studies have shown mixed results. Two studies found no relationship between smoking and insomnia in alcohol-dependent patients [22, 23], one study found

a significant relationship in bivariate but not multivariate analyses [24], and one study found a positive relationship in multivariate analyses [25]. Unlike these previous studies, this study analyses relationships between the severity of insomnia and several different parameters of smoking, including the severity of TD, number of cigarettes per day, and years of smoking in alcoholic patients. We hypothesised that all three parameters would correlate with the severity of insomnia. We also evaluated other factors previously associated with insomnia in alcohol-dependent patients. Specifically, we examined relationships between insomnia and coexisting psychiatric symptoms, impulsivity, severity of alcohol dependence, and amount of alcohol consumed before entering treatment programme.

## ■ MATERIAL AND METHODS

### Participants

There were 386 patients enrolled in the study. All patients were alcohol-dependent according to DSM-IV-TR classification. They were recruited in addiction treatment centres in the area of Warsaw, Poland. Most of them ( $n = 352$ ) were recruited from residential (inpatient) centres, only 34 were outpatients. The study protocol was approved by the Bioethics Committee at the Medical University of Warsaw and the Medical Institutional Review Board at the University of Michigan. Excluding criteria were: age under 18 years, active symptoms or complications of alcohol withdrawal and a diagnosis of mental disorders requiring medications (assessed by the M.I.N.I., Mini-International Neuropsychiatric Interview) [26], use of other psychoactive substance (except from nicotine) and serious cognitive deficits as assessed by a score of  $< 24$  on the Mini-Mental State Examination [27]. Each participant signed an informed consent form before entering the study, understanding that participation was voluntary.

### Procedure

Patients were recruited to the study at the beginning of the treatment programme within the first week after admission. Those subjects who met study inclusion criteria completed a self-administered questionnaire and two clinician-administered interviews: the M.I.N.I. and the Timeline Follow-Back (TLFB). These proce-

dures usually took 2-3 hours to complete. Polish translations of all questionnaires and interviews were used.

### Measures

To assess smoking: **Fagerström Test of Nicotine Dependence (FTND)**. This six-item questionnaire measures the severity of TD. Score under 2 points suggests a low probability of TD, 3-4 points indicates low severity, 5 – moderate severity, 6-7 – high severity and 8-10 – very high severity of TD [28]. Additionally, we asked about the age of onset and number of years of smoking.

To assess sleep problems: **Athens Insomnia Scale (AIS)**. This is a cross-culturally validated, 8-item questionnaire which assessed symptoms and consequences of insomnia that occurred during the last month [29]. In this study AIS was used mainly to assess dichotomised current insomnia status. The cut-off score for insomnia in AIS is 6.

**Short version of Sleep Disorder Questionnaire SDQ (SDQ-7)**. This 7-item scale, derived from the 175-item Sleep Disorders Questionnaire [30]. In contrast to AIS, SDQ asks about symptoms of insomnia during longer period (previous 6 months) [22]. In this study SDQ-7 was used to assess severity of sleeping problems.

**Brief Symptom Inventory (BSI)** was used to assess the severity of psychiatric symptoms, including depression and anxiety over the previous two weeks. It consists of 53 items. For the purposes of this study, the Global Severity Index of the BSI was used [31].

**Barratt Impulsiveness Scale (BIS-11)** a 30-item measure often applied as a gold-standard in studies on impulsive behaviours was used to assess subjective level of impulsivity [32].

**Medical Outcomes Study Social Support Survey (MOSSSS)** [33] contains 20-items to assess various aspects of social support in patients with chronic health conditions.

**Timeline Follow-Back (TLFB)** was used to evaluate amount of alcohol consumption per day and frequency of drinking in the last 90 days before the beginning of treatment. As a measure of consumed ethanol a standard drink (1 standard drink = 10 g of pure ethanol) was used. The total amount of alcohol consumed during last three months before entering the treatment was mea-

sured. Each questionnaire had a simple guide how to transform different kinds of alcoholic beverages (beer, wine, vodka) into standard drinks. Trained staff assisted to fill-in the calendar [34].

**Michigan Alcohol Screening Test (MAST)** consists of 24 yes/no questions to screen for alcohol dependence. The total score was used in this study to assess the severity of alcohol dependence [35] with higher scores indicating greater severity.

### Statistical analysis

Collected data were analysed with STATISTICA software version 10.0. First, we used the Kolmogorov-Smirnov test to check for normal distributions. In bivariate analyses of continuous variables, Pearson's correlation analysis for parametric variables and Spearman rank correlation analysis for non-parametric variables were conducted. Next, we analysed the association between two major measures: nicotine dependence (FTND score) and insomnia (AIS, SDQ-7) with alcohol drinking (TLFB, MAST), global BSI score, BIS-11, and MOSSSS. For continuous variables we used Student's *t* tests and ANOVAs. Finally, multivariate linear regression analysis was conducted to evaluate predictors of insomnia severity. The SDQ-7 score was used as the dependent variable to assess sleep problems during last 6 months. Those variables that had significant associations with insomnia in bivariate analyses were entered into the regression model as independent variables. The regression analysis also controlled for age and gender.

## ■ RESULTS

Two hundred eighty four males and one hundred two females, all Caucasian, were enrolled in the study. The participants were  $43.8 \pm 10$  years old on average and they had been dependent on alcohol for  $19.1 \pm 10$  years; the median age at onset of problem drinking was 22 (19.5; 29.5) years. In the study population, 79.1% ( $n = 306$ ) were current smokers, 12.4% ( $n = 44$ ) were former smokers and 8.5% ( $n = 36$ ) never smoked. The median age of smoking initiation was 17 (interquartile range: 15; 19) years. Among current smokers 7.8% ( $n = 24$ ) scored FTND  $\leq 2$  which means low probability of TD. Scores between 3 and 4 got 13.4% ( $n = 41$ ), 15.4% ( $n = 47$ ) were moderate smokers (FTND = 5). Severity of TD was high

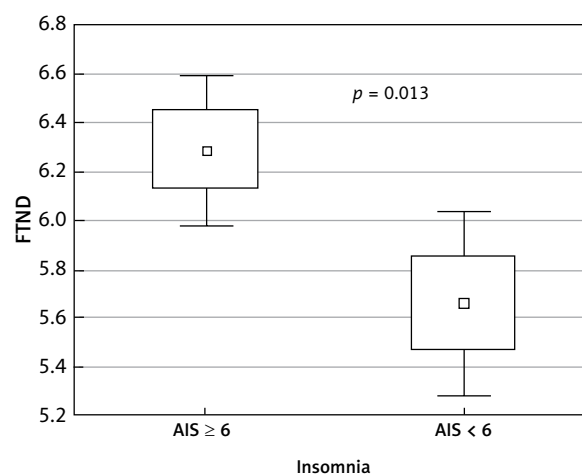
(FTND = 6-7) in 35% ( $n = 107$ ) and very high ( $> 8$  in FTND) in 28.4% ( $n = 87$ ). The mean score in FTND was  $6.12 \pm 2.09$ . Almost half of current smokers (47.9%,  $n = 146$ ) declared between 11 and 20 cigarettes smoked per day. One third (34.5%,  $n = 106$ ) smoked between 21 and 30 cigarettes per day and 30 participants (9.7%) admitted over 30 cigarettes while 24 (7.8%) smokers smoked less than 10 cigarettes.

In the study sample, insomnia was observed in 237 (61.4%) participants who had a total score  $\geq 6$  in AIS. There was no difference between smokers and non-smokers in prevalence of insomnia (chi square = 0.826,  $df = 1$ ,  $p = 0.363$ ) but within the group of current smokers, those with higher scores in FTND more often suffered from insomnia ( $p = 0.013$ ; see Fig. 1). In addition, there was a significant association between the number of cigarettes smoked per day and severity of sleep problems (SDQ-7:  $F = 3.08$ ,  $p = 0.028$ ; see Fig. 2). We also noted a significant, albeit small, positive correlations between severity of TD (FTND) and severity of insomnia (see Table I). Patients who started regular smoking earlier in their lives reported more severe insomnia during the preceding month (AIS). There was no significant correlation between duration of chronic smoking and severity of insomnia (AIS; SDQ-7).

The statistical analysis revealed a significant positive correlation between the severity of insomnia during the previous 6 months and severity of alcohol dependence. Specifically, insomnia severity correlated significantly with the lifetime duration of heavy drinking ( $r = 0.123$ ,  $p = 0.036$ ), larger amounts of alcohol consumed during the past 90 days (TLFB:  $r = 0.175$ ,  $p = 0.002$ ), and higher MAST scores ( $r = 0.157$ ,  $p = 0.003$ ).

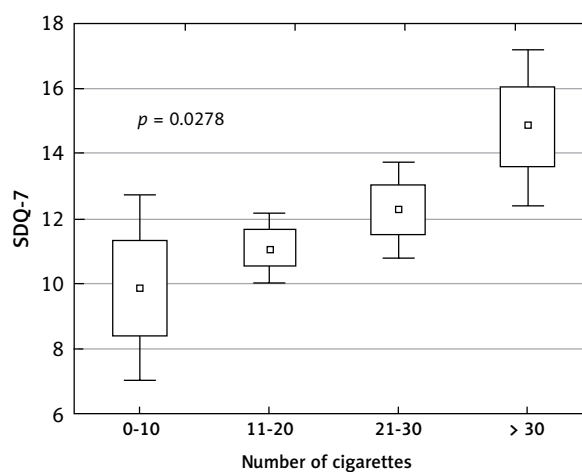
Subsequently, we analysed correlations between insomnia severity and psychiatric symptoms severity, levels of impulsivity, and social support. Participants with higher scores on the SDQ-7 had higher scores on the Global Severity Index of the BSI ( $r = 0.439$ ,  $p < 0.0005$ ), were more impulsive (BIS-11:  $r = 0.199$ ,  $p < 0.0005$ ), and had lower social support (MOSSSS:  $r = -0.198$ ,  $p = 0.002$ ).

A multivariate linear regression analysis was conducted to evaluate independent correlates of severity of insomnia. All variables were significantly associated with SDQ-7 scores (FTND, MAST, TFLB, duration of alcohol dependence, social support, global psychiatric symptom severity,



AIS – Athens Insomnia Scale  
FTND – Fagerström Test for Nicotine Dependence

**Figure 1.** Association between presence of insomnia (AIS  $\geq 6$ ) and severity of nicotine dependence (FTND)



SDQ-7 – Sleep Disorder Questionnaire

**Figure 2.** Association between severity of sleep problems and number of cigarettes smoked per day

**Table I.** Relationships between severity of sleep disorder and severity of nicotine dependence, age of smoking onset and number of smoking years

	FTND	The age of smoking onset	Years of smoking
AIS	$r = 0.162$ $p = 0.004$	$R = -0.165$ $p = 0.002$	$r = -0.008$ $p = 0.898$
SDQ-7	$r = 0.230$ $p < 0.0005$	$R = -0.101$ $p = 0.060$	$r = 0.052$ $p = 0.338$

AIS – Athens Insomnia Scale, SDQ-7 – Sleep Disorder Questionnaire, FTND – Fagerström Test for Nicotine Dependence  
 $r$  – Pearson correlation coefficient,  $R$  – Spearman correlation coefficient  
Significant values are presented in bold.

**Table II.** Multivariate regression analysis for predictors of insomnia severity (SDQ-7)

Predictive factor	Beta coefficient	<i>p</i>
Psychopathology (BSI)	<b>0.424</b>	<b>&lt; 0.0005</b>
Severity of nicotine dependence (FTND)	<b>0.140</b>	<b>0.014</b>
Intensity of drinking (TLFB)	<b>0.123</b>	<b>0.037</b>
Age	0.118	0.119
Severity of alcohol dependence (MAST)	0.085	0.180
Social support (MOSSSS)	-0.074	0.199
Impulsiveness (BIS-11)	0.024	0.710
Duration of alcohol dependence	0.019	0.807
Gender	0.013	0.834

BSI – Brief Symptom Inventory, FTND – Fagerström Test for Nicotine Dependence, TLFB – Timeline Follow-Back, MAST – Michigan Alcoholism Screening Test, MOSSSS – Medical Outcomes Study Social Support Survey, BIS-11 – Barratt Impulsiveness Scale

Significant values are presented in bold. Model:  $F = 11.496$ ;  $df = 9.237$ ;  $corr. R^2 = 0.277$ ;  $p < 0.0005$

impulsiveness) and were entered along with two control variables (gender and age). Two variables were excluded from the regression analysis to avoid multicollinearity. First was the number of smoked cigarettes per day that was highly correlated with FTND scores and second the age of onset of smoking, which was correlated with the years of smoking. The linear regression model was statistically significant ( $corr. R^2 = 0.277$ ,  $p < 0.0005$ ). The strongest predictor of insomnia severity was psychiatric symptoms severity (BSI). Two other variables that remained significant were severity of tobacco dependence (FTND score) and the quantity of alcohol consumed (total number of drinks) during the last three months (TFLB). Severity of alcohol dependence (MAST), social support, duration of alcohol dependence, impulsiveness, age and gender did not predict insomnia after controlling for other variables in the study sample (see Table II).

## ■ DISCUSSION

### Insomnia and tobacco dependence

In order to assess the relationship between insomnia and tobacco smoking, we collected data from a sample of alcohol-dependent patients participating in addiction treatment programmes in Poland. In our sample, the prevalence of both insom-

nia and current smoking was consistent with earlier findings on alcohol-dependent patients [36, 37]. Importantly, patients were beginning treatment and initiating abstinence from alcohol. This may explain in part the high frequency of insomnia. We did not observe differences between smokers and non-smokers in the prevalence of insomnia. Our data suggest that smoking severity, whether measured by age of onset, cigarette consumption, or FTND scores, was a better predictor of insomnia than dichotomised smoking status.

It has been documented that nicotine, similarly to alcohol, changes sleep patterns [38, 39]. In former research on sleep EEG of smokers compared to non-smokers higher percentage of alpha frequency was observed, which is typical for arousal [40, 41]. Simultaneously, lower percentage of delta frequency was recorded, which is typical for deep sleep states [39]. These changes in brain activity at night may be responsible for non-restorative sleep and increased drowsiness during the day, despite normal sleep duration. Durazzo *et al.* analysed neuroimaging studies of smokers and non-smokers in order to compare structural differences in CNS of alcohol-dependent individuals in recovery from alcohol [42]. They observed lower total volumes of grey matter, especially in temporal lobes, and larger volumes of white matter in the frontal lobes of alcohol-dependent smokers compared to non-smokers. These researchers suggested that continuous smoking delays brain regeneration during abstinence from alcohol.

Another reason for poor sleep associated with nicotine use are the night-time awakenings caused by nicotine craving [43]. Due to the short half-life of nicotine, its blood concentration rapidly decreases at night and smokers with sleep problems awaken and smoke cigarettes. According to Rieder *et al.*, 20% of heavy smokers experience symptoms of nicotine withdrawal at night and wake up for a cigarette [44]. Alcohol-dependent patients are usually heavy smokers, thus the prevalence of “night smoking” might be even greater.

In our sample, patients who started smoking earlier in their lives suffered more often from symptoms of insomnia across the last 30 days. According to Brook *et al.*, early smoking initiation during adolescence induces neuropathological changes in the CNS, which can lead to sleep abnormalities that may be difficult to reverse in adulthood [45, 46]. Nicotine and ethanol are often used concurrently

and chronically. In our study sample, participants started regular smoking before the age of onset of problem drinking, which may have exacerbated alcohol-associated insomnia.

While analysing the relationship between insomnia and tobacco dependence (TD), it is important to mention tobacco-related somatic disorders, especially those associated with the cardiovascular and respiratory systems that may lead to insomnia. One cause of disrupted sleep due to frequent night unconscious awakenings is obstructive sleep apnea syndrome (OSAS). Presence of OSAS leads to non-restorative sleep and subjective sense of drowsiness during the day. Recently, it has been documented that chronic heavy smoking induces symptoms of OSAS independently of other factors such as obesity [47].

#### Other correlates of insomnia in alcohol-dependent patients

Since a variety of factors leads to the development of insomnia in alcohol-dependent population, we analysed selected comorbid conditions in our study sample. Consistent with other research, our results confirmed that severity of psychiatric symptoms as measured by the Brief Symptom Inventory and drinking quantity before entering the addiction treatment programmes were predictors of insomnia severity [48]. As previously observed, in the early period of abstinence, patients often report psychiatric symptoms, especially anxiety or mood instability. Insomnia often accompanies those symptoms and conversely, sleep problems relevantly impair psychological well-being. In our study, those patients with greater insomnia severity were characterised by higher levels of impulsivity, lower social support and higher severity of alcohol dependence. However, none of these factors was significant in multivariate regression analysis.

**Strengths and limitations correlates.** A major strength of the study was that it assessed multiple parameters of smoking, rather than simply dichotomising the sample into smokers and non-smok-

ers. Previous studies in alcohol-dependent patients using dichotomisation had conflicting results with respect to insomnia [22, 23, 25]. As with two of those studies [22, 23], this study found no differences between smokers and non-smokers, though it did find differences when smoking severity, number of cigarettes, or age on smoking onset were analysed. The goal of treatment of alcohol dependence is to achieve long-term abstinence or at least harm reduction. Despite therapeutic efforts, treatment outcomes are generally not satisfactory. Thus it is essential to analyse factors associated with insomnia, because the latter is known to influence relapse in many studies [1]. Modifiable factors such as smoking are especially clinically relevant for insomnia that persists despite abstinence from alcohol. Therefore, smokers should be encouraged and provided treatment to stop smoking.

One important limitation of our study is the lack of the assessment of night and day smoking patterns. Smoking during the night and short time between last cigarette and the bed time can significantly affect sleep problems. In addition, we did not measure the level of cotinine, the main metabolite of nicotine in blood, which could provide more reliable data. Moreover, we did not use any objective measure of insomnia such as actigraphy or polysomnography. The study protocol was based on subjective individual reports. Subjective and objective data might be inconsistent. However, in the population of alcohol-dependent individuals, subjective measures might have clinical value because they truly reflect risk for relapse associated with insomnia [49].

## ■ CONCLUSIONS

The study outcomes show that smoking is important factor influencing the course of alcohol dependence. The severity of nicotine dependence is linked to sleep disorders among alcohol-dependent individuals. It is important to provide the treatment of nicotine dependence in alcohol treatment programmes.

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#### Conflict of interest/Konflikt interesów

None declared./Nie występuje.

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### Ethics/Etyka

The work described in this article has been carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) on medical research involving human subjects, EU Directive (210/63/EU) on protection of animals used for scientific purposes, Uniform Requirements for manuscripts submitted to biomedical journals and the ethical principles defined in the Farmington Consensus of 1997.

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