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Sleep apnea severity and depressive symptoms in a population-based study

Annemarie I. Luik, PhD [a], Jenna Noteboom, BSc [a], Lisette A. Zuurbier, MSc [a], Harry Whitmore, RPSGT [b], Albert Hofman, MD, PhD [a], Henning Tiemeier, MD, PhD [a,c,d,*]

[a] Department of Epidemiology, Erasmus University Medical Center, Rotterdam, the Netherlands
[b] Section of Endocrinology in the Department of Medicine, University of Chicago, Chicago, IL, USA
[c] Department of Child and Adolescent Psychiatry, Erasmus University Medical Center, Rotterdam, the Netherlands
[d] Department of Psychiatry, Erasmus University Medical Center, Rotterdam, the Netherlands

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ABSTRACT

Objectives: Sleep apnea and depression often co-occur in clinical studies, but population-based studies demonstrated mixed results. We determined the association of sleep apnea severity and depressive symptoms in a population-based sample.
Design: Cross-sectional cohort study.
Setting: Population-based.
Participants: Four hundred ninety-one middle-aged and elderly persons of the Rotterdam Study (mean age 61.9 years; standard deviation, 5.4).
Measurements: Polysomnography recordings were collected to calculate the apnea hypopnea index (AHI). Depressive symptoms were assessed with the Center for Epidemiologic Studies Depression Scale.
Results: In the total sample, no associations for the severity of sleep apnea with depressive symptoms were found (multivariate adjusted: $B = 0.032$; 95% confidence interval [CI], −0.057 to 0.122). Only in men we found some evidence for a curvilinear association of the severity of sleep apnea with depressive symptoms (multivariable adjusted: $B = -0.126$; 95% CI, −0.224 to −0.028); men with an AHI between 5 and 15 (multivariable adjusted: $B = 0.378$; 95% CI, 0.037-0.718) or between 15 and 30 (multivariable adjusted: $B = 0.502$; 95% CI, 0.152-0.852) had significantly more depressive symptoms than those with an AHI equal to or greater than 30.
Conclusions: In this population-based sample, the severity of sleep apnea is not consistently related to depressive symptoms, although there was some evidence for an association of AHI with depressive symptoms in men.
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Introduction

Sleep apnea is common in the general population[1] and is associated with severe health problems, such as cognitive dysfunction,[2] diabetes,[3] osteoporosis,[4] hypertension,[5] cardiovascular diseases, and mortality.[6] Sleep apnea also shares several symptoms with depression, such as fatigue, disturbed sleep, irritability, depressed mood, and poor concentration.[7]

Previous studies showed high rates of sleep apnea in persons with major depressive disorder (18%) and high rates of major depressive disorder among persons with clinically diagnosed sleep apnea (17%-22%).[1,8–11] However, results from studies on sleep apnea severity and depressive symptoms have been inconsistent.[12,13] One study in patients with sleep apnea suggest that there is an association of low oxygen saturation with depressive symptoms but not between the apnea hypopnea index (AHI) with depressive symptoms,[14] whereas other studies suggest no association between indicators of sleep apnea severity with depressive symptoms.[15–17] A study including persons with suspected sleep apnea reported no consistent association between the severity of respiratory disturbances and depressive symptoms.[18] This is in line with results obtained from a large study of the general population.[19] In a longitudinal population-based study, a dose-response association between sleep apnea and depressive symptoms was found when participants with an AHI greater than zero were compared with persons with no apneas or hypopneas.[20] However, the number of population-based samples studying the association of the continuum of sleep apnea severity with depressive symptoms is limited and their results are conflicting.[19,20]

* Corresponding author at: Department of Epidemiology, Erasmus University Medical Center, P.O. Box 2040, 3000 CA Rotterdam, the Netherlands. Tel.: +31 10 7043489; fax: +31 10 7044657.
E-mail address: h.tiemeier@erasmusmc.nl (H. Tiemeier).

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Both sleep apnea and depression are diseases wherein pronounced differences between the sexes are seen. Sleep apnea is more common in men; estimates in population-based studies demonstrated that 9% to 15% of men have an AHI equal to or greater than 15, whereas 4% to 7% of women have an AHI equal to or greater than 15.[5] The prevalence of diagnosed obstructive sleep apnea syndrome is generally lower than than that with an AHI equal to or greater than 15 but does also show more men diagnosed with sleep apnea than women.[1] For depression, we see an opposite pattern; women tend to report more depressive episodes than men (5.3 vs 3.2, respectively).[1] Two studies have suggested differential associations for sleep-disordered breathing and depressive symptoms in men and women. A study in another large community sample found an association between self-reported apnea and depressive symptoms only in women.[21] Similarly, a more recent study suggested that the risk of female patients with sleep-disordered breathing for depressive disorder was particularly high.[22]

The purpose of this study was to investigate the association between sleep apnea severity and depressive symptoms in a study of middle-aged and elderly persons. Studying this association in an existing cohort increased the generalizability of our findings and created the possibility of extensive adjustment for confounders. We hypothesized that more severe sleep apnea, as measured with AHI, is related to more depressive symptoms. In addition, we assessed whether these associations differed between the sexes, as men typically are more prone to have sleep apnea whereas women typically report more depressive symptoms.

Participants and methods

Study sample

The current study is embedded in the Rotterdam Study (RS), a population-based cohort that investigates common health problems among middle-aged and elderly persons. In 2000, a new cohort was started by inviting all inhabitants 55 years and older (RS-II); and in 2006, the study was further extended by inviting inhabitants 45 years and older (RS-III). Participants were invited every 4 to 5 years for various medical examinations, such as, cardiovascular, endocrine, neurological, ophthalmic, psychiatric, and respiratory examinations.[23] All participants signed a written informed consent form before taking part in this study. The study was conducted in accordance with the guideline proposed in the World Medical Association Declaration of Helsinki and approved by the medical ethics committee according to the Wet Bevolkingsonderzoek ERGO (Population Study Act Rotterdam Study), executed by the Ministry of Health, Welfare, and Sports of the Netherlands.

From January 2012 until July 2013, 876 persons from the second cohort (RS-II, third examination) and the third cohort (RS-III, second examination) were invited to participate in a polysomnnographic (PSG) sleep study; 500 participants (57.1%) agreed. Women (53.8% vs 61.2%, $P = .028$) were less likely to participate in the PSG sleep study. Persons who participated in the PSG study did not significantly differ in age or prevalence of depressive symptoms from persons who refused to participate in the PSG study. We excluded 9 participants–the PSG recording was of insufficient quality in 7 participants, and we had no information on depressive symptoms in 2 persons. In total, data of 491 participants were available for analysis.

Assessment of sleep apnea

A home visit for the ambulant PSG was planned within 6 months of agreeing to enter the PSG sleep study. During the home visit, a trained research assistant placed the sensors and prepared the Vitaport 4 (Temec, Kerkrade, the Netherlands) to record a PSG according to the American Academy of Sleep Medicine guidelines.[24] The ambulant PSG included 6 electroencephalography channels—F3/A2, F4/A1, C3/A2, C4/A1, O1/A2, and O2/A1—bilateral electrooculography, electromyography, electrocardiography, respiratory belts on the chest and abdomen, oximetry, and a nasal pressure transducer and oronasal thermocouple to measure airflow. All recordings were scored according to American Academy of Sleep Medicine guidelines by one Registered Polysomnographic Technologist (HW), who was blinded to all participant characteristics.

The PSG recordings were scored in 30-second epochs for identification of sleep stages; each epoch was scored as W, N1, N2, N3, or REM sleep. Respiratory events were manually scored by the Registered Polysomnographic Technologist. An *apnea* was defined as a reduction of airflow of at least 90% of the baseline lasting at least 10 seconds. A *hypopnea* was defined as a reduction in airflow of at least 30% of the baseline for at least 10 seconds and a desaturation of at least 3% of the pre-event baseline or an arousal.[24] The *AHI* was defined as the number of apnea/hypopnea episodes per hour of sleep and was calculated using Prana software (PhiTools, Strasbourg, France).

Assessment of depressive symptoms

During a home interview, all participants were screened for depressive symptoms with the validated, Dutch version of the Center for Epidemiologic Studies Depression (CES-D) scale.[25,26] The CES-D consists of 20 questions that measure negative affect, lack of positive affect, and interpersonal and somatic problems scored on a 0 to 3 scale according to their severity, with a higher score indicating more depressive symptoms. If a person did not answer 1 to 5 questions (maximum 25% missing), we used the weighted average as the total score. In addition, we used a cutoff score of equal to or greater than 16 to dichotomize the depressive symptoms score. This cutoff score of 16 had high sensitivity (100%) and high weighted specificity (87%) in a sample aged 55 to 87 years.[26] For this article, we have defined *persons with clinically relevant depressive symptoms* as either people who had a CES-D score equal to or greater than 16 or people who reported the use of psychoanaleptics in the home interview.

Assessment of confounders

Partnership, employment status, education, body mass index (BMI), alcohol consumption, coffee, current smoking, cognitive functioning, diabetes mellitus, myocardial infarction, fatigue, total sleep time, and medication were selected based on previous studies to control for confounding.[27–29] During home interviews, all participants were asked about their partnerships, employment status, education, smoking, and use of medication. Education was subdivided into low, intermediate, and high education. Body mass index (kg/m^2) was calculated by measuring height and weight without shoes and heavy clothing. Alcohol, coffee, and sleep medication consumption were self-rated and indicated whether the participant used alcohol, coffee, or sleep medication at the night of the PSG. The current smoking variable indicated whether the participant smoked cigarettes, cigars, or pipe at the moment of the interview. During a center visit, cognitive functioning was measured with the Mini-Mental State Examination.[30] Total sleep time was estimated with PSG at the same night as the determination of the AHI. Fatigue was self-rated through a question at the evening before the PSG. Covariates were only included in the statistical model if they changed effect estimates by more than 10% or demonstrated a $P < .10$.

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