Journal of Occupational Health Psychology

Indirect Effects of Obstructive Sleep Apnea Treatments on Work Withdrawal: A Quasi-Experimental Treatment Outcome Study

Erica L. Carleton and Julian Barling

Online First Publication, April 30, 2020. http://dx.doi.org/10.1037/ocp0000183

AMERICAN PSYCHOLOGICAL ASSOCIATION

Journal of Occupational Health Psychology

ISSN: 1076-8998

http://dx.doi.org/10.1037/ocp0000183

Indirect Effects of Obstructive Sleep Apnea Treatments on Work Withdrawal: A Quasi-Experimental Treatment Outcome Study

Erica L. Carleton
University of Saskatchewan

Julian Barling
Queen's University

The effect of sleep on work is now receiving appropriate research attention, yet most results have been based on community (i.e., nonclinical) populations. Based on previous findings that clinical treatment for diagnosed obstructive sleep apnea benefits sleep quality, we hypothesized that sleep quality would mediate the effects of such treatment on work withdrawal behaviors (i.e., emotional exhaustion, cognitive distraction, work neglect, and partial absenteeism). A total of 125 adults with potential sleep apnea, who were referred to a midsized hospital's sleep disorders laboratory, participated in this 3-wave (pretest, posttest 1 month following initial treatment, and a follow-up 3 months later), quasi-experimental study. Clinical assessment using pretest data resulted in 83 participants being diagnosed with sleep apnea and receiving treatment (i.e., continuous positive airway pressure, $n = 62$; or positional therapy, $n = 21$); 42 patients who were not diagnosed with sleep apnea comprised the control group. Consistent with our hypotheses, treatment positively affected sleep quality, which in turn decreased emotional exhaustion, cognitive distraction, and partial absenteeism (but not work neglect). We discuss the implications of these findings for future research on sleep and its work-related consequences and organizational practice.

Keywords: sleep quality, sleep apnea, work withdrawal behaviors, continuous positive airway pressure, positional therapy

Employee work withdrawal is a costly organizational outcome that has long intrigued organizations and researchers (Berry, Lelchook, & Clark, 2012). Nonetheless, what constitutes work withdrawal remains somewhat uncertain. Work withdrawal has traditionally included visible behaviors where individuals are absent from work in some manner, such as lateness, full-day absenteeism, and turnover (Johns, 2001). Not all withdrawal behaviors, however, are as visible. Less visible work withdrawal behaviors (e.g., emotional exhaustion, cognitive distraction, work neglect) involve being "physically at work but not productive" (LeBlanc, Barling, & Turner, 2014, p. 401), and are potentially more costly to organizations because they are more difficult to monitor and manage (Carleton & Barling, 2016).

Understanding the antecedents of visible and less visible work withdrawal is important, and we explore how sleep apnea, one of the most frequently diagnosed sleep disorders, indirectly affects work withdrawal. Specifically, we investigate in a quasi-experimental longitudinal study how sleep apnea interventions indirectly affect work withdrawal behaviors through improved sleep quality. Our research examining an applied sleep intervention and its impact on work withdrawal (a) adds to the few studies on applied interventions on sleep and work, (b) extends our understanding of the consequences of sleep in the workplace, and (c) broadens the conceptualization of work withdrawal to include less visible forms of work withdrawal

Sleep and Work

The past decade has witnessed increased research on the pervasive interdependence of sleep problems (e.g., reduced sleep quality, increased daytime sleepiness) and workplace attitudes, affect, and behaviors (Barling, Barnes, Carleton, & Wagner, 2016). As a result, we now know that diminished sleep quantity and quality predict employee job dissatisfaction, diminished citizenship behaviors and workplace deviance, and burnout (Barnes, Miller, & Bostock, 2017). However, these findings have primarily been derived from nonclinical populations; we know very little about how sleep disorders such as sleep apnea affect work.

With very few exceptions (Garbarino, Guglielmi, Sanna, Mancardi, & Magnavita, 2016), there has been no focus on how clinically diagnosed sleep disorders affect work-related out-

Erica L. Carleton, Edwards School of Business, University of Saskatchewan; Julian Barling, Smith School of Business, Queen's University.

The authors express their appreciation to Michael Fitzpatrick, Helen Driver and Sydney Vogel for the assistance in conducting the study, and to Christopher M. Barnes, Amy Akers, Nick Turner and Melissa Trivisonno for constructive feedback. Financial assistance from the Social Sciences and Humanities Research Council of Canada is gratefully acknowledged. Portions of this research were presented at Conference of the World Sleep Society, Vancouver, 2019 and the Society for Industrial and Organizational Psychology, 2018.

Correspondence concerning this article should be addressed to Erica L. Carleton, Edwards School of Business, University of Saskatchewan, Saskatoon, SK S7N 5A7, Canada. E-mail: carleton@edwards.usask.ca

comes. This is a significant omission: *The International* Classification *of Sleep Disorders* (*ICSD-3*; American Academy of Sleep Medicine, 2014) identified more than 80 different sleep disorders and problems, with daytime sleepiness affecting approximately 15% to 24% of the U.S. adult population (Roth et al., 2011), with a steady increase in daytime sleepiness in the U.S. adult population (Ford, Cunningham, Giles, & Croft, 2015). Moreover, the prevalence of sleep disorders is increasing: In the United States, for example, the estimated prevalence of sleep disordered breathing has increased over the last 2 decades, with relative increases of between 14% and 55% depending on the subgroup (Peppard et al., 2013). As well, the number of adults in Canada diagnosed with sleep apnea more than doubled (3% to 6.4%) from 2009 to 2017 (Statistics Canada, 2018). Thus, the goal of our study is to examine how treatment for obstructive sleep apnea indirectly reduces work withdrawal through its beneficial effects on sleep quality.

Obstructive Sleep Apnea

Sleep disordered breathing is one of the most common sleep disorders in the general adult population. Obstructive sleep apnea is the most frequent category of sleep-disordered breathing, affecting 4% of adult males and 2% of adult females (*ICSD-3*; American Academy of Sleep Medicine, 2014). Sleep apnea is characterized by pauses in breathing during sleep that occur five to 30 times or more per hour and lasts between 10 to 30 seconds before the brain corrects the problem (Driver, 2016). With each episode, blood oxygen levels are reduced (hypoxia), and sleep is disturbed as individuals wake briefly to resume breathing. Because sleep quality is reduced over time, chronic sleep deprivation can develop, and sleep apnea is associated with negative work-related outcomes including stress and burnout (Guglielmi, Jurado-Gámez, Gude, & Buela-Casal, 2014), workplace injuries (Garbarino et al., 2016), missed work time and absenteeism (Jurado-Gámez, Guglielmi, Gude, & Buela-Casal, 2015), productivity and performance (Jurado-Gámez et al., 2015).

Treatment for sleep apnea is now routine, accessible in developed societies, and effective. The gold standard in treatment is continuous positive airway pressure (CPAP; Canessa et al., 2011). CPAP therapy keeps airways open during the night by providing a constant air through a mask worn while asleep, preventing airways from collapsing while breathing during sleep, thereby decreasing nightly awakening and improving sleep quality (Salepci et al., 2013). Effects are often immediate: In a randomized, prospective, double-blind, placebo-control study, sleep apnea symptoms and sleep quality improved after the first night of treatment (Loredo, Ancoli-Israel, Kim, Lim, & Dimsdale, 2006).

A second behavioral treatment for sleep apnea is positional therapy (Epstein et al., 2009). The influence of body position on sleep apnea is well established (Heinzer et al., 2012); symptom severity increases when sleeping in a supine position due to greater upper airway collapsibility (Joosten, O'Driscoll, Berger, & Hamilton, 2014). Positional therapy increases the likelihood that patients sleep on their sides (Epstein et al., 2009) through the use of positioning devices (e.g., pillow, backpack; Heinzer et al., 2012). Positional therapy is an effective secondary therapy for sleep apnea in patients with mild sleep apnea and/or poor compliance using a CPAP machine (Epstein et al., 2009). Although CPAP treatment is the gold standard, positional therapy is less invasive and less costly, thereby enjoying greater compliance and cost effectiveness (Barnes et al., 2017).

Treatment for sleep apnea provides a unique opportunity to assess the role of changes in sleep quality on work withdrawal behaviors, and we assign a critical mediating role to sleep quality: Because sleep quality improves following treatment for sleep apnea (Loredo et al., 2006), we expect that sleep treatment will reduce symptoms of sleep apnea, enhance sleep quality, and thereby improve work withdrawal behaviors.

Sleep and Work Withdrawal Behaviors

Poor sleep quality affects absenteeism (Åkerstedt, Kecklund, Alfredsson, & Selen, 2007; Jurado-Gámez et al., 2015). However, work withdrawal is a multidimensional concept that also includes less visible behaviors (i.e., emotional exhaustion, cognitive distraction, work neglect, taking extending breaks). Less visible forms of withdrawal are especially important with respect to sleep, as people are likely to attend work while suffering from poor sleep quality because feeling sleepy is not viewed as an illness and thus not viewed as a legitimate reason for absence.

To understand how sleep apnea and thus sleep quality are related to work withdrawal, we turn to the sleep physiology literature. Although nocturnal events inherent in sleep apnea are the focus of clinicians, daytime consequences of sleep apnea are important to patients who experience significant impairments in cognitive performance and quality of life (Silva, Goodwin, Vana, & Quan, 2016). Daily work functioning requires effortful exertion of the prefrontal cortex (Baumeister & Vohs, 2011), the region responsible for executive functioning (Domenech & Koechlin, 2015). Sleep apnea directly impairs the prefrontal cortex and thus executive functioning (Olaithe & Bucks, 2013). As a result, cognitive and emotional control tasks (i.e., executive functioning) are vulnerable to specific failures that go beyond those resulting from general low arousal and sleepiness (Lim & Dinges, 2010). Olaithe and Bucks' (2013) meta-analysis showed that all five domains of executive function (i.e., shifting, updating, inhibition, generativity, and fluid reasoning) had medium-to-large impairments owing to sleep apnea that were independent of age and disease severity. Moreover, treatment for sleep apnea reduces sleep apnea–related executive function difficulties (Olaithe & Bucks, 2013): Specifically, sleep quality, cognitive performance, vigilance, mental flexibility, attention, and mood all improve following sleep treatment (Bucks, Olaithe, & Eastwood, 2013; Davies & Harrington, 2016).

We expect that the initial changes in sleep quality following treatment for sleep apnea will mediate any subsequent effects of on work-related withdrawal (Figure 1). We focus on sleep quality because reduced sleep quality, not sleep quantity, is a symptom of sleep apnea (*ICSD-3*; American Academy of Sleep Medicine, 2014). This is because intermittent hypoxia and sleep fragmentation constitute the major pathophysiological consequences of obstructive sleep apnea, which cause reduced sleep quality (Ta-

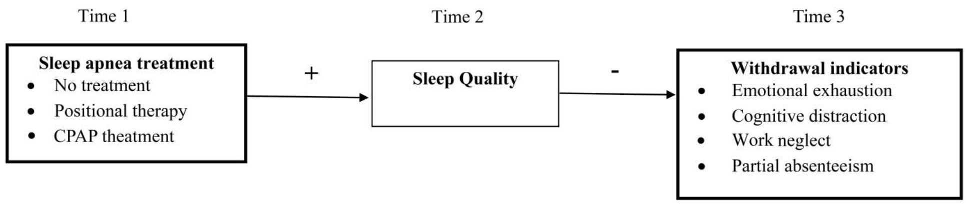

Figure 1. Conceptual model linking sleep apnea treatment to withdrawal indicators through sleep quality. CPAP = continuous positive airway pressure.

chikawa et al., 2017). Thus, sleep quality should improve following sleep apnea treatment.

Sleep quality and emotional exhaustion. As previously stated, poor quality sleep influences executive functions, as well as the amygdala, which controls emotional responses (Yoo, Gujar, Hu, Jolesz, & Walker, 2007), as a result of which we suggest that poor sleep quality will be associated with emotional exhaustion. The link between sleep quality and emotional exhaustion is supported by prior research. Of specific relevance to the current research, individuals diagnosed with sleep apnea experienced higher levels of emotional exhaustion (Guglielmi et al., 2014). Thus:

> *Hypothesis 1:* Sleep quality mediates the effects of the sleep apnea intervention on emotional exhaustion.

Sleep quality and cognitive distraction. We also suggest that poor sleep quality leads to cognitive distraction. Poor sleep quality is associated with greater vulnerability to cognitive failures and lapses in attention (Harrison & Horne, 2000; Lim & Dinges, 2010). For example, experimentally induced sleep deprivation (Beaumont et al., 2001) and prolonged periods of wakefulness decrease sustained attention in laboratory and field setting (Gobin, Banks, Fins, & Tartar, 2015). As well, people classified as at-risk for sleep disorders, including sleep apnea, are more likely to report difficulty concentrating (Swanson et al., 2011).

> *Hypothesis 2:* Sleep quality mediates the effects of the sleep apnea intervention on cognitive distraction.

Sleep quality and work neglect. Poor sleep quality depletes cognitive resources necessary for optimal performance including vigilance (Nilsson et al., 2005). We suggest that decreases in vigilance will lead to work neglect when individuals experience poor quality sleep. In terms of the work context, sleep deprivation is negatively associated with task performance (Kessler et al., 2011; Pilcher & Huffcutt, 1996) and positively associated with social loafing (Hoeksema-van Orden, Gaillard, & Buunk, 1998). Thus, we expect that improving sleep quality will result in diminished work neglect. In doing so, we conceptualize neglect as unintentional rather than intentional (i.e., similar to counterproductive work behaviors).

> *Hypothesis 3:* Sleep quality mediates the effects of the sleep apnea intervention on unintentional work neglect.

Sleep quality and partial absenteeism. We posit that sleep quality predicts partial absenteeism (i.e., arriving late, leaving early, taking extended breaks). First, the negative effects of sleep quality endure throughout the day (De Valck & Cluydts, 2003), potentially causing people to take breaks during the workday or leave work early. Second, poor sleep quality makes it difficult to stay awake, as a result of which people may take unscheduled breaks or naps. Although there is less research on partial absenteeism than absenteeism, the 2008 Sleep in America Poll survey of 1,000 employees indirectly supports the link between sleep apnea and partial absenteeism: 29% of respondents fell asleep or were significantly drowsy at work, 12% arrived late to work as a result of sleepiness, and 4% left work early as a result of sleepiness and sleep disorders such as sleep apnea (Swanson et al., 2011).

> *Hypothesis 4:* Sleep quality mediates the effects of the sleep apnea intervention on partial absenteeism.

Last, we do not expect the sleep apnea interventions will exert direct effects on any of the withdrawal behaviors, as there is no reason to suggest that physiological sleep changes directly affect workplace behaviors. Indeed, one cognitive–behavioral sleep intervention for insomnia only exerted indirect effects on organizational citizenship behavior and interpersonal deviance (Barnes et al., 2017).

Method

Participants and Design

We recruited individuals referred to a hospital's sleep disorders laboratory by their family physician for an overnight sleep assessment for suspected sleep disordered breathing (i.e., sleep apnea). Participants completed an overnight sleep polysomnography to diagnose sleep apnea (Leger, Bayon, Laaban, & Philip, 2012). The first set of questionnaires was completed before polysomnography began (prediagnosis). The first postintervention assessment took place 1 month following treatment; the second postintervention took place 3 months later.

Adults referred to the sleep disorders laboratory who were employed >30 hr a week were invited to participate in this study. Inclusion criteria in this study were an Epworth Sleepiness score >5 (Johns, 1991) and the ability to apply the Level III monitoring equipment, a portable monitoring device used to diagnose obstructive sleep apnea without supervision after initial training. Exclusion criteria included coexisting sleep disorders known to cause daytime sleepiness (e.g., insomnia), other serious medical condition (e.g., Chronic obstructive pulmonary disease (COPD), congestive heart failure), or psychiatric disorders associated with sleep disorders (e.g., depression). Participants were informed that treatment was not dependent on completing surveys at any stage and were entered into a draw for an iPad for participation in the study.

Initially, 125 participants agreed to participate, of whom 91 completed all three assessments (Figure 2). Of the initial 125 participants, 83 were diagnosed with sleep apnea, 62 of whom received CPAP treatment, and 21 received positional therapy. Decisions about assignment to treatment group were made independently by a sleep physician based on severity of sleep apnea gleaned from objectives sleep measures (e.g., apnea hypopnea index, REM sleep, total sleep time). The 42 participants not diagnosed with sleep apnea received no treatment and comprised the control group. Average age of the participants (61% males) was 45.04 years ($SD = 10.6$); average tenure of employment was 10.56 years ($SD = 10.10$).

Procedure

Participants' overnight appointment at the clinic allowed for a full polysomnography study.[1] Preintervention assessment gathered information about general medical history, medication usage, and demographic data. Baseline measures of sleep apnea and sleep quality, and work withdrawal measures, were collected.

Approximately 1 month following their overnight sleep study, patients received their diagnosis. Sleep apnea diagnoses ($n = 83$) were based on the *ICSD-3* (American Academy of Sleep Medicine, 2014), which includes self-reported sleep problems (e.g., sleepiness), specific results on the polysomnography (PSG) study along with other criteria.[2] Participants assigned to receive CPAP therapy received instructions in the use of the CPAP unit. Those prescribed positional therapy received education on how to sleep on their sides; both groups had a follow-up clinic appointment with a sleep specialist within 2 to 8 months of their overnight sleep study to monitor progress and treatment compliance. Those not diagnosed with sleep apnea (control group; $n = 42$) were asked to wait for a follow-up assessment to determine the cause of their sleep symptoms (Figure 2). There were no differences between the three groups regarding baseline self-reports of sleep apnea or sleep quality, or the four withdrawal variables. There were differences regarding hours worked, age, and tenure, and thus hours worked and age were controlled in all analyses.

Measures

Sleep apnea was diagnosed by the professional team based on the results of the overnight sleep PSG. Sleep PSG studies involve a comprehensive recording of the biophysical changes that occur during sleep, including brain waves, eye movements, heart rate, breathing pattern, blood oxygen level, body position, chest and abdominal movement, limb movement, and snoring and other noises made while sleeping.

Manipulation check. We used one item (i.e., "heavy snoring") from the Karolinska Sleep Questionnaire (Åkerstedt et al., 2002; where 1 = *never*, 5 = *always*) as a manipulation check for the two interventions. Heavy snoring is an appropriate manipulation check because snoring is the main symptom of sleep apnea (Romero, Krakow, Haynes, & Ulibarri, 2010).

Sleep quality was measured with the Karolinska Sleep Questionnaire's six-item Sleep Quality subscale (Åkerstedt et al., 2002). Questions (e.g., "Disturbed/restless sleep") were answered on a 5-point scale (1 = *never*, 5 = *always*). This scale has good internal consistency and construct validity and is used in clinical settings to assess sleep quality, nonrestorative sleep, sleep apnea, sleepiness, and nocturnal symptoms of insomnia (Nordin, Åkerstedt, & Nordin, 2013).

Emotional exhaustion. We used the three-item Emotional Exhaustion subscale from Melamed, Shirom, Toker, and Shapira's (2006) Burnout Measure. All items (e.g., "I feel I am not capable of investing emotionally in coworkers and customers") are rated on a 7-point frequency scale (1 = *almost never*, 7 = *almost always*).

Cognitive distraction was measured using the Cognitive Distraction subscale from Melamed et al.'s (2006) Burnout Measure. All five items (e.g., "I have difficulty concentrating") are rated on a 7-point frequency scale (1 = *almost never*, 7 = *almost always*).

Work neglect was measured with seven items from two scales. Three items were used from Kammeyer-Mueller and Wanberg's (2003) Work Withdrawal Scale (e.g., "fail to attend scheduled meetings") and four from Barling, Rogers, and Kelloway (2001; e.g., "stayed out of sight to avoid work"). Responses were measured on a 7-point scale (1 = *not at all*; 7 = *all of the time*).

Partial absenteeism. A modified version of Hepburn and Barling's (1996) five-item scale was used to assess partial absenteeism (e.g., "In the past month have you come in late?", "Take unauthorized extended breaks") One item from this scale was deleted ("How often have you been distracted at work?") because it is identical to an item used to measure cognitive distraction. Responses were measured on a 7-point scale (1 = *never*, 7 = *all of the time*).

Results

Intercorrelations and reliability of all variables for all three time periods appear in Table 1; descriptive statistics appear in Table 2.

[1] PSG studies are multiparametric tests used to diagnose sleep disorders. It involves a comprehensive recording of the biophysical changes that occur during sleep (http://www.nhlbi.nih.gov/health/health-topics/topics/slpst/types.html). Recordings were conducted using Sandman Elite SD32+ digital sleep recording system (Natus [Embla]; Ottawa, Canada), and include four electroencephalography channels (C4-A1, C3-A2, O2-A1, F3-A2), two electrooculography channels (ROC-A1, LOC-A2), submental electromyography, intercostal (diaphragmatic surface) EMG, bilateral anterior tibialis electromyography, electrocardiogram, respiratory piezo bands (chest and abdomen), finger pulse oximetry, a vibration snore sensor, nasal pressure airflow, and oronasal thermocouple. PSG recordings were conducted as either a diagnostic study or, in the event of severe sleep apnea, a split-night study. For split-night studies, the diagnostic period is followed by the introduction of treatment during the night.

[2] Diagnostic criteria for primary central sleep apnea (*ICSD-3*) Criteria A to D must be met: (A) The presence of at least one of the following: (a) sleepiness; (b) difficulty initiating or maintaining sleep, frequent awakenings or non-restorative sleep; (c) awakening short of breath; (d) snoring; and (e) witnessed apneas. (B) PSG demonstrates all of the following: (a) five or more central apneas and/or central hypopneas per hour of sleep (PSG), (b) the number of central apneas and/or central hypopneas is $>50\%$ of the total number of apneas and hypopneas, and (c) absence of CSB. (C) There is no evidence of daytime or nocturnal hypoventilation. (D) The disorder is not explained more clearly by another current sleep disorder, medical or neurological disorder, medication use or substance use disorder.

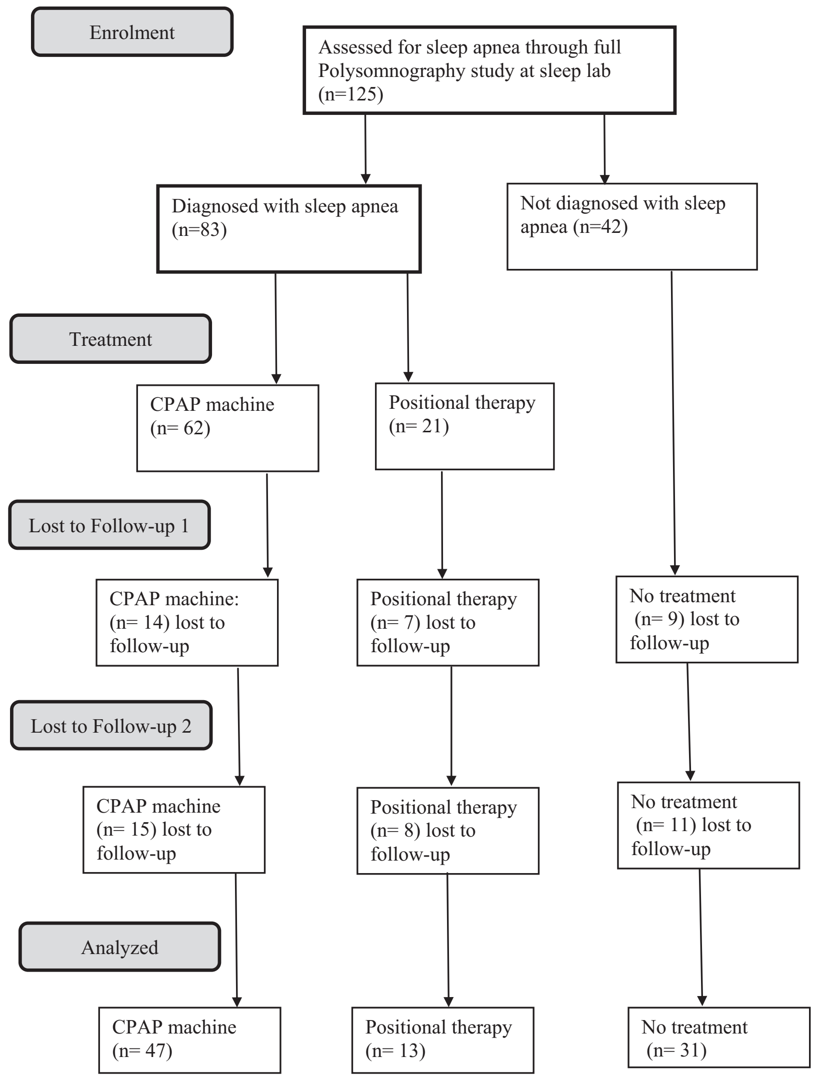

Figure 2. Flow diagram of participants. CPAP = continuous positive airway pressure.

Before computing preliminary analyses, we tested the normality of the data, and all the data satisfied the assumption of normality.[3]

Preliminary Analyses

Attrition analysis. We initially conducted multivariate analyses of variance to assess whether participants who completed all three waves of data differed from those who did not in terms of salient variables. No between-groups differences emerged regarding demographic characteristics (age, education, income, tenure in current position, hours worked per week), Pillai's $F(5, 105) = 1.227$, $p = .302$, $\eta^2 = .055$; initial sleep variables (sleep apnea, sleep quality), Pillai's $F(2, 120) = 0.693$, $p = .502$, $\eta^2 = .011$; or the preintervention outcome measures (emotional exhaustion, cognitive distraction, partial absenteeism, work neglect), Pillai's $F(4, 113) = 0.378$, $p = .824$, $\eta^2 = .013$. In addition, no sex differences emerged between those who completed all three waves and those who did not ($\chi^2 = .299$, $df = 1$, $p < .05$), suggesting that nonrandom attrition was not an issue.

Nonrandom assignment to conditions. Because we used a quasi-experimental design, we computed multivariate analyses of variance to assess whether pretreatment differences existed between the treatment and no-treatment control groups in terms of demographic variables: $F(5, 104) = 1.636$, $p = .098$, $\eta^2 = .072$. Post hoc analyses showed that participants in the CPAP treatment group were significantly older than those in the positional treat-

[3] To test normality of the data for the preliminary analyses, we examined the residuals of the independent variables (sleep apnea and sleep quality) in the preliminary analyses. We examined the skew and kurtosis of the residuals of both sleep apnea and sleep quality at all three time points. All values of skewness and kurtosis were less than 1 *SE*, which suggests that the values are not significantly different than the value of 0 from a normal distribution. We also examined the Shapiro–Wilk test for normality, the preferred normality test (Ghasemi & Saleh Zahediasl, 2012), which was nonsignificant for all three time points of sleep apnea and sleep quality. In addition, the Normal Q-Q Plots show that the residuals closely follow the diagonal which is the condition from the true normal distribution. Full results from these analyses are available from Erica L. Carleton on request.

Table 1
Intercorrelations for All Study Variables at All Three Time Periods (N = 104)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. Age	—																					
2. Hours	.10	—																				
3. AHI	.13	.03	—																			
4. TST	−.16	.11	−.55**	—																		
5. OSA T1	−.03	.07	.13	−.03	—																	
6. OSA T2	−.11	−.07	−.30**	.35**	.30**	—																
7. OSA T3	−.17	−.11	−.26*	.30**	.24*	.74**	—															
8. SQ T1	−.02	−.11	−.04	.01	.54**	.25*	.17	**(.80)**														
9. SQ T2	−.10	−.13	−.28**	.31**	.26**	.69**	.53**	.55**	**(.86)**													
10. SQ T3	−.03	−.12	−.27*	.39**	.19	.59**	.69**	.48**	.77**	**(.87)**												
11. EE T1	−.15	.07	−.09	.23*	.39**	.12	−.02	.39**	.15	.15	**(.91)**											
12. EE T2	−.08	.01	−.14	.26*	.06	.32**	.23*	.08	.30**	.28**	.43**	**(.95)**										
13. EE T3	−.05	.08	−.11	.17	.19	.24*	.31**	.20	.37**	.42**	.45**	.57**	**(.96)**									
14. CE T1	−.03	.04	−.25**	.26**	.22*	.26**	.21*	.49**	.37**	.41**	.48**	.24*	.25*	**(.87)**								
15. CE T2	.17	−.03	−.21*	.29**	.15	.40**	.32**	.28**	.43**	.45**	.30**	.57**	.41**	.57**	**(.91)**							
16. CE T3	.06	−.00	−.24*	.39**	.23*	.43**	.42**	.32**	.55**	.57**	.36**	.48**	.67**	.55**	.61**	**(.93)**						
17. WN T1	−.03	.08	.01	−.09	−.11	−.18	−.06	−.18*	−.33**	−.26*	−.19*	−.15	−.06	−.12	−.35**	−.16	**(.74)**					
18. WN T2	−.02	.14	.04	−.04	−.22*	−.10	−.05	−.24*	−.28*	−.13	−.14	−.05	.02	−.10	−.15	−.03	.62**	**(.78)**				
19. WN T3	−.07	.17	.10	−.13	−.09	.04	.19	−.04	−.04	.09	−.17	.06	.17	.04	−.01	.06	.59**	.60**	**(.76)**			
20. PA T1	−.07	.04	−.05	.00	−.04	.01	.09	−.06	−.09	−.10	−.03	.01	.07	.07	−.13	.02	.57**	.32**	.27*	**(.70)**		
21. PA T2	−.02	.06	−.06	−.05	−.06	.15	.11	.00	−.04	.04	.06	.10	.22	.15	−.03	.21	.31**	.51**	.42**	.72**	**(.80)**	
22. PA T3	−.08	−.02	−.05	−.08	−.05	.27*	.14	.06	.19	.11	.01	.05	.16	.18	.03	.18	.24*	.29**	.41**	.67**	.69**	**(.79)**

Note. T1 = Time 1; T2 = Time 2; T3 = Time 3; AHI = apnea hypopnea index; TST = total sleep time; OSA = obstructive sleep apnea; SQ = sleep quality; EE = emotional exhaustion; CE = cognitive distraction; WN = work neglect; PA = partial absenteeism. Cronbach's α indicated on the diagonal using boldface.
* $p < .05$ level (two-tailed). ** $p < .01$ level (two -tailed).

Table 2
Means and Standard Deviations Across All Three Time Periods

Variables	Time 1			Time 2			Time 3		
	n	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
Age	124	45.04	10.62	—	—	—	—	—	—
Hours worked	124	43.59	9.69	—	—	—	—	—	—
Sleep apnea	123	2.97	0.71	96	2.36	.75	91	2.22	.76
Sleep quality	123	3.12	0.85	96	2.52	.87	91	2.39	.85
Emotional exhaustion	123	2.07	0.90	95	1.95	.92	91	1.79	.90
Cognitive distraction	123	2.55	0.97	95	2.24	.87	91	2.03	.83
Work neglect	120	1.81	0.52	93	1.65	.48	87	1.63	.44
Partial absenteeism	120	1.71	0.53	93	1.55	.53	87	1.58	.49

ment and control groups (CPAP group, $M = 47.02$ years, $SD = 10.33$; positional therapy group, $M = 40.83$ years, $SD = 10.92$; control group, $M = 42.03$ years, $SD = 10.28$; $F(2, 108) = 3.778$, $p = .026$, $\eta^2 = .066$) and had significantly more tenure in their current position (CPAP group, $M = 12.73$ years, $SD = 11.24$; positional therapy group, $M = 5.44$ years, $SD = 5.63$; control group, $M = 7.37$ years, $SD = 6.80$), $F(2, 108) = 6.15$, $p = .003$, $\eta^2 = .102$. In addition, both the CPAP and positional treatment groups worked more hours per week than the control group (CPAP group, $M = 45.57$, $SD = 10.03$; positional therapy group, $M = 42.14$ hr, $SD = 8.69$; control group, $M = 40.91$ hr, $SD = 8.02$), $F(2, 108) = 3.08$, $p = .050$, $\eta^2 = .054$. However, no differences emerged between the three groups in terms of sleep apnea or sleep quality, $F(4, 240) = 1.524$, $p = .196$, $\eta^2 = .025$, or the four withdrawal variables, $F(8, 226) = 1.371$, $p = .210$, $\eta^2 = .046$.

Thus, to enhance internal validity, we control for age and number of hours worked per week in all subsequent analyses. Age is also controlled because it is correlated with sleep difficulties (Vitiello, 2009) and sleep apnea (Bixler, Vgontzas, Ten Have, Tyson, & Kales, 1998). We did not control for tenure due to its significant correlation with age, $r = .461$, $p = .000$.

Manipulation check. A necessary first step is to confirm the construct validity of the intervention. A repeated-measures analysis of variance (ANOVA) showed that after controlling for age and hours worked, there was a significant Treatment × Time interaction on the sleep apnea manipulation check, $F(2, 84) = 7.663$, $p = .001$, $\eta^2 = .154$, and a significant treatment effect, $F(2, 84) = 7.078$, $p = .001$, $\eta^2 = .144$. A one-way ANOVA revealed no significant differences on the sleep apnea manipulation check at Time 1 between participants in the control and two treatment conditions. However, significant differences emerged at Time 2, $F(2, 95) = 12.692$, $p = .000$, and Time 3, $F(2, 90) = 14.405$, $p = .000$, between those in the control condition and those in the two treatment conditions.

Effects of Sleep Apnea Treatment on Sleep Quality

We used a repeated-measures ANOVA to test the direct effects of the two treatments on sleep quality over time. The Treatment × Time effect was significant, $F(2, 84) = 4.495$, $p = .014$, $\eta^2 = .097$, as was the treatment effect, $F(2, 84) = 5.412$, $p = .006$, $\eta^2 = .114$. Post hoc univariate ANOVAs further confirmed the effects of the interventions: There were no differences in sleep quality between the three groups at T_1, $F(2, 117) = 2.648$, $p = .075$, $\eta^2 = .043$, but sleep quality significantly improved for those in the two treatments condition at both T_2, $F(2, 95) = 6.577$, $p < .002$, $\eta^2 = .128$, and T_3, $F(2, 85) = 7.489$, $p < .001$, $\eta^2 = .150$.

Effects of Sleep Apnea Treatment on Work Withdrawal

To respect the longitudinal nature of the data, initial levels of the respective outcome at T_1 are controlled in all analyses, as are age and hours worked. The indirect effects of CPAP treatment at T_1 on the respective outcome at T_3 through the mediating effects of sleep quality at T_2 were assessed using Hayes' (2018) PROCESS 3.1 macro. Statistical significances of all indirect effects were determined using bias-corrected 95% confidence interval (CI) and based on 5,000 bootstrapped resamples (MacKinnon, Lockwood, & Williams, 2004) because the sampling distribution of indirect effects is nonnormal. Unstandardized regression coefficients are reported for all effects. Because the independent variable had three conditions, we used the multicategorical option in which coding for the categorical variables was as follows: ×1 (positional treatment vs. other), ×2 (CPAP treatment vs. other).

We followed Hayes' (2018) recommendations in assessing the magnitude of the indirect effects. Given the categorical nature of the predictor variable, we used the partially standardized effect, which represents the magnitude of the indirect effect in terms of standard deviation units in Y (Preacher & Kelley, 2011) to assess the effect size of the indirect effects.

Emotional exhaustion. After controlling for age and hours worked, and T_1 emotional exhaustion, Hypothesis 1 was supported (Table 3): Positional treatment exerted a significant indirect effect on emotional exhaustion at T_3 through the mediating effects of T_2 sleep quality ($b = -.266$, $SE = .108$, 95% CI [−.502, −.082]), as did CPAP treatment ($b = -.201$, $SE = .102$, 95% CI [−.441, −.041]). Using the partially standardized effect size calculation, participants receiving CPAP treatment were 0.229 *SD*s lower in emotional exhaustion at T_3 than those who did not ($b = -.229$, $SE = .108$, 95% CI [−.469, −.049]); participants receiving positional therapy were 0.303 *SD*s lower on T3 emotional exhaustion compared with those who did not ($b = -0.303$, $SE = .115$, 95% CI [−.554, −.104]).

Cognitive distraction. After again controlling for age and hours worked per week, and T_1 cognitive distraction, Hypothesis 2 was supported (Table 4): Both positional treatment, $b = -.236$, $SE = .102$, 95% CI [−.458, −.063], and CPAP treatment exerted a significant indirect effect on cognitive distraction at T_3 through the mediating effects of T_2 sleep quality, $b = -.162$, $SE = .099$,

Table 3
Conditional Indirect Effects of Sleep Apnea Treatment Condition on Emotional Exhaustion Through Sleep Quality (N = 89)

Predictor	*b*	*SE*	*t*	Confidence interval *LL*	Confidence interval *UL*
Mediator variable: Sleep quality at Time 2					
X1 (Positional therapy vs. other)	−.81	.26	−3.10*	−1.33	−.30
X2 (CPAP vs. other)	−.61	.20	−3.09*	−1.01	−.22
Age	−.01	.01	−.96	−.03	.01
Hours	−.01	.01	−.97	−.03	.01
Emotional exhaustion at Time 1	.25	.10	2.41*	.04	.45
Model summary: R^2 = .21, F(5, 83) = 4.47, p = .001					
Outcome variable: Emotional exhaustion at Time 3					
X1 (Positional therapy vs. other)	.10	.27	.38	−.43	.63
X2 (CPAP vs. other)	.01	.20	.03	−.40	.41
Sleep quality	.33	.11	3.08*	.12	.54
Age	−.01	.01	−.12	−.02	.02
Hours	.01	.01	.70	−.01	.02
Emotional exhaustion at Time 1	.39	.10	3.78*	.18	.59
Model summary: R^2 = .29, *F*(6, 82) = 5.64, *p* = .001					
Indirect effect: Sleep apnea treatment condition—Emotional exhaustion via sleep quality					
X1 (Positional therapy vs. other)	−.27	.11		−.50	−.08
X2 (CPAP vs. other)	−.20	.10		−.44	−.04

Note. CPAP = continuous positive airway pressure. Unstandardized regression coefficients reported throughout.
* $p < .05$.

95% CI [−.386, −.005]. Cognitive distraction at T_3 was lowered by 0.198 *SD*s for those who receive CPAP treatment compared with those who did not ($b = -.198$, $SE = .117$, 95% CI [−.471, −.013]) and by 0.288 *SD*s for those who receive positional treatment compared to those who did not ($b = -.288$, $SE = .119$, 95% CI [−.553, −.079]).

Work neglect. Hypothesis 3 was not supported. The indirect effect of neither positional therapy ($b = -.046$, $SE = .080$, 95%

Table 4
Conditional Indirect Effects of Sleep Apnea Treatment Condition on Cognitive Distraction Through Sleep Quality (N = 89)

Predictor	*b*	*SE*	*t*	CI *LL*	CI *UL*
Mediator variable: Sleep quality at Time 2					
X1 (Positional therapy vs. other)	−.62	.25	−2.49*	−1.12	−.12
X2 (CPAP vs. other)	−.43	.18	−2.34*	−0.79	−.06
Age	−.01	.01	−1.31	−0.03	.01
Hours	−.01	.01	−1.09	−0.03	.01
Cognitive distraction T1	.33	.08	4.14*	0.17	.49
Model summary: R^2 = .30, *F*(5, 83) = 7.15, *p* = .001					
Outcome variable: Cognitive distraction Time 3					
X1 (Positional therapy vs. other)	−.03	.22	−0.14	−0.47	.40
X2 (CPAP vs. other)	−.06	.16	−0.35	−0.37	.26
Sleep quality	.38	.09	4.10*	0.20	.56
Age	.01	.01	1.63	−0.01	.03
Hours	.01	.01	0.08	−0.01	.01
Cognitive distraction T1	.32	.07	4.33*	0.17	.47
Model summary: R^2 = .46, *F*(6, 82) = 11.64, *p* = .001					
Indirect effect: Sleep apnea treatment condition–Cognitive distraction via sleep quality					
X1 (Positional therapy vs. other)	−.24	.10		−0.46	−.06
X2 (CPAP vs. other)	−.16	.10		−0.39	−.01

Note. CPAP = continuous positive airway pressure. Unstandardized regression coefficients reported throughout.
* $p < .05$.

CI [−.212, .101]) nor CPAP treatment on T_3 work neglect through T_2 sleep quality were significant ($b = -.036$, $SE = .063$, 95% CI [−.177, .074]).

Partial absenteeism. Hypothesis 4 was supported (Table 5). Both positional treatment ($b = -.212$, $SE = .098$, 95% CI [−.428, −.046]) and CPAP treatment exerted a significant indirect effect on partial absenteeism at T_3 through the mediating effects of T_2 sleep quality ($b = -153$, $SE = .076$, 95% CI [−.326, −.030]). Partial absenteeism at T_3 was lower by 0.331 *SD*s for those receiving CPAP treatment compared with those who did not ($b = -.331$, $SE = .155$, 95% CI [−.679, −.067]) and by 0.460 *SD*s for those who received positional treatment than those who did not ($b = -.460$, $SE = .197$, 95% CI [−.881, −.112]).Last, consistent with predictions about the indirect effects of CPAP treatment on work withdrawal, none of the direct effects from CPAP treatment or positional therapy on any of the T_3 outcomes were significant.[4]

Supplementary Analyses

Because sleep quality was significantly associated with nonrestorative sleep, $r = .621$, $p < .001$, daytime sleepiness, $r = .529$, $p < .001$, and sleep apnea, $r = .536$, $p < .001$, from the initial measurements on the Karolinska Sleep Questionnaire (Åkerstedt et al., 2002), we recomputed the indirect effect analyses reported earlier to assess whether these three variables played a mediating role, thereby providing a test of construct redundancy. In addition, although sleep quality and quantity were not related (r = −.050, $p = .587$), we investigated the potential mediating effect of sleep quantity, given its role in employees' counterproductive workplace behaviors (Barnes, Ghumman, & Scott, 2013). Only one of these 16 post hoc analyses (nonrestorative sleep on partial absenteeism) was significant for both positional therapy and CPAP treatment. Thus, the likelihood that construct redundancy is a threat is minimized, and only sleep quality consistently mediates the effects of treatment for sleep apnea on the three withdrawal behaviors.[5]

Discussion

Our goal was to examine whether and how two clinical interventions for obstructive sleep apnea reduce work withdrawal behaviors. The results provide strong support for the indirect effects of CPAP and positional therapy on work withdrawal. First, replicating findings from the sleep literature, both interventions had a significant effect on apnea symptoms. Second, both interventions had a significant effect on sleep quality. Third, the interventions reduced emotional exhaustion, cognitive distraction, and partial absenteeism (but not work neglect) through the mediating effects of sleep quality; the specific role of sleep quality is supported by the supplementary analyses that demonstrated none of the other measured sleep related variables served a similar mediating role as sleep quality.

This study contributes to our understanding of the link between sleep and work by (1) adding to a small but growing list of longitudinal studies (Barnes et al., 2017; Gunia, Barnes, & Sah, 2014; Welsh, Ellis, Christian, & Mai, 2014). (2) Most interventions that examined the relationship between sleep and work had a negative focus (e.g., examining the indirect effects of sleep loss on workplace injuries; Barnes & Wagner, 2009). Focusing on positive sleep interventions such as CPAP and positional therapy expands knowledge on sleep and organizational behavior (Barber, Taylor, Burton, & Bailey, 2017) and practical options available to organizations. (3) Because both Barnes et al.'s (2017) study on cognitive–behavioral therapy treatment for insomnia and the current study were based on controlled interventions, we can now move tentatively toward causal inferences about the effects of interventions for insomnia and sleep apnea on workplace outcomes. (4) Our findings reaffirm the mediating role of sleep quality because (a) sleep quality mediated the effects of the two different interventions, (b) the intervention exerted no direct effects on work withdrawal behaviors, and (c) sleep quantity did not fulfil a similar mediating role.

What remains unanswered is why the two sleep apnea treatments had no indirect effects on work neglect. We initially conceptualized work neglect as unintentional behaviors, and one possibility is that work neglect may reflect a more deliberate choice to disengage from work-related tasks (Barling et al., 2001).

Strengths and Limitations

There are several strengths inherent in this research. First a major methodological strength of our study is its longitudinal nature, with data collected across three time periods showing that the effects of the sleep apnea interventions lasted 3 months following treatment. Second, our multidimensional approach to work withdrawal behaviors provides insight regarding the differential effects of changes in sleep quality on different forms of work withdrawal. Previous research has largely focused on absenteeism or turnover, which are visible behaviors that are amenable to research and management in the workplace. The current findings point to the importance of expanding the conceptualization of work withdrawal to include less visible forms of work withdrawal that may predict future absenteeism and turnover (Berry et al., 2012). Third, assessments of sleep apnea were based on standard medical diagnostic criteria using polysomnographic analysis rather than self-report data. Last, unlike previous research using only one intervention for reducing any indirect effects of insomnia on work-related behaviors (Barnes et al., 2017), mono-operation threats to validity are minimized by showing similar indirect effects across different treatments.

Similar to all research based on quasi-experimental designs, conceptual and methodological strengths cannot exclude all possible threats to validity (Shadish, Cook, & Campbell, 2002). First, reactive self-report changes could threaten external validity: The self-reported obstructive sleep apnea item was completed by participants before they knew if they would be assigned to a treatment group, and motivation to received treatment—which provided treatment at no cost to participants—might have been high. As a result, participants may have intentionally or unintentionally inflated their response to this item. Some empirical support for this possibility emerges from the intercorrelation of T_1, T_2 and T_3 sleep

[4] Because there is some concern that including statistical controls in analyses might inadvertently influence the results (Becker et al., 2016), we re-computed all analyses reported without controlling for age and number of hours worked per week. Doing so had no substantive effect on results reported. All these results are available from Erica L. Carleton on request.

[5] These analyses are available from the senior author upon request.

Table 5
Conditional Indirect Effects of Sleep Apnea Treatment Condition on Partial Absenteeism Through Sleep Quality (N = 66)

Predictor	*b*	*SE*	*t*	Confidence interval *LL*	Confidence interval *UL*
Mediator variable: Sleep quality at Time 2					
X1 (Positional therapy vs. other)	−1.42	.36	−3.96*	−2.13	−.70
X2 (CPAP vs. other)	−1.02	.29	−3.46*	−1.61	−.43
Age	−.01	.01	−.52	−.03	.02
Hours	−.01	.01	−.62	−.03	.01
Partial absenteeism at Time 1	−.06	.21	−.31	−.47	.35
Model summary: R^2 = .241, $F(5, 60)$ = 3.815, p = .005					
Outcome variable: Partial absenteeism at Time 3					
X1 (Positional therapy vs. other)	.17	.17	1.00	−.17	.50
X2 (CPAP vs. other)	.03	.13	.21	−.24	.29
Sleep quality	.15	.05	2.81*	.04	.26
Age	.01	.01	.96	−.01	.01
Hours	−.01	.01	−1.10*	−.01	.01
Partial absenteeism at Time 1	.67	.09	7.87	.50	.84
Model summary: R^2 = .544, $F(6, 59)$ = 11.748, p = .001					
Indirect effect: Sleep apnea treatment condition—Partial absenteeism via sleep quality					
X1 (Positional therapy vs. other)	−.21	.10		−.43	−.05
X2 (CPAP vs. other)	−.15	.08		−.33	−.03

Note. CPAP = continuous positive airway pressure. Unstandardized regression coefficients reported throughout
* $p < .05$.

apnea scores because correlations of T_2 and T_3 scores with T_1 (r's = .297, and .235, respectively) were substantially lower than that between T_2 and T_3 (r = .739). However, reactive self-report changes pose no threat, as diagnostic decisions were based on the objective sleep data scores from participants' PSG study by sleep professionals and not this one-item scale

Second, the potential for nonrandom participant attrition exists; 34 of the original 125 participants at T_1 were lost to attrition by T_3 of this study. However, as there were no systematic differences in terms of demographic, sleep or work withdrawal indicators at T_1 between participants who provided data at all three time periods and those who did not, systematic attrition bias is unlikely.

Third, ethical considerations required that all participants diagnosed with sleep apnea receive treatment. Physicians' and care providers' professional and ethical obligations take precedence over those as a researcher, and once a sleep disorder diagnosis was made, treatment cannot be withheld (Miller & Weijer, 2006). We approached this ethical obligation by using a nonrandomized group of participants with self-identified sleep problems but no clinical diagnosis for sleep apnea as the control condition; potential threats to internal validity arising from the nonrandom assignment were minimized as no between-groups differences emerged regarding baseline self-reports of sleep apnea or sleep quality, or any of the four withdrawal variables. In addition, age and hours worked per week were controlled statistically in all analyses because of pretest differences.

Fourth, several threats to construct validity need to be addressed before causal inferences can be justified. (a) All data for sleep apnea (but not the diagnosis of obstructive sleep apnea), sleep quality, and the work withdrawal variables were based on participant self-reports. Mono-method bias is not an issue for several reasons. The diagnosis for sleep apnea and subsequent assignment to treatment was based on a combination of self-report, sleep and physiological data. In addition, the within-person nature of the design controls for possible response bias, and the two interventions reduce the likelihood that mono-operation bias might account for the results, whereas the use of self-reported data for withdrawal behaviors such as absenteeism are common, and evidence adequate reliability and validity (Johns & Miraglia, 2015). (b) Treatment diffusion might be an issue. Participants in the control group may have been motivated to deal with their sleep difficulties and purchased readily available CPAP or positional therapy devices themselves. In the extent to which future research uses a waitlist control groups, any threats to construct validity through reactive self-report changes and treatment diffusion would be further minimized. (c) Participants denied access to a sleep intervention who found themselves in the control condition might have been sufficiently motivated to improve their sleep (i.e., compensatory equalization) using other methods such as over-the-counter medication or cognitive–behavioral therapy self-help books, (Espie, 2006). Both treatment diffusion and compensatory equalization could underestimate the effects of the intervention, and future research should conduct postintervention manipulation checks to exclude such threats.

Directions for Future Research

Our findings point to several opportunities for further research. First, although some research shows correlations between objective and subjective sleep measures (Barnes, Schaubroeck, Huth, & Ghumman, 2011) others do not (Barber et al., 2017; Crain et al., 2014); future research on sleep apnea and work outcomes should use objective PSG data across the duration of the study. Where that is not possible, actigraphy recordings (small electronic units worn either on wrists or ankles that measure motion during sleep) may be useful.

Second, sleep quality mediated the effects of sleep apnea treatments on work withdrawal. In contrast, no support emerged for sleep quantity as a mediator and limited, and inconsistent support emerged for nonrestorative sleep and daytime sleepiness as mediators. While sleep apnea may be related to sleep quantity the relationship may be more complex because sleep apnea can also affect sleep quantity in different ways. For example, sleep duration in individuals with sleep apnea may be shortened because of frequent awakenings or it may be prolonged as compensation for poor sleep quality (Tachikawa et al., 2017). One recent study (Tachikawa et al., 2017) examining the effects of CPAP treatment on sleep quantity showed that only sleep quality, and not sleep quantity, was improved. However, there was one specific subgroup (i.e., those with shorter sleep duration, greater daytime sleepiness, and good CPAP adherence) that did enjoy enhanced sleep quantity. Thus, further examination of the relationship between sleep apnea, its treatment and sleep quantity is necessary.

Last, like Barnes et al. (2017), our findings establish how validated treatments for sleep disorders influence work outcomes. Establishing whether other treatments (e.g., mindfulness training) for different sleep disorders (e.g., restless leg syndrome; Bablas, Yap, Cunnington, Swieca, & Greenwood, 2016) affect workplace outcomes could extend the theoretical and organizational implications of this research.

Practical Implications

Both CPAP and positional treatment for sleep apnea indirectly reduced work withdrawal, offering organizations practical interventions if work withdrawal is a function of sleep disorders. However, although CPAP treatment remains the "gold standard" (Barnes et al., 2017), not all health insurance plans cover CPAP devices. Thus, the fact that similar benefits were obtained from positional therapy, a less expensive, less intrusive option enabling greater treatment compliance and significant cost effectiveness, may make it especially attractive to organizations. Nonetheless, making clinical interventions for sleep disorders available to employees through the organization is complicated. Organizations have traditionally avoided interfering in the private lives of their employees. However, organizations could ensure that employees have the resources (e.g., time, financial support) to seek effective treatment for clinically diagnosed sleep disorders through existing wellness or employee-assistance programs that provide information on sleep disorders and available treatment options. In doing so, organizations could compare the utility of specific (e.g., cognitive–behavioral therapy; Barnes et al., 2017) versus generalized (e.g., health promotion interventions; Olson et al., 2015) interventions on employees' sleep.

Conclusion

Our findings show that two home-based interventions for sleep apnea reduce work withdrawal. Added to this, Barnes et al.'s (2017) research demonstrates that Internet-based cognitive–behavioral therapy was effective in reducing insomnia. The practical utility of these three interventions should be highlighted: In the extent to which treatment for sleep disorders is effective, and financially and personally accessible (e.g., through Internet or home-based treatment), participants' and organizations' motivation to participate in treatment programs for sleep disorders will increase (Cohen, O'Leary, & Foran, 2010), with substantial benefits accruing to organizations and their members.

References

Åkerstedt, T., Kecklund, G., Alfredsson, L., & Selen, J. (2007). Predicting long-term sickness absence from sleep and fatigue. *Journal of Sleep Research, 16,* 341–345. http://dx.doi.org/10.1111/j.1365-2869.2007.00609.x

Åkerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L., & Kecklund, G. (2002). Sleep disturbances, work stress and work hours: A cross-sectional study. *Journal of Psychosomatic Research, 53,* 741–748. http://dx.doi.org/10.1016/S0022-3999(02)00333-1

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders* (3rd ed.). Darien, IL: American Academy of Sleep Medicine.

Bablas, V., Yap, K., Cunnington, D., Swieca, J., & Greenwood, K. M. (2016). Mindfulness-based stress reduction for restless legs syndrome: A proof of concept trial. *Mindfulness, 7,* 396–408. http://dx.doi.org/10.1007/s12671-015-0457-9

Barber, L. K., Taylor, S. G., Burton, J. P., & Bailey, S. F. (2017). A self-regulatory perspective of work-to-home undermining spillover/crossover: Examining the roles of sleep and exercise. *Journal of Applied Psychology, 102,* 753–763. http://dx.doi.org/10.1037/apl0000196

Barling, J., Barnes, C. M., Carleton, E. L., & Wagner, D. T. (Eds.). (2016). *Work and sleep: Research insights for the workplace*. New York, NY: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780190217662.001.0001

Barling, J., Rogers, A. G., & Kelloway, E. K. (2001). Behind closed doors: In-home workers' experience of sexual harassment and workplace violence. *Journal of Occupational Health Psychology, 6,* 255–269. http://dx.doi.org/10.1037/1076-8998.6.3.255

Barnes, C. M., Ghumman, S., & Scott, B. A. (2013). Sleep and organizational citizenship behavior: The mediating role of job satisfaction. *Journal of Occupational Health Psychology, 18,* 16–26. http://dx.doi.org/10.1037/a0030349

Barnes, C. M., Miller, J. A., & Bostock, S. (2017). Helping employees sleep well: Effects of cognitive behavioral therapy for insomnia on work outcomes. *Journal of Applied Psychology, 102,* 104–113. http://dx.doi.org/10.1037/apl0000154

Barnes, C. M., Schaubroeck, J., Huth, M., & Ghumman, S. (2011). Lack of sleep and unethical conduct. *Organizational Behavior and Human Decision Processes, 115,* 169–180. http://dx.doi.org/10.1016/j.obhdp.2011.01.009

Barnes, C. M., & Wagner, D. T. (2009). Changing to daylight saving time cuts into sleep and increases workplace injuries. *Journal of Applied Psychology, 94,* 1305–1317. http://dx.doi.org/10.1037/a0015320

Baumeister, R. F., & Vohs, K. D. (2011). Self-regulation and the executive function of the self. In M. R. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 181–197). New York, NY: Guilford Press.

Beaumont, M., Batejat, D., Pierard, C., Coste, O., Doireau, P., Van Beers, P., . . . Lagarde, D. (2001). Slow release caffeine and prolonged (64-h) continuous wakefulness: Effects on vigilance and cognitive performance. *Journal of Sleep Research, 10,* 265–276. http://dx.doi.org/10.1046/j.1365-2869.2001.00266.x

Becker, T. E., Atinc, G., Breaugh, J. A., Carlson, K. D., Edwards, J. R., & Spector, P. E. (2016). Statistical control in correlational studies: 10 essential recommendations for organizational researchers. *Journal of*

Organizational Behavior, 37, 157–167. http://dx.doi.org/10.1002/job.2053

Berry, C. M., Lelchook, A. M., & Clark, M. A. (2012). A meta-analysis of the interrelationships between employee lateness, absenteeism, and turnover: Implications for models of withdrawal behavior. *Journal of Organizational Behavior, 33,* 678–699. http://dx.doi.org/10.1002/job.778

Bixler, E. O., Vgontzas, A. N., Ten Have, T., Tyson, K., & Kales, A. (1998). Effects of age on sleep apnea in men: I. Prevalence and severity. *American Journal of Respiratory and Critical Care Medicine, 157,* 144–148. http://dx.doi.org/10.1164/ajrccm.157.1.9706079

Bucks, R. S., Olaithe, M., & Eastwood, P. (2013). Neurocognitive function in obstructive sleep apnoea: A meta-review. *Respirology, 18,* 61–70. http://dx.doi.org/10.1111/j.1440-1843.2012.02255.x

Canessa, N., Castronovo, V., Cappa, S. F., Aloia, M. S., Marelli, S., Falini, A., . . . Ferini-Strambi, L. (2011). Obstructive sleep apnea: Brain structural changes and neurocognitive function before and after treatment. *American Journal of Respiratory and Critical Care Medicine, 183,* 1419–1426. http://dx.doi.org/10.1164/rccm.201005-0693OC

Carleton, E. L., & Barling, J. (2016). Sleep and work withdrawal. In J. Barling, C. M. Barnes, E. L. Carleton, & D. T. Wagner (Eds.), *Work and sleep: Research insights for the workplace* (pp. 193–212). New York, NY: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780190217662.003.0010

Cohen, S., O'Leary, K. D., & Foran, H. (2010). A randomized clinical trial of a brief, problem-focused couple therapy for depression. *Behavior Therapy, 41,* 433–446. http://dx.doi.org/10.1016/j.beth.2009.11.004

Crain, T. L., Hammer, L. B., Bodner, T., Kossek, E. E., Moen, P., Lilienthal, R., & Buxton, O. M. (2014). Work-family conflict, family-supportive supervisor behaviors (FSSB), and sleep outcomes. *Journal of Occupational Health Psychology, 19,* 155–167. http://dx.doi.org/10.1037/a0036010

Davies, C. R., & Harrington, J. J. (2016). Impact of obstructive sleep apnea on neurocognitive function and impact of continuous positive air pressure. *Sleep Medicine Clinics, 11,* 287–298. http://dx.doi.org/10.1016/j.jsmc.2016.04.006

De Valck, E., & Cluydts, R. (2003). Sleepiness as a state-trait phenomenon, comprising both a sleep drive and a wake drive. *Medical Hypotheses, 60,* 509–512. http://dx.doi.org/10.1016/S0306-9877(02)00444-9

Domenech, P., & Koechlin, E. (2015). Executive control and decision-making in the prefrontal cortex. *Current Opinion in Behavioral Sciences, 1,* 101–106. http://dx.doi.org/10.1016/j.cobeha.2014.10.007

Driver, H. (2016). Sleep disorders at work. In J. Barling, C. M. Barnes, E. L. Carleton, & D. T. Wagner (Eds.), *Work and sleep: Research insights for the workplace* (pp. 31–52). New York, NY: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780190217662.003.0003

Epstein, L. J., Kristo, D., Strollo, P. J., Jr., Friedman, N., Malhotra, A., Patil, S. P., . . . Weinstein, M. D. (2009). Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *Journal of Clinical Sleep Medicine, 5,* 263–276. http://dx.doi.org/10.5664/jcsm.27497

Espie, C. (2006). *Overcoming Insomnia and Sleep Problems: A self-help guide using cognitive behavioural techniques*. London, United Kingdom: Constable & Robinson.

Ford, E. S., Cunningham, T. J., Giles, W. H., & Croft, J. B. (2015). Trends in insomnia and excessive daytime sleepiness among U.S. adults from 2002 to 2012. *Sleep Medicine, 16,* 372–378. http://dx.doi.org/10.1016/j.sleep.2014.12.008

Garbarino, S., Guglielmi, O., Sanna, A., Mancardi, G. L., & Magnavita, N. (2016). Risk of occupational accidents in workers with obstructive sleep apnea: Systematic review and meta-analysis. *Sleep, 39,* 1211–1218. http://dx.doi.org/10.5665/sleep.5834

Ghasemi, A., & Zahediasl, S. (2012). Normality tests for statistical analysis: A guide for non-statisticians. *International Journal of Endocrinology and Metabolism, 10,* 486–489.

Gobin, C. M., Banks, J. B., Fins, A. I., & Tartar, J. L. (2015). Poor sleep quality is associated with a negative cognitive bias and decreased sustained attention. *Journal of Sleep Research, 24,* 535–542. http://dx.doi.org/10.1111/jsr.12302

Guglielmi, O., Jurado-Gámez, B., Gude, F., & Buela-Casal, G. (2014). Job stress, burnout, and job satisfaction in sleep apnea patients. *Sleep Medicine, 15,* 1025–1030. http://dx.doi.org/10.1016/j.sleep.2014.05.015

Gunia, B. C., Barnes, C. M., & Sah, S. (2014). The morality of larks and owls: Unethical behavior depends on chronotype as well as time of day. *Psychological Science, 25,* 2272–2274. http://dx.doi.org/10.1177/0956797614541989

Harrison, Y., & Horne, J. A. (2000). The impact of sleep deprivation on decision making: A review. *Journal of Experimental Psychology: Applied, 6,* 236–249. http://dx.doi.org/10.1037/1076-898X.6.3.236

Hayes, A. F. (2018). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach* (2nd ed.). New York, NY: Guilford Press.

Heinzer, R. C., Pellaton, C., Rey, V., Rossetti, A. O., Lecciso, G., Haba-Rubio, J., . . . Lavigne, G. (2012). Positional therapy for obstructive sleep apnea: An objective measurement of patients' usage and efficacy at home. *Sleep Medicine, 13,* 425–428. http://dx.doi.org/10.1016/j.sleep.2011.11.004

Hepburn, C. G., & Barling, J. (1996). Eldercare responsibilities, interrole conflict, and employee absence: A daily study. *Journal of Occupational Health Psychology, 1,* 311–318. http://dx.doi.org/10.1037/1076-8998.1.3.311

Hoeksema-van Orden, C. Y., Gaillard, A. W., & Buunk, B. P. (1998). Social loafing under fatigue. *Journal of Personality and Social Psychology, 75,* 1179–1190. http://dx.doi.org/10.1037/0022-3514.75.5.1179

Johns, G. (2001). The psychology of lateness, absenteeism, and turnover. In N. Anderson, D. S. Ones, H. K. Sinangil, & C. Viswesvaran (Eds.), *Handbook of industrial, work, and organizational psychology: Organizational psychology* (Vol. 2, pp. 232–252). Thousand Oaks, CA: Sage. http://dx.doi.org/10.4135/9781848608368.n13

Johns, G., & Miraglia, M. (2015). The reliability, validity, and accuracy of self-reported absenteeism from work: A meta-analysis. *Journal of Occupational Health Psychology, 20,* 1–14. http://dx.doi.org/10.1037/a0037754

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep, 14,* 540–545. http://dx.doi.org/10.1093/sleep/14.6.540

Joosten, S. A., O'Driscoll, D. M., Berger, P. J., & Hamilton, G. S. (2014). Supine position related obstructive sleep apnea in adults: Pathogenesis and treatment. *Sleep Medicine Reviews, 18,* 7–17. http://dx.doi.org/10.1016/j.smrv.2013.01.005

Jurado-Gámez, B., Guglielmi, O., Gude, F., & Buela-Casal, G. (2015). Workplace accidents, absenteeism and productivity in patients with sleep apnea. *Archivos de Bronconeumologia, 51,* 213–218. http://dx.doi.org/10.1016/j.arbr.2014.12.002

Kammeyer-Mueller, J. D., & Wanberg, C. R. (2003). Unwrapping the organizational entry process: Disentangling multiple antecedents and their pathways to adjustment. *Journal of Applied Psychology, 88,* 779–794. http://dx.doi.org/10.1037/0021-9010.88.5.779

Kessler, R. C., Berglund, P. A., Coulouvrat, C., Hajak, G., Roth, T., Shahly, V., . . . Walsh, J. K. (2011). Insomnia and the performance of U.S. workers: Results from the America insomnia survey. *Sleep, 34,* 1161–1171. http://dx.doi.org/10.5665/SLEEP.1230

LeBlanc, M. M., Barling, J., & Turner, N. (2014). Intimate partner aggression and women's work outcomes. *Journal of Occupational Health Psychology, 19,* 399–412. http://dx.doi.org/10.1037/a0037184

Leger, D., Bayon, V., Laaban, J. P., & Philip, P. (2012). Impact of sleep apnea on economics. *Sleep Medicine Reviews, 16,* 455–462. http://dx.doi.org/10.1016/j.smrv.2011.10.001

Lim, J., & Dinges, D. F. (2010). A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychological Bulletin, 136,* 375–389. http://dx.doi.org/10.1037/a0018883

Loredo, J. S., Ancoli-Israel, S., Kim, E.-J., Lim, W. J., & Dimsdale, J. E. (2006). Effect of continuous positive airway pressure versus supplemental oxygen on sleep quality in obstructive sleep apnea: A placebo-CPAP-controlled study. *Sleep, 29,* 564–571. http://dx.doi.org/10.1093/sleep/29.4.564

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the produce and resampling methods. *Multivariate Behavioral Research, 39,* 99–128. http://dx.doi.org/10.1207/s15327906mbr3901_4

Melamed, S., Shirom, A., Toker, S., & Shapira, I. (2006). Burnout and risk of type 2 diabetes: A prospective study of apparently healthy employed persons. *Psychosomatic Medicine, 68,* 863–869. http://dx.doi.org/10.1097/01.psy.0000242860.24009.f0

Miller, P. B., & Weijer, C. (2006). Trust based obligations of the state and physician-researchers to patient-subjects. *Journal of Medical Ethics, 32,* 542–547. http://dx.doi.org/10.1136/jme.2005.014670

Nilsson, J. P., Söderström, M., Karlsson, A. U., Lekander, M., Akerstedt, T., Lindroth, N. E., & Axelsson, J. (2005). Less effective executive functioning after one night's sleep deprivation. *Journal of Sleep Research, 14,* 1–6. http://dx.doi.org/10.1111/j.1365-2869.2005.00442.x

Nordin, M., Åkerstedt, T., & Nordin, S. (2013). Psychometric evaluation and normative data for the Karolinska Sleep Questionnaire. *Sleep and Biological Rhythms, 11,* 216–226. http://dx.doi.org/10.1111/sbr.12024

Olaithe, M., & Bucks, R. S. (2013). Executive dysfunction in OSA before and after treatment: A meta-analysis. *Sleep, 36,* 1297–1305. http://dx.doi.org/10.5665/sleep.2950

Olson, R., Crain, T. L., Bodner, T. E., King, R., Hammer, L. B., Klein, L. C., . . . Buxton, O. M. (2015). A workplace intervention improves sleep: Results from the randomized controlled Work, Family, and Health Study. *Sleep Health, 1,* 55–65. http://dx.doi.org/10.1016/j.sleh.2014.11.003

Peppard, P. E., Young, T., Barnet, J. H., Palta, M., Hagen, E. W., & Hla, K. M. (2013). Increased prevalence of sleep-disordered breathing in adults. *American Journal of Epidemiology, 177,* 1006–1014. http://dx.doi.org/10.1093/aje/kws342

Pilcher, J. J., & Huffcutt, A. I. (1996). Effects of sleep deprivation on performance: A meta-analysis. *Sleep, 19,* 318–326. http://dx.doi.org/10.1093/sleep/19.4.318

Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods, 16,* 93–115. http://dx.doi.org/10.1037/a0022658

Romero, E., Krakow, B., Haynes, P., & Ulibarri, V. (2010). Nocturia and snoring: Predictive symptoms for obstructive sleep apnea. *Sleep and Breathing, 14,* 337–343. http://dx.doi.org/10.1007/s11325-009-0310-2

Roth, T., Coulouvrat, C., Hajak, G., Lakoma, M. D., Sampson, N. A., Shahly, V., . . . Kessler, R. C. (2011). Prevalence and perceived health associated with insomnia based on *DSM–IV–TR*; international statistical classification of diseases and related health problems, tenth revision; and research diagnostic criteria/international classification of sleep disorders, criteria: Results from the America insomnia survey. *Biological Psychiatry, 69,* 592–600. http://dx.doi.org/10.1016/j.biopsych.2010.10.023

Salepci, B., Caglayan, B., Kiral, N., Parmaksiz, E. T., Comert, S. S., Sarac, G., . . . Gungor, G. A. (2013). CPAP adherence of patients with obstructive sleep apnea. *Respiratory Care, 58,* 1467–1473. http://dx.doi.org/10.4187/respcare.02139

Shadish, W. R., Cook, T. D., & Campbell, D. T. (2002). *Experimental and quasi-experimental designs for generalized causal experiments*. New York, NY: Houghton Mifflin.

Silva, G. E., Goodwin, J. L., Vana, K. D., & Quan, S. F. (2016). Obstructive sleep apnea and quality of life: Comparison of the SAQLI, FOSQ, and SF-36 questionnaires. *Southwest Journal of Pulmonary and Critical Care, 13,* 137–149. http://dx.doi.org/10.13175/swjpcc082-16

Statistics Canada. (2018). *Health fact sheets: Sleep Apnea in Canada, 2016 and 2017*. Ottawa, ON: Government of Canada.

Swanson, L. M., Arnedt, J. T., Rosekind, M. R., Belenky, G., Balkin, T. J., & Drake, C. (2011). Sleep disorders and work performance: Findings from the 2008 National Sleep Foundation Sleep in America poll. *Journal of Sleep Research, 20,* 487–494. http://dx.doi.org/10.1111/j.1365-2869.2010.00890.x

Tachikawa, R., Minami, T., Matsumoto, T., Murase, K., Tanizawa, K., Inouchi, M., . . . Chin, K. (2017). Changes in habitual sleep duration after continuous positive airway pressure for obstructive sleep apnea. *Annals of the American Thoracic Society, 14,* 986–993. http://dx.doi.org/10.1513/AnnalsATS.201610-816OC

Vitiello, M. V. (2009). *Aging and sleep*. Washington, DC: National Sleep Foundation. Retrieved from https://sleepfoundation.org/sleep-topics/aging-and-sleep/page/0/3

Welsh, D. T., Ellis, A. P., Christian, M. S., & Mai, K. M. (2014). Building a self-regulatory model of sleep deprivation and deception: The role of caffeine and social influence. *Journal of Applied Psychology, 99,* 1268–1277. http://dx.doi.org/10.1037/a0036202

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—A prefrontal amygdala disconnect. *Current Biology, 17,* R877–R878. http://dx.doi.org/10.1016/j.cub.2007.08.007

Received June 9, 2019
Revision received December 13, 2019
Accepted March 18, 2020 ■