Obstructive sleep apnea (OSA) is a common sleep disorder characterized by recurrent complete or partial airway collapse, resulting in frequent episodes of apnea and hypopnea. Untreated OSA is a serious condition that increases daytime sleepiness and cognitive dysfunction, increases the risk of fatigue-related crashes,3,4 and impairs quality of life.5 Untreated OSA is also strongly associated with major adverse health outcomes, including cardiovascular disease,6-8 hypertension,9,10 insulin resistance,11,12 stroke,13,14 and depression.15-17

In an occupational setting, untreated OSA also represents a significant health and safety risk for both employees and the public. OSA is most prevalent in middle-aged, overweight men,18 who make up the majority of the workforce in many occupations.19-23 Other factors that affect sleepiness, such as long work hours, chronic sleep deficiency and shift work, are also associated with some occupations, which further increases the risk of sleepiness-related errors, accidents, and injuries.24 Given the potentially high prevalence of OSA in some occupational groups,19-23 the development of large-scale occupational sleep apnea diagnostic programs could help target high-risk individuals and provide timely treatment for those unwilling or unable to seek assessment through traditional channels. Screening based solely on use of questionnaires is quick and inexpensive, but can suffer from inaccurate or biased reporting, particularly if there is a risk of censure for reporting sleepiness, for example in truck drivers.25 Simple-to-use portable devices that patients can use unattended in their own homes represent a possible method to bridge the large unmet gap for identifying employees at high risk for OSA while providing a more accurate assessment than subjective questionnaires.
A number of lower cost and more practical assessment methods have been developed to enable portable or ambulatory monitoring. These methods include respirometry, peripheral arterial tonometry, blood oximetry, blood pressure recordings, or a combination of these methods. While many devices have proven to be useful for detecting OSA in high-risk populations, such as those already attending a sleep clinic, there are few studies testing the ability of such devices to confirm absence of OSA risk in a population known not to have the disorder.

The aim of our study was to assess the ability of a portable device to quantify OSA risk under laboratory and home-based conditions in participants with and without OSA, confirmed using PSG. We chose to study the ApneaLink device (ResMed Corporation, Poway, CA), which is a single-channel device that derives and assesses airflow from an intra-nasal pressure signal and is considered by the AASM as an ambulatory Type 4 sleep monitor. ApneaLink Plus is a more recent version of the device that uses the same method to assess airflow, in addition to pulse oximetry and respiratory effort, but it was not available at the time of the study. There is a growing body of evidence assessing the utility of single-channel nasal pressure transducer devices to identify OSA in high-risk patient populations, such as those with suspected sleep apnea hypopnea syndrome or type 2 diabetes. There are no validation studies, however, using ApneaLink or other Type 4 devices that have included a control group at low risk for OSA. Knowledge of device performance in healthy participants is vital in developing large-scale occupational health diagnostic programs, in which participants have not been referred based on sleepiness or other symptoms.

METHODS

Participants

We chose to study police officers for this study as part of a larger occupational screening program for sleep disorders supported by the Centers for Disease Control. As part of the study, 605 state police officers completed a comprehensive sleep disorders screening questionnaire, including the Berlin Questionnaire, a validated tool for assessment of OSA risk. A score ≥ 2 (range 0–4) on 2 of the 3 domains (frequency of snoring behavior, wake time sleepiness/fatigue, current obesity or current hypertension or history of it) were considered positive for a high risk of OSA. One hundred thirty-one officers were invited to participate in the ApneaLink substudy. The selected sample included both symptomatic and asymptomatic individuals according to the Berlin questionnaire in approximately equal proportions. Sixty-three of these participants consented to the study; these participants had a slightly higher BMI (mean ± SD, 30.6 ± 4.3 kg/m²) than those who did not volunteer (28.2 ± 3.6 kg/m²; p < 0.05, Student t-test) and a lower proportion of individuals not at risk of sleep apnea according to the Berlin Questionnaire (41% versus 65%; p < 0.05, Fisher exact test). Eight participants withdrew prior to data collection; therefore, 55 officers underwent full PSG in the sleep laboratory with simultaneous recordings with ApneaLink. The study protocol was approved by the Partners Healthcare Human Research Committee. Each participant was asked to sign a written informed consent form before the initial recording and was paid $400 for participation.

Diagnostic Polysomnography

Each participant underwent overnight polysomnography (PSG) at Sleep HealthCenters, in Brighton MA. The sleep study included acquisition of standard PSG signals, obtained with the Alice-5 polysomnography System (Respironics, Inc., Pittsburgh, PA). For PSG, the Alice-5 System electrodes were attached to the face and scalp to measure sleep stages (electroencephalography, electrooculography, and chin electromyography) and to the chest to monitor the electrocardiogram. Chest and abdomen movement was detected using piezoelectric crystal effort straps, which are part of the sensor kit included in the Alice-5 polysomnography system; a transducer was clipped onto the finger to measure oxygen levels (SpO₂); and electrodes were pasted onto the lower legs to measure leg movements.

All PSG recordings were scored by Sleep HealthCenters’ registered polysomnographic technologists and then independently interpreted by an experienced analyst (SAS), who was blind to study condition, for confirmation of AHI and clinical relevance of the results. PSG sleep was staged according to standard criteria in 30-sec epochs from a high-resolution computer display using Alice software versions 3 and 4. Arousals were detected from ≥ 3-sec changes in electroencephalography and electromyography, using standardized criteria. Respiratory events (apneas and hypopneas) were scored according to the recommendations of the Task Force of the American Academy of Sleep Medicine (the “Chicago Criteria”). Thus, apnea was scored when cessation of airflow ≥ 10 sec was observed. Apneas were further classified as obstructive or central depending on the presence or absence of chest wall breathing movements. Hypopneas were identified based on a discernible decrease in breathing ≥ 10 sec (observed in the respiratory strain gauge, nasal pressure, or thermistor recordings), followed by either arterial oxyhemoglobin desaturation ≥ 3% or an arousal. Hypopneas were not categorized as obstructive or central events. These events were quantified as the AHI (number of respiratory disturbances per hour of sleep).

ApneaLink

ApneaLink is a single-channel portable device which measures intra-nasal pressure via a nasal cannula connected to a pressure transducer. Pressure is proportional to airflow if resistance in the system remains constant, but when used in an open system via nasal cannula, there is typically a nonlinear pressure-flow relationship, which can be effectively linearized by using the square root of the pressure signal. The device is battery operated and is held in place by a belt worn around the user’s chest. The pressure signal is recorded with a sampling rate of 100 Hz and is pre-processed by linearizing the signal to estimate airflow, filtering the noise, and zeroing the baseline. The ApneaLink software automatically analyzed the data generated by the estimated airflow signal and provided a report. The device is capable of monitoring breathing patterns and measures apneas or hypopneas as well as flow limitation, snoring sounds, and inspiratory flow. For the purpose of this study, only AHI was analyzed. For laboratory recordings, ApneaLink was connected to one end of a Y-shaped nasal cannula. The other end of the Y-shaped cannula was directly connected to a pressure transducer in the Alice system to enable nasal pressure to be recorded simultaneously by both devices.
The ApneaLink default settings for apneas and hypopneas were used: an apnea is defined as a decrease in airflow by 80% of baseline for ≥ 10 seconds. The ApneaLink default maximum apnea duration was set at 80 seconds. A hypopnea was defined as a decrease in airflow by 50% to 80% of baseline for ≥ 10 seconds. The ApneaLink default maximum hypopnea duration was set at 100 seconds. The ApneaLink scoring version 5.26 was used for the analysis. The AHI derived from the PSG was based on total sleep time, in contrast the AHI from ApneaLink, which was based on total evaluation time.

Participants were asked to wear the ApneaLink at home for 2 full nights within a 7-day period. Participants were sent the device with comprehensive instructions on how to use the equipment, including fitment and recording procedures. A contact telephone number was provided in case of additional questions or problems. Participants were required to attend the clinic the following week with the device for download or return the device using pre-paid packages provided within 7 days.

**Data Analysis and Statistics**

Data were included in the analysis if an ApneaLink total evaluation time ≥ 2 h was obtained. Participants who did not have ≥ 2 h of usable data per night were asked to repeat the ApneaLink evaluation for that night.

ApneaLink was validated against standard PSG recordings. This validation included analysis of sensitivity and specificity as well as positive (PPV) and negative predictive values (NPV) for a range of AHI thresholds. Sensitivity and specificity comparisons were plotted graphically using receiver operator characteristics (ROC) curves. In an ROC curve, the true positive rate (sensitivity) is plotted against the reciprocal of the false positive rate (100-specificity) for different cutoff points. Each point on the ROC plot represents a sensitivity/specificity pair corresponding to a particular decision threshold. A test with perfect discrimination (no overlap in the 2 distributions) has an ROC plot that passes through the upper left corner (100% sensitivity, 100% specificity). Therefore the closer the ROC plot is to the upper left corner and the higher the resulting area under the curve (AUC), the higher the overall reliability of the test. ROC curves were computed to establish optimal performance at AHI thresholds of PSG AHI ≥ 5/h, ≥ 10/h, and ≥ 15/h. Correlation analyses were performed using Pearson correlation coefficients. Finally, Bland-Altman plots were also constructed as a graphic representation of the observed differences between paired measurements. The differences between the 2 techniques (PSG and ApneaLink) were plotted against the average of the 2 techniques. The mean difference provides an estimate of whether the two techniques, on average, return similar results. Descriptive statistics are presented for the demographic data, whereas continuous data are presented by mean values ± standard deviation, and categorical data by a numeric value and a percentage.

**RESULTS**

Data recordings were obtained from 55 officers (Figure 1). Forty-eight participants underwent simultaneous PSG and ApneaLink recordings in the laboratory. Of these, 4 participants were excluded from further analysis for having recording times < 2 hours. Forty participants also wore the ApneaLink device in their own homes for 1 night; 37 wore the device for a second night. During these 2 nights, 12 participants had a night’s recording < 2 h in duration; these participants were asked to repeat the recording on a subsequent night. After repeat nights, 38 participants had suitable data for analysis of the ApneaLink device in the home setting for 1 night, and 36 participants had suitable data for 2 nights. Participants were excluded from the analysis if they did not complete a PSG laboratory night (n = 7) or if they did not complete a study night in their own home (n = 3).

Forty-three participants had successful simultaneous PSG and ApneaLink recordings in the laboratory. Of these, 25/43 (58.1%) did not have OSA (PSG AHI < 5/h); 41% had an AHI
≥ 5; 23% had an AHI ≥ 10; and 16% had an AHI ≥ 15, based on the AHI obtained from the PSG study (Table 1).

ApneaLink (Laboratory) AHI vs PSG AHI

The mean overall sleep time for PSG studies was 348 min, and the mean overall evaluation time for the ApneaLink studies was 316 minutes. Table 2 shows the sensitivity, specificity, and positive and negative predictive values for the comparison of the Apnealink-derived AHI with PSG-derived AHI using the same absolute AHI values (e.g., AHI ≥ 5 events/h when measured with PSG compared to a cutoff ≥ 5 events/h when measured with ApneaLink device).

For an AHI ≥ 15, the sensitivity and specificity of the ApneaLink were 100% and 92%, respectively, with a PPV and NPV of 70% and 100%, respectively. The area under the ROC curve was also high at 0.994 (Figure 2A). These data suggest a very high level of performance of the ApneaLink compared to PSG under supervised laboratory conditions to identify patients with frank OSA. ApneaLink was not as robust a method to identify mild-moderate OSA (AHI ≥ 5) in the laboratory, with good sensitivity (89%) and NPV (88%), but low specificity (56%) and PPV (59%).

There was a significant positive correlation between the 2 recording methods when collected simultaneously ($r = 0.93$, Table 1—Demographic characteristics of study participants

<table>
<thead>
<tr>
<th>Demographic Characteristic</th>
<th>All</th>
<th>&lt; 10</th>
<th>≥ 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of participants</td>
<td>43</td>
<td>33</td>
<td>10</td>
</tr>
<tr>
<td>Age, yrs</td>
<td>45.4 ± 10.8</td>
<td>44.0 ± 11.1</td>
<td>50.2 ± 8.9</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30.4 ± 5.3</td>
<td>29.5 ± 5.3</td>
<td>33.4 ± 4.5</td>
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<tr>
<td>Sex</td>
<td>Male: 38; Female: 5</td>
<td>Male: 28; Female: 5</td>
<td>Male: 10; Female: 0</td>
</tr>
<tr>
<td>PSG Sleep Efficiency (%)</td>
<td>84.3 ± 9.5</td>
<td>84.5 ± 9.6</td>
<td>83.7 ± 9.5</td>
</tr>
<tr>
<td>ESS (0-21)</td>
<td>8.8 ± 3.9</td>
<td>9.0 ± 4.2</td>
<td>8.7 ± 2.8</td>
</tr>
<tr>
<td>PSG AH1</td>
<td>9.4 ± 17.1</td>
<td>2.9 ± 2.6</td>
<td>30.8 ± 26.1</td>
</tr>
<tr>
<td>PSG Respiratory Disturbance Index</td>
<td>17.1 ± 19.5</td>
<td>9.0 ± 6.1</td>
<td>43.9 ± 24.6</td>
</tr>
<tr>
<td>Baseline SpO₂</td>
<td>96.0 ± 1.2</td>
<td>96.0 ± 1.1</td>
<td>95.8 ± 1.5</td>
</tr>
</tbody>
</table>

AHI refers to apnea hypopnea index derived from polysomnography, PPV, positive predictive value, NPV, negative predictive value, AUC, area under the curve.

Data are presented as mean ± standard deviation. Apnea-hypopnea (AHI) values are based on polysomnographic (PSG) data.

Table 2—Sensitivity and specificity of the ApneaLink AHI compared to PSG AHI during simultaneous laboratory testing in participants with more than 2 hours of ApneaLink data recording (n = 44)

<table>
<thead>
<tr>
<th>PSG AHI</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>AUC</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 5</td>
<td>88.9</td>
<td>56.0</td>
<td>59.3</td>
<td>87.5</td>
<td>0.883</td>
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<tr>
<td>≥ 10</td>
<td>80.0</td>
<td>75.8</td>
<td>50.0</td>
<td>92.6</td>
<td>0.918</td>
</tr>
<tr>
<td>≥ 15</td>
<td>100.0</td>
<td>91.7</td>
<td>70.0</td>
<td>100.0</td>
<td>0.994</td>
</tr>
</tbody>
</table>

AHI refers to apnea hypopnea index derived from polysomnography, PPV, positive predictive value, NPV, negative predictive value, AUC, area under the curve.

≥ 5; 23% had an AHI ≥ 10; and 16% had an AHI ≥ 15, based on the AHI obtained from the PSG study (Table 1).
Evaluation of ApneaLink to Detect OSA Risk

\( r^2 = 0.86 \ p < 0.01 \) (Figure 3A). A Bland-Altman plot (Figure 4) revealed most of the AHI measurements fell within a range of 2 standard deviations from the mean value, demonstrating a reasonably tight distribution of the differences. The discrepancy between the ApneaLink and PSG widened for AHI values > 15, indicating that ApneaLink overestimated the AHI score.

(A) Bland-Altman plot of ApneaLink apnea-hypopnea index (AHI) and polysomnographic (PSG) AHI data during the laboratory study. The dashed lines indicate the upper and lower confidence limits, and the solid line indicates the mean of the difference between the two methods of detection. (B) Bland-Altman plot of ApneaLink AHI and PSG AHI data collected in the home for 1 night and (C) Bland-Altman plot of ApneaLink AHI and PSG data collected in the home for the average of 2 nights.

(A) Correlation of apnea-hypopnea index (AHI) from assessment by simultaneous polysomnography (PSG) and ApneaLink \( (r^2 = 0.86) \). (B) Linear regression of AHI from PSG and ApneaLink recorded in the home for 1 night \( (r^2 = 0.69) \). (C) Linear regression of AHI from PSG and ApneaLink recorded in the home for the average of 2 nights \( (r^2 = 0.70) \).
Table 3—Sensitivity and specificity: PSG AHI vs. ApneaLink AHI at home for 1 night (n = 38) or averaged over 2 nights (n = 36)

<table>
<thead>
<tr>
<th>AHI</th>
<th>Sensitivity 1 night</th>
<th>Sensitivity 2 nights</th>
<th>Specificity 1 night</th>
<th>Specificity 2 nights</th>
<th>PPV 1 night</th>
<th>PPV 2 nights</th>
<th>NPV 1 night</th>
<th>NPV 2 nights</th>
<th>AUC 1 night</th>
<th>AUC 2 nights</th>
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<tbody>
<tr>
<td>≥ 5</td>
<td>81.3</td>
<td>87.5</td>
<td>77.3</td>
<td>85.0</td>
<td>72.2</td>
<td>82.4</td>
<td>85.0</td>
<td>89.5</td>
<td>0.921</td>
<td>0.944</td>
</tr>
<tr>
<td>≥ 10</td>
<td>77.8</td>
<td>77.8</td>
<td>82.8</td>
<td>88.9</td>
<td>58.3</td>
<td>70.0</td>
<td>92.3</td>
<td>92.3</td>
<td>0.889</td>
<td>0.903</td>
</tr>
<tr>
<td>≥ 15</td>
<td>66.7</td>
<td>66.7</td>
<td>90.6</td>
<td>93.3</td>
<td>57.1</td>
<td>66.7</td>
<td>93.5</td>
<td>93.3</td>
<td>0.953</td>
<td>0.958</td>
</tr>
</tbody>
</table>

AHI refers to apnea hypopnea index, PPV, positive predictive value, NPV, negative predictive value, AUC, area under the curve.

ApneaLink (Home) AHI vs PSG AHI

The results comparing the AHI from the ApneaLink device obtained from the PSG compared to the AHI obtained from ApneaLink in the home are shown in Table 3 for both 1 night (n = 38) and the average of 2 nights (n = 36). Use of the ApneaLink device at home showed generally similar performance characteristics as when measured in the laboratory simultaneous with PSG, except for lower sensitivity. For an AHI ≥ 15 when ApneaLink results were averaged over 2 nights at home, the sensitivity and specificity of ApneaLink were 67% and 93%, respectively, with a PPV and NPV of 67% and 93%, respectively. The area under the ROC curve was also high at 0.958 (Figure 2B). For an AHI ≥ 5, ApneaLink performed even better, with a sensitivity and specificity of 88% and 85%, respectively, and PPV and NPV of 82% and 90%, respectively, with an ROC area of 0.944 (Figure 2C).

Given that the average of 2 nights increased specificity and PPV, we examined the night-to-night variability in individual AHI values between 1 night and 2 nights. A correlation between these values showed a strong relationship between AHI values collected on separate nights in the same individual (r² = 0.69, p < 0.001).

The correlations between ApneaLink AHI results collected from the participants’ homes are shown in Figure 3. Data from a single night (Figure 3B: r² = 0.69, p < 0.001) or from the average of 2 nights (Figure 3C: r² = 0.70, p < 0.002) show a significant positive relationship to AHI results from PSG. Bland-Altman plots again reveal tight associations between PSG and ApneaLink when collected in the home setting, either on 1 night (Figure 4B) or averaged over 2 nights (Figure 4C).

On the whole, compliance with the use of ApneaLink was reasonable. Over half of all the recording times (64.5%) were > 4 h in duration. On the first night of use at home, 88.6% of participants produced recordings which were > 2 h in duration. Five participants obtained recordings that were < 2 h and so required a repeat night. The second night produced similar results; 82.9% of participants produced ≥ 2 h of data. Over the 2 nights, there were 12 participants who were asked to perform a repeat night in their home.

DISCUSSION

The primary aim of the study was to evaluate the validity and utility of using the ApneaLink device for assessment of AHI as a marker of OSA presence and severity. The broader aim of the study was to understand the advantages and potential limitations of the ApneaLink device in relation to its use in large-scale occupational diagnostic programs for OSA. The results show that ApneaLink demonstrated clinical utility as a diagnostic device for OSA, including correct identification of a healthy “true negative” group of participants who were confirmed not to have OSA. In comparison with PSG data obtained simultaneously in the laboratory, ApneaLink showed a high level of sensitivity (> 75%) at all AHI levels, confirming the capacity of ApneaLink to identify patients with significant levels of OSA when present, consistent with previous studies of high-risk groups. ApneaLink was not as robust a method to identify mild-moderate OSA (AHI ≥ 5) in the laboratory, but home-based ApneaLink assessment of mild-moderate OSA performed well versus laboratory PSG.

The utility of the ApneaLink device is its use in a community setting. We asked participants to complete two home-based assessment nights with ApneaLink in order to (i) increase the likelihood of obtaining at least one night of home-based evaluation for use in comparison with PSG; and (ii) to evaluate whether averaging data over two nights increased the accuracy of the home-based evaluation and reduced the impact of any “first-night” effect in the home. Use of the ApneaLink device at home showed generally similar performance characteristics when measured in the laboratory simultaneous with PSG, except for lower sensitivity. Using the average AHI from two nights of home-based evaluation improved the performance of the ApneaLink relative to PSG for most evaluations, particularly for specificity and PPV. In developing future programs, it may be worthwhile to determine the cost-benefit relationship between the time, cost, and inconvenience of a second home-based evaluation versus the improvement in performance, particularly in those patients who do not exhibit frank OSA on the first assessment night. In patients with type 2 diabetes, home recordings shorter than 4 hours led to more frequent false negatives results at AHI levels ≥ 15. Our results, while overall consistent with those of Erman et al, found a higher compliance for use of the device, even when recordings were between two and four hours. Almost two-thirds of participants (64.5%) using ApneaLink in their homes, even on the first night, had good success at obtaining recordings of four hours or more. Moreover, regarding assessment feasibility, most participants who had adequate data on two nights also had good data on one night, suggesting that if an individual is trained and motivated to collect data, then sufficient information can be collected in a single night (notwithstanding the possible tradeoff in accuracy versus time with adding a second night). A subset of participants, however, were unable or unwilling to provide a home-based evaluation and, as part of a larger program, these participants may require additional support or a supervised home-based or laboratory assessment.

In the current study, the AASM “Chicago criteria” were used to score AHI. The choice of specific scoring criteria will cer-
ertainly affect results, and many scoring criteria exist (although all require such respiratory events to be ≥ 10 sec duration). For instance, Ruehland et al. have compared AHIs using the Chicago criteria with other commonly used AASM scoring criteria. Those authors found that AHIs scored with the Chicago criteria (≥ 50% reduction in breathing, or a clear reduction in breathing together with ≥ 3% decrease in arterial oxyhemoglobin saturation or an arousal from sleep) invariably were higher than AHIs using two other commonly used hypopnea definitions, i.e., ≥ 30% reduction in breathing and ≥ 4% desaturation; or ≥ 50% airflow reduction and ≥ 3% desaturation or arousal. Thus, it is likely that the sensitivity and specificity comparisons in the current study would be systematically altered in the current study if these different criteria were used, and this should be considered in coming to any clinical decision.

A limitation of our study was the relatively low number of people with more severe OSA (AHI ≥ 15, 6/38 participants), which most likely accounts for the relatively low sensitivity (67%) when comparing the home-based ApneaLink assessment with laboratory PSG. Our data are generally consistent, however, with data from previous studies showing that single-channel flow assessment using ApneaLink performs well at identifying people with frank OSA. The greater challenge is to identify those with mild-moderate OSA who may not have suspected disease based on traditional risk factors and/or do not have robust symptoms. Our study demonstrates that the ApneaLink performs well in identifying those with an AHI ≥ 5, with all performance parameters between 82% and 90%. As a first-step tool, this level of performance seems reasonable and would identify those who are at risk of OSA but who may not necessarily be strongly symptomatic, and would also triage those who do or do not need further evaluation based on an ApneaLink AHI of ≥ 5 or < 5, respectively.

In summary, this study has demonstrated the utility of a relatively simple, automated single-channel device in the identification of OSA in a heterogeneous occupational population. Development of large-scale occupational diagnosis programs for OSA using portable devices has enormous potential to identify previously undiagnosed patients and reduce the health and safety burden of this disease.

REFERENCES

27. Pittman SD, Ayas NT, MacDonald MM, Malhotra A, Fogel RB, White DP. Using a wrist-worn device based on peripheral arterial tonometry to diagnose obstructive sleep apnea: in-laboratory and ambulatory validation. Sleep 2004;27:923-33.


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**DISCLOSURE STATEMENT**

This study was supported by a research grant from the ResMed Foundation to Dr. Lockley, and grants to Dr. Czeisler from the National Institute of Justice (2004-FS-BX-001) and the Centers for Disease Control and Prevention (RO1 OH008496). ApneaLink equipment and associated consumables were donated by ResMed Corporation. Dr. Czeisler has received consulting fees from or served as a paid member of scientific advisory boards for Bombardier, Inc., Boston Celtics, Cephalon, Delta Airlines, Eli Lilly, Global Ground Support, Johnson & Johnson, Koninklijke Philips Electronics, N.V., Minnesota Timberwolves, Portland Trail Blazers, Respironics, Sleep Multimedia, Inc., Somnus Therapeutics, Inc., Yanda Pharmaceuticals, and Zeo Inc. He owns an equity interest in Lifetrac, Inc., Somnus Therapeutics, Inc., Yanda Pharmaceuticals, and Zeo Inc., and has received royalties from the Massachusetts Medical Society, New England Journal of Medicine, McGraw Hill, the New York Times Penguin Press, and Philips Respironics. Dr. Czeisler has participated in speaking engagements for the American Academy of Sleep Medicine, National Academy of Sciences, National Sleep Foundation, New England College of Occupational and Environmental Medicine (NECOEM), North East Sleep Society, Rockpointe, the University of Chicago, and the University of Colorado. Dr. Czeisler has also received research support from Cephalon, Merck, and the American Academy of Sleep Medicine. His research laboratory at the Brigham and Women’s Hospital has received research support from Cephalon and ResMed. Dr. Czeisler is the incumbent of an endowed professorship provided to Harvard University by Cephalon, Inc. and holds a number of process patents in the field of sleep/circadian rhythms (e.g., photic resetting of the human circadian pacemaker). Since 1985, Dr. Czeisler has also served as an expert witness on various legal cases related to sleep and/or circadian rhythms. Dr. Lockley has received research support from the ResMed Foundation, ResMed Inc, Respironics, Philips Lighting, Apollo Lighting, Alcon Inc., and Vanda Pharmaceuticals. He has consulted to Apollo Lighting and Wyle Integrated Science and Engineering (NASA) and holds current consulting contracts with Naturebright, Sound Oasis and Wyle Integrated Science and Engineering (NASA); He has received unrestricted equipment gifts from Philips Lighting and Bionetics Corporation; an unrestricted monetary gift to support research from Swinburne University of Technology, Australia; a fellowship gift from Optalert, Pty, Melbourne, Australia; advance author payment and royalties from Oxford University Press; honoraria from Servier Inc. for writing an article for Dialogues in Clinical Neuroscience; and honoraria from AMO Inc., for writing an educational monograph. Dr. Lockley has participated in speaking engagements for the 2nd International Symposium on the Design of Artificial Environments, American Academy of Sleep Medicine, American Society for Photobiology, Apollo Lighting, Bar Harbor Chamber of Commerce, Bassett Research Institute, Canadian Sleep Society, Committee of Interns and Residents, Coney Island Hospital, FASEB, Harvard University, Illinois Coalition for Responsible Outdoor Lighting, International Graduate School of Neuroscience, Japan National Institute of Occupational Safety and Health, Lightfair, National Research Council Canada, New York Academy of Sciences, North East Sleep Society, Ontario Association of Fire Chiefs, Philips Lighting, Thomas Jefferson University, University of Montreal, University of Tsukuba, University of Vermont College of Medicine, Utica College, Vanda Pharmaceuticals, Velux, Warwick Medical School, Woolcock Institute of Medical Research, and Wyle Integrated Science and Engineering (NASA). Dr. Lockley holds a process patent for the use of short-wavelength light for resetting the human circadian pacemaker and improving alertness and performance which is assigned to the Brigham and Women’s Hospital per Hospital policy. He has also received revenue from a patent on the use of short-wavelength light which is assigned to the University of Surrey. Dr. Lockley has also served as a paid expert witness on behalf of two public bodies on arbitration panels related to sleep, circadian rhythms and work hours. The other authors have indicated no financial conflicts of interest.

**SUBMISSION & CORRESPONDENCE INFORMATION**

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