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ORIGINAL ARTICLE

Effect of artificial dawn light on cardiovascular function, alertness, and balance in middle-aged and older adults

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Abstract

Study Objectives: When arising in the morning, many older people experience dizziness and difficulty maintaining proper balance, as the cardiovascular system is not able to compensate to the postural shift (standing) and maintain sufficient blood flow to the brain. Such changes in cardiovascular function are observed in young individuals exposed to a dawn simulation light. In this study, we examined whether exposure to a dawn simulation light could impact cardiovascular function and consequent changes in balance in middle-aged and older adults.

Methods: Twenty-three participants (67.3 ± 8.8 y), 12 of whom reported a history of dizziness in the morning, underwent two overnight stays in our laboratory. During both nights, they slept in complete darkness, except for the last 30 minutes of one of the nights during which a dawn simulation light was used. Continuous blood pressure (BP) and heart rate (HR) were monitored. Subjective and objective alertness, salivary cortisol, and mobile and standing balance were examined upon arising.

Results: Dawn simulation light decreased (33%) the amount of sleep before morning awakening, lowered BP (6.24 mmHg), and increased HR (0.93 bpm). Despite these changes in physiology, there was no significant impact of dawn simulation on subjective or objective alertness, measures of standing or ambulatory balance, morning cortisol awakening response, or cardiovascular function after awakening.

Conclusion: While the dawn simulation did cause an increase in wake and a change in cardiovascular function prior to morning arousal in older adults, we could find no evidence of a functional change in either cardiovascular function or balance upon standing.

Clinical Trial: Registered on Clinicaltrials.gov, #NCT02632318, https://clinicaltrials.gov/ct2/show/NCT02632318

Statement of Significance

Dawn simulation lights are consumer devices, but whether their utility extends beyond the subjective phenomenology of awakening is unknown. Previous literature indicates that these devices might be able to change cardiovascular function in young adults. We show that in middle-aged and older adults, dawn simulation light negatively impacts sleep and has only minor effects on cardiovascular function during sleep. Importantly, these changes are not associated with functional changes in standing or mobile balance after arising, nor are they associated with changes in concomitant waking cardiovascular function. As this study was conducted on a single overnight, future work should focus on repeated exposure to dawn simulation to determine if one could adapt to sleep disruption and benefit from the cardiovascular changes.

Key words: light; aging; balance; alertness; blood pressure; heart rate; heart rate variability; sleep inertia

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Introduction

One of the most physiologically demanding things that older people do every day is to get up in the morning. After spending a night lying flat, gravity distributes blood evenly across the body. Upon standing, metabolically costly changes occur in response to the postural change in order to maintain blood flow to the brain. Among these changes are a constriction in arterioles and veins and an increase in heart rate (HR) and stroke volume to maintain blood pressure (BP) in response to the redistribution of blood to lower extremities upon standing [1]. While the cardiovascular system in younger individuals is usually able to compensate for this shift, it is insufficient in many older adults and can lead to dizziness or even falls. BP medications or behavioral means (e.g. prolonged stepwise exit from bed) are often used as a countermeasure to mitigate the risk of dizziness and falls.

A novel alternative to pharmacologic and behavioral interventions for morning dizziness is the use of light. Light can have a direct impact on alertness [2], balance [3], and autonomic function [4]. We have recently found that exposure to a "dawn simulator" light (an artificial light source that gradually increases in brightness just prior to anticipated wake time) in young individuals shifted cardiovascular function from a "sleep" to "wake" state prior to individuals arising [5]. This dawn simulation light reduced rapid sleep-wake evoked increases in HR, as well as cardiac sympathovagal modulation in the young population. Moreover, these effects occurred without any changes in the sleep pattern preceding the wake-up time [5]. By the time that these healthy young individuals got out of bed, their cardiovascular system was already primed to compensate for the postural shift.

While it has been shown in young healthy individuals that the dawn simulator light can increase cardiovascular function such that levels normally observed during wake are present prior to wakefulness [5], we do not know whether this will have a similar effect on cardiovascular function in older adults. Additional effects of dawn simulation light have been observed, including reducing sleep inertia, improving subjective well-being, and increasing morning cortisol levels [5–8]. As such, we also wanted to test whether dawn simulation induced changes in cardiovascular function were associated with these functional changes in older adults, as well as middle-aged adults, upon getting out of bed as well as the clinically relevant change in standing and walking balance.

Our a priori hypotheses were that we would observe in response to the dawn simulation (1) a gradual increase in HR and BP before arising, (2) an improvement in standing and walking balance, (3) an improvement of cognitive performance, and (4) a decrease of sleepiness after wake. If our hypotheses were proved, light exposure could be a complementary therapy to medication, as a countermeasure to orthostatic hypotension.

Methods

Study participants

Healthy middle-aged and older adults (>55 years old) were recruited through online or poster advertisements. The screening procedure began with a telephone interview, involving a detailed explanation of the study. During the in-person screening appointment, all participants completed a consent form, a general medical questionnaire, the reduced Horne-Ostberg Morningness Eveningness Questionnaire (rMEQ) [9], the Pittsburgh Sleep Quality Index (PSQI) [10], the Alcohol Use Disorders Identification Test (AUDIT) [11], the Balance self-Efficacy Scale (BES) [12], the Cognitive Abilities Screening Instrument (CASI) [13], and the Ishihara test for color blindness [14]. Exclusion criteria included unstable current health (e.g. current infection), current smoker, abnormal color vision or other visual deficits (glasses were permitted), use of illegal drugs or drugs that could impact light sensitivity or sleep, drugs that increase BP, diagnosis of dementia, and excessive alcohol intake (AUDIT > 19) [11]. Individuals who had balance problems that could be explained by peripheral neuropathy were excluded. To control for circadian phase misalignment, we excluded shift workers and study applicants who had trans-meridian flights 3 months before study participation. The protocol was approved by the Stanford University Institutional Review Board and all procedures adhered to the principles outlined in the Declaration of Helsinki.

One week before the study, participants were instructed to keep a regular sleep-wake schedule (~8-hour sleep at night and to try to avoid daytime naps) to ensure stable circadian entrainment. Compliance to this outpatient segment of the study was verified using wrist actigraphs (Motionlogger, Ambulatory Monitoring Inc., Ardsley NY) and self-reported sleep logs.

Laboratory protocol

The study consisted of two 12-hour protocols, performed in a balanced crossover design, separated by at least 1 week. Participants came to the laboratory 3 hours before their habitual bedtime and were screened for proximal use of alcohol (Alco-Screen 02, Chematics, Inc., North Webster, IN) and illegal drugs (DrugCheck3, Express Diagnostics, Blue Earth, MN), the use of either of which was exclusionary. The timing of sleep during the week prior to entry was determined through examination of sleep logs [15] and actigraphy data (Motionlogger, Ambulatory Monitoring Inc., Ardsley, NY). From these data, an average sleep onset time was calculated and use as the study sleep onset time. The average sleep offset set was also calculated and used as the end of sleep time in the study, less 30 minutes (e.g. average at-home wake time = 07:26, in lab wake time = 06:56).

From the time of entry into the laboratory, participants remained in a windowless, light-controlled, sound-attenuated bedroom. Light levels in the room were produced by a broad spectrum fluorescent white light (~150 lux in the horizontal angle of gaze). Two hours prior to getting into bed, participants had their mobile and standing balance tested, as was their subjective and objective alertness. At 30 minutes before habitual sleep time, overhead lighting was decreased to a dim light (<15 lux) and polysomnographic and BP recording equipment was attached to the participant. At bedtime and during the first 7 hours of scheduled sleep, the room was in complete darkness (<0.05 lux), and participants were asked to try to sleep. Participants were allowed to sleep until 30 minutes prior to their habitual wake time (i.e. average wake time during the at-home monitoring), at which time they were awakened by a technician entering the room and turning the overhead fluorescent lights to ~150 lux. During the 30 minutes prior to the technician-enforced awakening, participants were exposed to either darkness (Dark) or a dawn simulation light (Light). Upon awakening and prior to getting out of bed, a saliva sample was collected in an untreated polypropylene tube. Once out of bed and within 2 minutes of

the room lighting being turned on, participants were tested for standing and mobile balance, given a test of objective and subjective alertness, then a second saliva sample was obtained.

Dawn simulation light

Dawn simulation consisted of a polychromatic light source (Philips Wake-up Light, HF3470) that exponentially increased from 0 to 250 lux during 30 minutes before wake-up time. The light illuminance, photon density, and correlated color temperature at 45 cm from the device were: (1) at 5 minutes after light onset: 1.2 lux, 1.9E+16/m^2·s, 1090°K; (2) at 15 minutes after light onset: 13 lux, 1.4E+17/m^2·s, 1500°K; (3) at 30 minutes after light onset: 250 lux, 2.4E+18/m^2·s, 2750°K.

Assessment of alertness

Subjective sleepiness was assessed using the Stanford Sleepiness Scale (SSS) [16], which measures immediate feelings of sleepiness using a one-question, 7-point Likert-like scale. Objective alertness was assessed using an auditory version of the psychomotor vigilance test (aPVT) [17, 18]. The aPVT was administered with a custom-built unit based on an Arduino Uno microcontroller board and hardware button. Data was logged by the Arduino and interfaced via Python (v. 2.7.12) with a computer running Linux (v. 4.8.0). The participant was asked to press a button as quickly as possible in response to a 1000 Hz tone. Tones were separated by a random interval of 1 to 6 seconds; the entire test lasted 10 minutes. Tones were delivered at a volume that the individual participant could hear and found comfortable. The median of the reaction times, and the number of lapses were analyzed for each session of the aPVT.

Salivary cortisol

Once collected, saliva samples were immediately frozen and transferred to a –80°C freezer within 24 hours. All samples were batch assayed for cortisol concentration at the end of the study using an enzyme-linked immunosorbent assay (Salimetrics, Carlsbad, CA). The functional sensitivity was 0.028 µg/dL and intra-assay coefficient of variances were <7% for values as low as 0.06 µg/dL. The change in salivary cortisol concentrations during the 30 minutes between the first and second cortisol sample (i.e. the so-called "awakening response") was calculated.

Standing and mobile balance

Standing and mobile balance were assessed using a Zeno Walkway (ProtoKinetics, Havertown, PA), a walkable sensor pad (0.61 × 4.3 m) with 16 levels of dynamic pressure detected through embedded 1-cm pressure sensors placed every 1.3 cm. For standing balance, participants were asked to stand motionless on the walkway in a comfortable position with their arms along their body and looking straight ahead with their eyes open for the first 30 seconds and then with their eyes closed for another 30 seconds. After standing balance was assessed, we asked the participant to walk back and forth on the walkway three times at a self-selected pace. They started the walk from an open end of the walkway towards a wall where they had to turn around and walk back through the open end of the walkway for one more meter. To reduce bias due to slowing during the turn, only the three walks from the wall to the open end were taken into account for the analysis. Balance-related metrics and temporal and spatial measures of gait were automatically calculated (ProtoKinetics Movement Analysis Software, v. 5.07C7) as the average of the three walks. To determine standing balance, we examined movement during the 30 seconds of standing while eyes closed by calculating the center of pressure path length [3]. To determine mobile balance, we examined the variance in stride velocity (stride length divided by gait cycle time) [3]. Of the 23 participants, 20 had usable results for both conditions of the standing balance assessment and 21 had usable results for both conditions of the mobile balance assessment.

Polysomnography

Polysomnography (PSG) included electroencephalography (C3/4, O1/2; [19]) referenced to an electrically neutral auricular electrode, bilateral electro-oculogram, and submental electromyogram (Siesta, Compumedics, Charlotte, NC). These data were analyzed using automated sleep scoring software (https://github.com/stanford-stages/stanford-stages) [20] which examined each 15 seconds of data to determine the probability of each stage of sleep (wake, N1, N2, N3, or rapid eye movement) in each 15-second epoch. To determine the impact of the lighting condition on sleep, we averaged across participants the probability of wake in each of the 15-second epochs occurring in the last 30 minutes of sleep (time during which the dawn simulator was active or would have been active). Of the 23 participants, due to failed PSG, 18 participants had usable data for both conditions and were included in subsequent analyses.

BP and HR measurements

HR and BP were monitored continuously throughout the night (Finapres Nova, Finapres Medical Systems, Enschede, Netherlands). The Finapres Nova uses infrared finger plethysmography to measure beat-to-beat BP non-invasively [21]. The device has been validated in older adults [22] and does not cause substantive interference with sleep [23]. A five-lead electrocardiogram (EKG) was also recorded on the device. BP data were cleaned (scored artifact, nonsense values, orphan points, and missing points; LabView 2018, v18.0) then binned into 20-second windows overlapping every 10 seconds. Windows with less than 50% of usable data were removed from the analysis. HR and R–R intervals (i.e. time between the R peaks of consecutive QRS complexes) were calculated, and all traces were visually checked for artifacts (Kubios HRV Premium, v.3.0.0). Occasional ectopic beats were identified and replaced with interpolated R–R interval data. Cleaned HR data were binned into 20-second windows overlapping every 10 seconds. Windows with less than 50% of usable data were removed from the analysis.

To avoid excluding large sections of the recording contaminated by movement artifacts, we used a sampling period of 2.5 minutes for HRV estimation. Power densities in the low-frequency (LF) band (0.04–0.15 Hz) and in the high-frequency (HF) band (0.15–0.50 Hz) were calculated for each 2.5-minute segment. Moreover, the LF-to-(LF + HF) ratio [LF/(LF + HF)] was

Table 1. Daytime function under the Dark and Light condition

	Morning			
	Dark		Light	
SSS (n = 23)	2.91 ± 1.08		3.09 ± 1.11	
PVT RT (ms) (n = 23)	327.15 ± 95.14		306.00 ± 56.59	
Number of PVT lapses (n = 23)	4.52 ± 6.25		2.70 ± 3.02	
Path length (cm) (n = 20)	1.04 ± 0.51		0.97 ± 0.36	
Coef. of variation of velocity (%) (n = 21)	6.75 ± 2.37		7.66 ± 3.98	
	Wake time		Wake time + 30 minutes	
	Dark	Light	Dark	Light
Cortisol (μg/dL) (n = 17)	0.26 ± 0.13	0.25 ± 0.12	0.44 ± 0.20	0.48 ± 0.20

PVT, Psychomotor Vigilance Test; RT, reaction time. Mean ± SD of several daytime function. The table displays the data of participants during their morning session after the awakening under the Dark and Light condition. No significant differences were found.

used as an index of sympathovagal balance. After cleaning all the data, 14 participants had usable data for both conditions and were included for the analysis of BP and 16 participants were included in analyses of HR and heart rate variability (HRV).

Statistical analysis

All data were analyzed with R (v. 3.5.0) and the R package lme4 [24]. Statistical analyses were carried out for each variable separately with the linear mixed-effects model analysis of variance for repeated measures with the fixed variables "light condition" (Light or Dark) and "time" and the random factors "participants" and "visit order." The p-values were based on Satterthwaite's corrected degree of freedom. Data were segmented into baseline (60→30 minutes prior to wake time), treatment (30→0 minutes prior to wake time), and post-wake (0→8 minutes after wake time) and analyzed separately for each segment. The sleep/wake transition was calculated using the last 5 minutes before wake and the minutes 3 to 8 after wake to be able to examine the changes in BP and HR due to standing. During baseline, all participants were scheduled to be sleeping in the dark. During treatment, all participants were receiving the dawn simulation or were in dark (crossover). During post-wake, all participants were awake and actively engaged with a member of the study team. All data are presented as mean ± SD.

Results

Out of a pool of approximately 115 applicants, 23 participants (10 men and 13 women, aged 67.3 ± 8.8 years) were in stable enough health and successfully fulfilled all study criteria. Of the 23 participants, 12 had a self-reported history of disrupted balance upon awakening. As such, in addition to analyzing the entire group of participants, we also examined subgroups of "Balance" (those who self-reported having a history of dizziness in the morning, 4 men and 8 women, aged 67.7 ± 9.1 years) and "Control" (those who did not self-report a history of dizziness in the morning, 6 men and 5 women, aged 66.5 ± 8.2 years).

Alertness

No significant effects of Light were found for the assessment of sleepiness (Table 1). The morning SSS scores, as well as median reaction time and the number of lapses of the aPVT test were not different between conditions. No differences were observed for the measures of sleepiness between the Balance and Control groups (Supplementary Table 1).

Salivary cortisol

While there was the expected increase in salivary cortisol concentrations after 30 minutes awake, we did not observe an effect of condition (Table 1). Similarly, there was no effect of condition on either the Balance or Control group when examined separately (Supplementary Table 1).

Standing and mobile balance

There was no impact of condition on either standing or mobile balance (Table 1). When examined separately, there was no effect of condition on either the Balance or Control group (Supplementary Table 1). Standing balance in the morning, however, was worse in the Balance Group as compared to the Control group ($p < 0.05$) (Supplementary Table 1).

PSG sleep

The PSG recording for the whole night of sleep did not show a significant difference between both groups for total sleep time (TST, Control group: 5.35 hours ± 1.2 hours vs. Dark group: 5.28 hours ± 0.9 hours) or sleep efficiency (SE, Control group: 77.5% ± 16.6% vs. Dark group: 77.4% ± 11.1%). However, if we look closer to the last hour of sleep, there was an impact of condition on sleep ($p < 0.0001$) such that the probability of wake gradually increased during the first 15 minutes of the light exposure, after which it plateaued until the enforced wake time (Figure 1C). Under Light, the overall probability of wake of the participants increased by 32.8%. In the Balance group, the timing of the increase in wake is delayed relative to the Control group (Supplementary Figure 1C).

Blood pressure

During Light, there was a decrease of 6.24 mmHg in systolic BP as compared to Dark ($p < 0.0001$) (Figure 1A). The time course of the change is such that under Dark, there is a gradual increase in systolic BP prior to wake time and this rise is dampened in Light.

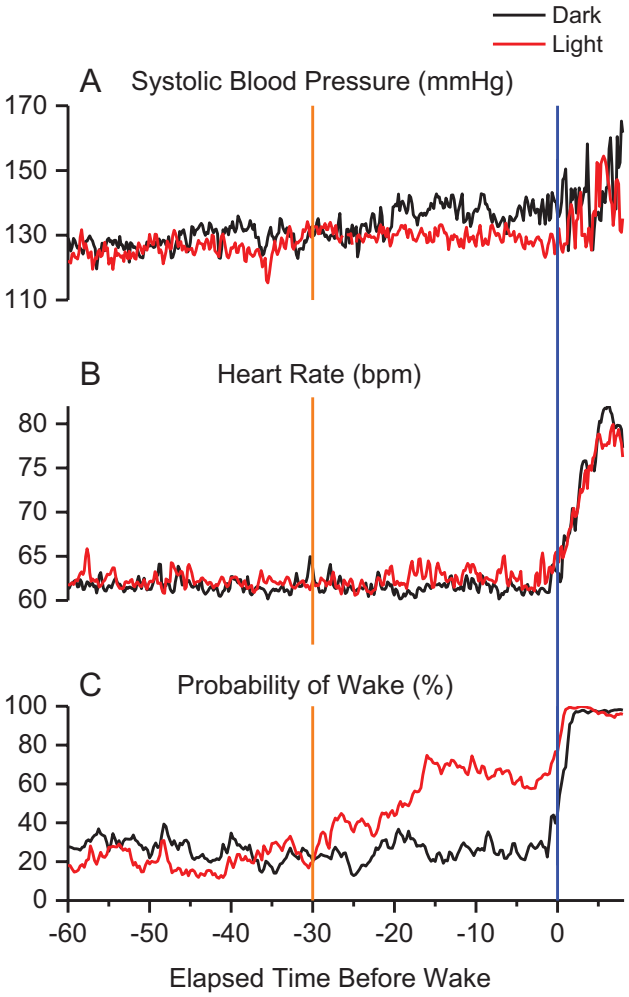

Figure 1. Profile of the blood pressure, heart rate, and probability of wake. Time course of the (A) systolic blood pressure, (B) heart rate, and the (C) probability of wake under the dark condition (black lines) or light condition (red lines). Data are plotted as a mean for each 20-second (blood pressure and heart rate) or 15-second (sleep) bin relative to elapsed time (minutes) before wake-up. The start of the experimental segment (Dark vs. Light condition) is represented with the orange line and the wake-up time with the blue line.

Upon arising, there continues to be a decrease of 2.65 mmHg in the systolic BP when previously exposed to Light ($p < 0.0001$). In comparing the difference of systolic BP between 5 minutes after wake while standing (minutes 3 to 8) and 5 minutes before wake (minutes –5 to 0), in the Dark, there is an increase of 7.13 ± 23.6 mmHg, while in the Light, there is an increase of 16.25 ± 17.44 mmHg; these are, however, not significantly different from one another ($p = 0.24$). In the Balance group, the increase observed after Light compare to Dark is 4.82 ± 25.6 mmHg, while it is increased by 15.14 ± 29.45 mmHg in the Control group. While large in value, these differences between the Control and Balance groups' response to Light are not significantly different ($p = 0.53$).

Heart rate

In the Dark, there is normally a dip in HR prior to awakening. In the Light, this dip is slightly attenuated by 0.93 bpm ($p < 0.001$, Figure 1B). After awakening, this effect is reversed, with a slight increase in HR following Dark as compared to Light (0.11 bpm, $p < 0.05$, Figure 1B).

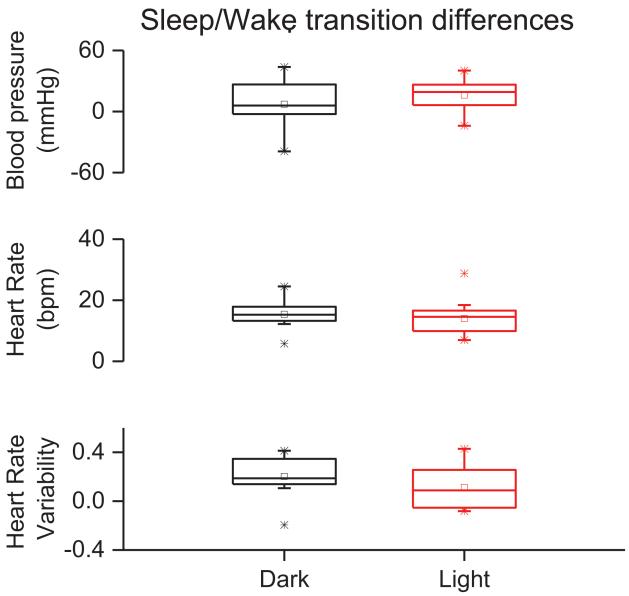

Figure 2. Sleep/wake transition differences of blood pressure, heart rate, and heart rate variability. Sleep–wake transition differences of blood pressure (A), heart rate (B), and heart rate variability (cardiac sympathetic [LF/(LF + HF) ratio]) (C) under dark (black) and light (red) condition. Data represent the difference between minute 3 to 8 after wake time and minute –5 to 0 before wake time.

When looking at the difference of HR between 5 minutes after wake (minute 3 to 8) and 5 minutes before wake (minute –5 to 0), there is an increase of HR of 15.38 ± 5.08 bpm following the Dark and 14.02 ± 5.46 bpm following the Light, though this difference is not significant ($p = 0.37$) (Figure 2B).

Those in the Balance group, as compared to the Control group, had an elevated HR (Control: 57.8 ± 7.00 bpm, Balance: 65.0 ± 3.79 bpm, $p < 0.001$) prior to wake time in both conditions (Supplementary Figure 1B). Following wake time, there was still an elevation, though it was smaller in magnitude (Control: 17.1 ± 5.94 bpm, Balance: 13.2 ± 4.29 bpm, $p < 0.05$). Additionally, in response to Light, there was an attenuation of the decrease in HR in the Control group ($p < 0.05$), but not in the Balance group ($p = 0.13$) (Supplementary Figure 1B).

Heart rate variability

Similar to HR, the decrease in the HRV LF/(LF+HF) ratio that occurs in Dark is attenuated in Light ($p < 0.05$). When looking at the difference of HRV at the sleep/wake time transition, we can see an increase of the ratio from 0.20 ± 0.16 after Dark and 0.13 ± 0.17 after Light; however, this difference is not significant ($p = 0.32$).

The Balance group has a lower HRV ratio in the Dark (Control: 0.66 ± 0.11, Balance: 0.52 ± 0.19, $p < 0.05$) (Supplementary Figure 2). There was no impact of Light on either group.

Discussion

In this study, we investigated whether a dawn simulation light is able to change cardiovascular function in middle-aged and older adults such that they would have reduced or absent problems with balance when they awoke in the morning. Our data show that while this dawn simulation light increases HR and HRV and

decreases BP, it does not have a significant impact on immediate measures of alertness or balance.

The results of the cardiac activity are aligned with previous studies. Scheer et al. showed that there is a larger increase in HR after a light exposure in the middle of the night or in the early morning in young adults [25]. Similarly, Viola et al. showed an increase in HR and in the cardiac sympathetic modulation during the light exposure before wake time in young people [5]. These effects might be attenuated in our study as, unlike the previous study, we also included women. Given our limited sample size, however, we did not have the power to accurately determine whether the impact of light was sex-dependent in our sample. We were also unable to determine if the dawn simulation had an impact on more delayed components of orthostasis, such as would occur after 10 or more minutes of standing.

Similarly, previous studies showed an increase of cortisol level after light exposure in the morning [8, 26, 27]; however, our results do not display such an increase in older people. There have been previously reported a difference in light sensitivity between young and older individuals in regards to phase shifting of the circadian clock [28]. Given the likely similar origins of the retinohypothalamic pathway impacting circadian and cardiovascular function [29, 30], it may be that older individuals are less sensitive to the impact of light on cardiovascular function. As the light exposure that was used was sufficient to disrupt sleep in the older participants, unlike previously observed in younger individuals [5], perhaps due to a decreased thalamic ability to filter out light information [31], increasing the light intensity would not be a tenable solution. Rather, the impact of increased light after awakening could be examined in future experiments. Further, potentially ensuring that the light only started in individuals who were asleep might have reduced the impact of light on inducing wake and the subsequent impacts on cardiovascular function.

The lack of significance while analyzing the HRV data could also be due to our specific methods. Indeed, recent studies have shown that the LF/HF ratio might not accurately measure the cardiac sympathovagal balance, due to the large variation that can occur while recording LF component [32–34]. However, we included this measure in our paper to be comparable with the previous study using a dawn simulation light. Additionally, we did not control for respiratory frequency in this study and thus present other variables less impacted by the breathing. But as it is a main determinant of HRV, respiratory frequency should be considered in future studies.

While there were some impacts on cardiovascular function, we did not observe any impacts on daytime function that we had anticipated might change. Measures of subjective and objective alertness were not different between conditions, which is consistent with that observed in the young following a similar stimulus [7, 35]. As our participants had generally good sleep prior to study and were not sleep-restricted, it might be that they were already at a peak performance level in the control condition such that the light exposure could not further increase their performance on this test. Moreover, it has been shown that sleep inertia significantly impaired cognition in younger but not older adults [36], reinforcing, even more, the hypothesis that the older participants were at a peak performance level at wake time.

Similarly, we did not observe an impact of light on either standing or mobile balance. Balance in the morning was notably worse in the participants who had reported a history of morning dizziness. There was not, however, a differential impact of light on balance in either the Balance or Control groups. The measures of standing and mobile balance that we selected are useful as predictors of future falls [37–40]. It is possible that other aspects of balance are impacted by the changes in sleep or cardiovascular function, but the ones most associated with risk of falling were not. We also did not study whether the light enhanced subjective perception of balance, which is a critical moderator of actual balance [39] and should be considered in future studies.

As this was an acute study, we did not examine whether repeated exposure to the dawn simulation light would result in adaptive changes. That is, would older individuals exposed to such a stimulus for weeks adapt to the light and have less induced wake, and if such a reduction would enhance or diminish the cardiovascular impact of the light. Future studies with a higher number of participants will be needed to examine such longer-term responses. To better understand the physiology of people at risk of falling, future studies could include a formal clinical exam that would be helpful in determining specific pathologies that might be differentially impacted by the dawn simulation light. An analysis of BP and HR during the whole night could also be relevant to help have a clearer interpretation of the results.

Taken together, our data indicate that a dawn simulation light exposure does not improve morning balance in older individuals. In the acute setting, it does, however, increase the likelihood of wake at the end of the sleep episode, which is a notable limitation of such a device in this population.

Supplementary material

Supplementary material is available at *SLEEP* online.

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References

1. Jones PK, *et al*. Orthostatic hypotension: managing a difficult problem. *Expert Rev Cardiovasc Ther*. 2015;**13**(11):1263–1276.
2. Cajochen C, *et al*. Dose-response relationship for light intensity and ocular and electroencephalographic correlates of human alertness. *Behav Brain Res*. 2000;**115**(1):75–83.
3. McBean AL, *et al*. Standing balance and spatiotemporal aspects of gait are impaired upon nocturnal awakening in healthy late middle-aged and older adults. *J Clin Sleep Med*. 2016;**12**(11):1477–1486.
4. Scheer FA, *et al*. Cardiovascular control by the suprachiasmatic nucleus: neural and neuroendocrine mechanisms in human and rat. *Biol Chem*. 2003;**384**(5):697–709.
5. Viola AU, *et al*. Dawn simulation light: a potential cardiac events protector. *Sleep Med*. 2015;**16**(4):457–461.

6. Giménez MC, *et al*. Effects of artificial dawn on subjective ratings of sleep inertia and dim light melatonin onset. *Chronobiol Int*. 2010;**27**(6):1219–1241.
7. Van De Werken M, *et al*. Effects of artificial dawn on sleep inertia, skin temperature, and the awakening cortisol response. *J Sleep Res*. 2010;**19**(3):425–435.
8. Gabel V, *et al*. Effects of artificial dawn and morning blue light on daytime cognitive performance, well-being, cortisol and melatonin levels. *Chronobiol Int*. 2013;**30**(8):988–997.
9. Adan A, *et al*. Horne & Östberg morningness-eveningness questionnaire: a reduced scale. *Personality and Individual Differences*. 1991;**12**(3):241–253.
10. Buysse DJ, *et al*. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;**28**(2):193–213.
11. Saunders JB, *et al*. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption–II. *Addiction*. 1993;**88**(6):791–804.
12. Rose DJ. *Fallproof!: A Comprehensive Balance and Mobility Training Program*. Champaign, IL: Human Kinetics.; 2003.
13. Teng EL, *et al*. The Cognitive Abilities Screening Instrument (CASI): a practical test for cross-cultural epidemiological studies of dementia. *Int Psychogeriatr*. 1994;**6**(1):45–58; discussion 62.
14. Ishihara S. *Ishihara's Tests for Colour Deficiency*. Tokyo, Japan: Kanehara Trading Inc.; 2007.
15. Carney CE, *et al*. The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep*. 2012;**35**(2):287–302.
16. Hoddes E, *et al*. Quantification of sleepiness: a new approach. *Psychophysiology*. 1973;**10**(4):431–436.
17. Dinges DF, *et al*. Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Behav Res Methods Instrum Comput*. 1985;**17**(6):652–655.
18. Gabel V, *et al*. Auditory psychomotor vigilance testing in older and young adults: a revised threshold setting procedure. *Sleep Breath*. 2019;**23**(3):1021–1025.
19. Klem GH, *et al*. The ten-twenty electrode system of the International Federation. The International Federation of Clinical Neurophysiology. *Electroencephalogr Clin Neurophysiol Suppl*. 1999;**52**:3–6.
20. Stephansen JB, *et al*. Neural network analysis of sleep stages enables efficient diagnosis of narcolepsy. *Nat Commun*. 2018;**9**(1):5229.
21. Waldron M, *et al*. Inter-day reliability of finapres ® cardiovascular measurements during rest and exercise. *Sports Med Int Open*. 2018;**2**(1):E9–E15.
22. Rongen GA, *et al*. Comparison of intrabrachial and finger blood pressure in healthy elderly volunteers. *Am J Hypertens*. 1995;**8**(3):237–248.
23. Wibmer T, *et al*. Impact of continuous, non-invasive blood pressure measurement on sleep quality during polysomnography. *Sleep and Biological Rhythms*. 2013;**11**:254–260.
24. Bates D, *et al*. Fitting Linear mixed-effects models using lme4. *J Stat Soft*. 2015;**67**(1):1–48.
25. Scheer FA, *et al*. Light and diurnal cycle affect human heart rate: possible role for the circadian pacemaker. *J Biol Rhythms*. 1999;**14**(3):202–212.
26. Scheer FA, *et al*. Light affects morning salivary cortisol in humans. *J Clin Endocrinol Metab*. 1999;**84**(9):3395–3398.
27. Thorn L, *et al*. The effect of dawn simulation on the cortisol response to awakening in healthy participants. *Psychoneuroendocrinology*. 2004;**29**(7):925–930.
28. Duffy JF, *et al*. Decreased sensitivity to phase-delaying effects of moderate intensity light in older subjects. *Neurobiol Aging*. 2007;**28**(5):799–807.
29. Chellappa SL, *et al*. In a heartbeat: light and cardiovascular physiology. *Front Neurol*. 2017;**8**:541.
30. Johnson RF, *et al*. Retinohypothalamic projections in the hamster and rat demonstrated using cholera toxin. *Brain Res*. 1988;**462**(2):301–312.
31. Daneault V, *et al*. Light-sensitive brain pathways and aging. *J Physiol Anthropol*. 2016;**35**:9.
32. Shaffer F, *et al*. An overview of heart rate variability metrics and norms. *Front Public Health*. 2017;**5**:258.
33. Shaffer F, *et al*. A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. *Front Psychol*. 2014;**5**:1040.
34. Billman GE. The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. *Front Physiol*. 2013;**4**:26.
35. Gabel V, *et al*. Dawn simulation light impacts on different cognitive domains under sleep restriction. *Behav Brain Res*. 2015;**281**:258–266.
36. Frey DJ, *et al*. Influence of zolpidem and sleep inertia on balance and cognition during nighttime awakening: a randomized placebo-controlled trial. *J Am Geriatrics Soc*. 2011;**59**:73–81.
37. Piirtola M, *et al*. Force platform measurements as predictors of falls among older people - a review. *Gerontology*. 2006;**52**(1):1–16.
38. Muir JW, *et al*. Dynamic parameters of balance which correlate to elderly persons with a history of falls. *PLoS One*. 2013;**8**(8):e70566.
39. Maki BE. Gait changes in older adults: predictors of falls or indicators of fear. *J Am Geriatr Soc*. 1997;**45**(3):313–320.
40. Mortaza N, *et al*. Are the spatio-temporal parameters of gait capable of distinguishing a faller from a non-faller elderly? *Eur J Phys Rehabil Med*. 2014;**50**(6):677–691.