Strategies to decrease social jetlag: Reducing evening blue light advances sleep and melatonin

Giulia Zerbini[1,2] | **Thomas Kantermann**[1,3,4] | **Martha Merrow**[1,2]

[1]Chronobiology Unit, Groningen Institute for Evolutionary Life Sciences, University of Groningen, Groningen, The Netherlands

[2]Institute of Medical Psychology, LMU Munich, Munich, Germany

[3]SynOpus, Bochum, Germany

[4]University of Applied Sciences for Economics and Management (FOM), Essen Germany

Correspondence
Giulia Zerbini and Martha Merrow, Institute of Medical Psychology, LMU Munich, Munich, Germany.
Emails: giulia.zerbini87@gmail.com and merrow@lmu.de

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Abstract

The timing of sleep is under the control of the circadian clock, which uses light to entrain to the external light-dark cycle. A combination of genetic, physiological and environmental factors produces individual differences in chronotype (entrained phase as manifest in sleep timing). A mismatch between circadian and societal (e.g., work) clocks leads to a condition called social jetlag, which is characterized by changing sleep times over work and free days and accumulation of sleep debt. Social jetlag, which is prevalent in late chronotypes, has been related to several health issues. One way to reduce social jetlag would be to advance the circadian clock via modifications of the light environment. We thus performed two intervention field studies to describe methods for decreasing social jetlag. One study decreased evening light exposure (via blue-light-blocking glasses) and the other used increased morning light (via the use of curtains). We measured behaviour as well as melatonin; the latter in order to validate that behaviour was consistent with this neuroendocrinological phase marker of the circadian clock. We found that a decrease in evening blue light exposure led to an advance in melatonin and sleep onset on workdays. Increased morning light exposure advanced neither melatonin secretion nor sleep timing. Neither protocol led to a significant change in social jetlag. Despite this, our findings show that controlling light exposure at home can be effective in advancing melatonin secretion and sleep, thereby helping late chronotypes to better cope with early social schedules.

KEYWORDS

behaviour, chronotype, circadian, light, phase of entrainment

1 | INTRODUCTION

Sleep is a basic human need. It is essential for good health and optimal performance. A two-process model of sleep regulation posits that an interaction between homeostatic (sleep pressure) and circadian processes leads to a consolidated bout of sleep within a given window of time (Borbély, 1982; Daan, Beersma, & Borbély, 1984). The circadian clock synchronizes (entrains) to the external light-dark cycle, thereby adopting a specific phase relationship (Duffy & Wright, 2005; Roenneberg & Merrow, 2007; Wright et al., 2013). Variations in genetic background, sex, age and light exposure

Abbreviations: CoG_{act}, center of gravity actigraphy; DLMO, Dim-Light Melatonin Onset; MCTQ, Munich ChronoType Questionnaire; MSF, midpoint of sleep on work-free days; MSF_{sc}, midpoint of sleep on work-free days (sleep corrected); MSW, midpoint of sleep on workdays; PSQI, Pittsburgh Sleep Quality Index; SD_w, sleep duration on workdays; SE_w, sleep end on workdays; SJL, social jetlag; SO_w, sleep onset on workdays.

lead to a distribution of entrained circadian phases or chronotypes (Duffy & Wright, 2005; Hamet & Tremblay, 2006; Roenneberg & Merrow, 2007; Roenneberg et al., 2004, 2007). Several phase markers have been adopted to estimate entrained phase. In this work, we use timing of sleep, center of gravity of activity (CoG_{act}) and changes in Dim-Light Melatonin Onset (DLMO) relative to local time as phase markers for behavior and physiology.

The difference between local time (external and collective) and biological time (internal and individual) can be substantial. When standardized social schedules are imposed broadly, such as school and work times, different chronotypes study and work at the same local time, although their peak cognitive and physical performance typically varies according to biological time. The mismatch between biological and social time is called social jetlag (SJL; Wittmann, Dinich, Merrow, & Roenneberg, 2006). SJL can be assessed as the absolute difference between the midpoint of sleep on workdays (MSW) and on work-free days (MSF). SJL is usually greater in later chronotypes (those who sleep late), which also means that late chronotypes are typically more sleep deprived during the school/working week (Roenneberg et al., 2007; Wittmann et al., 2006).

In previous studies, SJL (>2 hr) has been associated with a decline in academic performance (Haraszti, Ella, Gyöngyösi, Roenneberg, & Káldi, 2014) and with several health issues, such as increased risk for cigarettes and alcohol consumption, overweight, cardiovascular risk, diabetes and depression (Kantermann et al., 2013; Koopman et al., 2017; Larcher et al., 2016; Levandovski et al., 2011; Mota, Silva, Balieiro, Fahmy, & Crispim, 2017; Roenneberg, Allebrandt, Merrow, & Vetter, 2012; Rutters et al., 2014; Wittmann et al., 2006; Wong, Hasler, Kamarck, Muldoon, & Manuck, 2015). Here, we sought to reduce SJL through practical interventions using light, given that light is the strongest zeitgeber for human behavioural entrainment (Duffy & Wright, 2005; Roenneberg & Foster, 1997; Roenneberg & Merrow, 2007).

All light qualities—such as its timing, spectral quality, intensity and duration– contribute to circadian entrainment (Duffy & Wright, 2005; Roenneberg & Foster, 1997). When exposed to light at different times of day, all circadian clocks, including those in humans, can respond with advances or delays (Khalsa, Jewett, Cajochen, & Czeisler, 2003). In particular, light exposure during the evening (beginning of the biological night) leads to phase delays, and light exposure during the morning (end of the biological night) results in phase advances (Khalsa et al., 2003; Roenneberg, Hut, Daan, & Merrow, 2010). Some field studies have already shown that interventions involving morning and/or evening light exposure can influence phase of entrainment estimated via DLMO (Appleman, Figueiro, & Rea, 2013; Geerdink, Walbeek, Beersma, Hommes, & Gordijn, 2016). The colour of light also plays an important role, with short-wavelength light (480 nm) being most effective in shifting the phase/timing of DLMO (Brainard et al., 2001; Santhi et al., 2012; Wright & Lack, 2009). If wavelength is kept constant, the response to different light intensities and durations is dose-dependent, albeit not always with a linear relationship (Gooley et al., 2010; St Hilaire et al., 2012; West et al., 2011; Zeitzer, Dijk, Kronauer, Brown, & Czeisler, 2000).

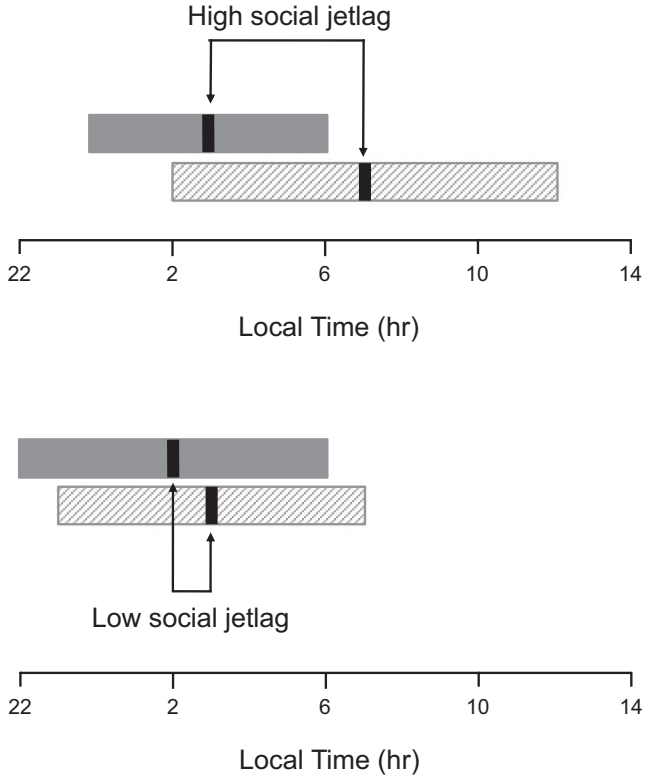

FIGURE 1 Decreasing social jetlag (SJL) with light. Sleep timing and duration are represented with grey bars for workdays and striped bars for work-free days. The vertical black lines represent the midpoint of sleep on workdays (MSW) and on work-free days (MSF). SJL is calculated as the absolute difference between MSW and MSF. Both interventions (blue-light-blocking glasses late in the day and increased indoor light early in the day) are expected to advance phase of entrainment (assessed via Dim-Light Melatonin Onset; DLMO) and thus sleep timing (see bottom cartoon vs. top). This should lead to longer sleep duration on weekdays, thus a reduction in accumulated sleep debt and less oversleep on work-free days. SJL is predicted to decrease via a better alignment between MSW and MSF

Here, we developed two in situ protocols to reduce SJL in late chronotypes by advancing their phase of entrainment. In Study 1, subjects used blue-light-blocking glasses (orange filters) to decrease evening blue light. In Study 2, subjects were asked to sleep with opened curtains in order to selectively increase morning light entering their bedrooms. We hypothesized that both protocols (less evening or more morning light) should advance the phase of entrainment in late chronotypes. This advance should lead to earlier sleep times,

longer sleep on workdays, reduced oversleep on work-free days, and should thereby decrease SJL, i.e., lead to a better alignment of sleep times on work- and on free days (Figure 1). In both protocols, we aimed to test the effectiveness of simple, in-home interventions on entrained phase with the goal of applications suited to real-life conditions.

2 | METHODS

2.1 | Study 1—Decreasing blue light at the end of the internal day

2.1.1 | Participants

The study was run in February 2015 in Groningen (53°13′N/6°33′E), The Netherlands. Participants were recruited via flyer and online advertisement. Eighty-six individuals were screened. Ten individuals were not anymore interested in participating in the study after the screening phase. Of the remaining 76 eligible participants, 40 (24 females) were selected for the study based on their amount of SJL (at least 1.5 hr of SJL was required). SJL was assessed via the Munich Chronotype Questionnaire (Roenneberg, Wirz Justice, & Merrow, 2003) as the absolute difference between the midpoint of sleep on work-free days (MSF) and the midpoint of sleep on workdays (MSW; Wittmann et al., 2006). The sleep data collected with the MCTQ were further used to assess chronotype. The MCTQ references chronotype to the midpoint of sleep on work-free days (MSF), corrected for sleep debt accumulated on workdays (MSF_{sc}; Roenneberg et al., 2012).

Participants were generally healthy, had no sleep complaints, and did not report use of sleep medication. They had a regular working schedule (at least four working days per week), had not performed shift work during the past 5 years, and had not travelled across more than two time zones during the month prior to the study. Females were selected only if they were premenopausal and made use of hormonal contraceptives (to avoid possible fluctuations in melatonin levels dependent on the phase of the menstrual cycle; Lee Barron, 2007).

At the beginning of the study, two participants (one female) dropped out (one for each group), leaving a cohort of 38 participants of 19–47 years of age (mean age 23.7 ± *SD* 5.5). The reasons for dropping out were personal and not related to the study (e.g., moving away because of a new job).

2.1.2 | Protocol

The study lasted 4 weeks (Figure 2a; from 02.02.15 to 01.03.15). The participants were randomly assigned to either the control group or the intervention group. Stratification was used during randomization to achieve a similar distribution of the variables age, sex, MSF_{sc} and SJL in the two groups.

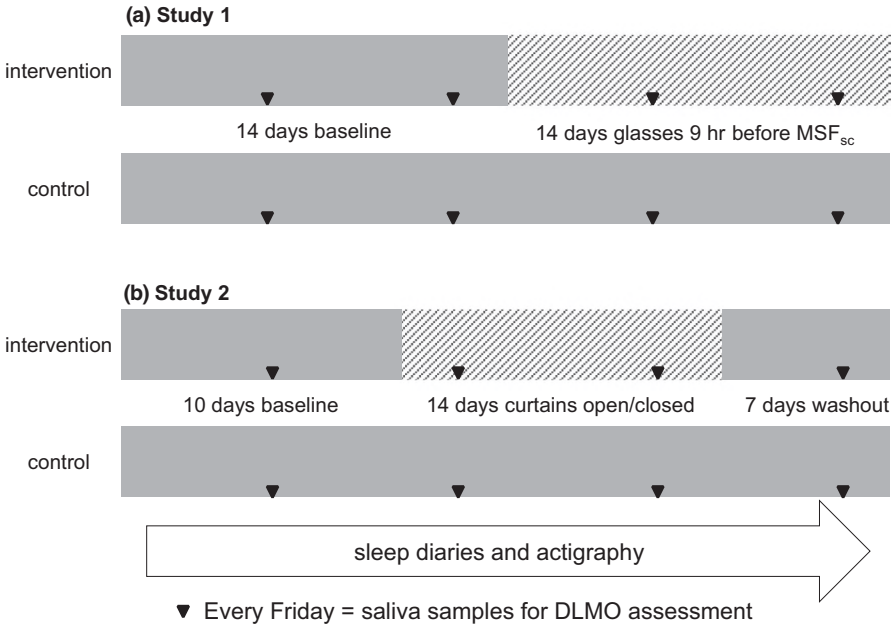

FIGURE 2 Experimental design. (a) Protocol used in Study 1. Following 2 baseline weeks of assessment (grey blocks), the intervention group wore orange (blue-light-blocking) glasses for 2 weeks (striped grey blocks). The control group wore glasses with clear lenses. (b) Protocol used in Study 2. Following 10 baseline days (grey blocks), the intervention group slept with bedroom curtains open for 2 weeks (striped grey blocks) and then with bedroom curtains closed for 1 week (grey blocks). The control group slept with bedroom curtains closed throughout the study. In both studies, participants collected saliva samples every Friday (4 times), filled in a sleep diary daily, and wore an actimeter continuously (for light and activity measurements). The days corresponding to saliva collection are indicated with black triangles

After 2 weeks of baseline, participants wore a special pair of glasses (AugenLichtSchutz, Königswinter, DE) every evening for the remaining 2 weeks of the study. The control group wore glasses with clear lenses (no filter of blue light between 400 and 500 nm, general decrease of light intensity: 8%; for more details see Supporting Information Figure S1). The intervention group wore glasses with blue-light-blocking lenses (89%–99.9% filter of blue light between 400 and 500 nm, general decrease of light intensity: 50%; for more details see Supporting Information Figure S2). The participants started wearing the glasses 9 hr before their internal midnight. Therefore they wore the glasses 9 hr before the local time indicated by their chronotype (MSF_{sc}) and wore them until they turned the lights off to go to sleep. In this way, we aimed to apply the intervention at the same internal time for all participants.

2.2 | Study 2—Increasing bedroom light at the beginning of the internal day

2.2.1 | Participants

The study was conducted in March 2016 in Groningen (53°13′N/6°33′E), The Netherlands. Participants were recruited via flyers, online advertisements and internal posting at the University of Groningen. The same inclusion and exclusion criteria (general health, regular working schedule, no shiftwork or travelling across time zones) were applied to select the participants as described for Study 1. In addition, only people who habitually slept with dark and closed curtains could sign up for the study. Of the 118 individuals that were screened, 94 met the inclusion criterion regarding the use of the curtains. The 40 (22 females) eligible applicants with the latest chronotypes were selected for the study. Two dropouts at the beginning of the study (both in the intervention group) left 38 participants (20 females, mean age 22.8 ± *SD* 3.1, range 19–35) who completed the study. The reasons for dropping out were personal and not related to the study (e.g., moving away because of a new job).

2.2.2 | Protocol

The study lasted 30 days (Figure 2b; from 26.02.16 to 26.03.16). Participants were randomly assigned to either the control or the intervention group. Stratification was used during randomization to achieve a similar distribution of the variables age, sex, MSF_{sc} and SJL in the two groups.

The control group slept with curtains closed throughout the protocol. The intervention group slept with curtains closed for 10 days (baseline), then for 14 days with curtains open (intervention weeks) and again with curtains closed for 7 days (wash-out week). After both baseline and intervention weeks, the participants filled in the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) to assess whether sleeping with open curtains influenced subjective sleep quality.

2.3 | Studies 1 and 2

Both studies were conducted according to the principles of the Medical Research Involving Human Subjects Act (WMO, 2012), and the Declaration of Helsinki (64th WMA General Assembly, Fortaleza, Brazil, October 2013). The Medical Ethical Committee of the University Medical Center Groningen approved both studies. The participants signed a written informed consent and received financial compensation for taking part in the studies.

2.3.1 | Behavioural and physiological assessment

During both protocols, the participants filled in a daily sleep diary and continuously wore an actimeter at the wrist on their non-dominant arm (Study 1: Daqtometer Version 2.3, Daqtix GbR, Suettorf, DE; Study 2: MotionWatch 8, CamNtech, Cambridge, UK). The actimeters recorded both activity and light intensity levels (Daqtometer: relative light levels; MotionWatch 8: light levels expressed in lux). Data were stored in intervals of 30 s and subsequently binned into 10-min intervals for further analysis. Actigraphy data (sleep timing and center of gravity of activity; CoG_{act}) were processed with *ChronoSapiens* (version 9.0; Roenneberg et al., 2015). Since eight participants had missing actigraphy data, we decided to only report sleep times derived from the sleep diaries. Sleep times assessed with sleep diaries and with actigraphy were significantly correlated (e.g., sleep onset: $R^2 = 0.58$, $p < 0.0001$). In Study 2, participants also used a light sensor (HOBO pendant temperature/light 64K data logger, Onset, Bourne, MA, USA) in the bedroom to assess light intensities (relative changes compared to baseline) throughout the study.

In addition to chronotype and CoG_{act}, circadian phase was also estimated by assessing Dim-Light Melatonin Onset (DLMO) from saliva samples. Every Friday evening, the participants collected seven saliva samples at home in hourly intervals, starting 5 hr before and finishing 1 hr after habitual sleep onset (weighted average sleep onset on workdays and on work-free days, based on the participants' answers to the MCTQ). Participants were requested to dim their home lighting, and to start wearing a pair of blue-light blocking glasses (if not already doing so as part of their protocols) half an hour before the collection of the first sample and until the collection of the last sample. During this time, the use of toothpaste and the ingestion of coffee, tea, alcohol, chocolate, bananas and food with artificial additives were not allowed. The saliva samples were collected using Salivettes (Sarstedt, Nümbrecht, DE). The samples were kept in the refrigerator and sent per mail to our lab within 3 days. Upon arrival, the samples were frozen at −80°C and subsequently analyzed using direct saliva melatonin radioimmunoassay (RIA) test kits (Bühlmann, Schönenbuch, CH).

DLMO was calculated by linear interpolation between the time points before and after melatonin concentrations crossed and stayed above the threshold of 3 pg/ml. In Study 1, the melatonin levels of 12 participants in week 1 and 14 participants in week 2 did not reach (or stayed above) the threshold of 3 pg/ml, and it was therefore not possible to assess DLMO in these participants. Similarly in Study 2, the melatonin levels of 10 participants in week 1 and 11 participants in week 2 did not reach (or stayed above) the threshold of 3 pg/ml. These numbers of participants did not differ statistically between the two groups of Study 1 and Study 2. The lower limit of detection of the kit was <0.5 pg/ml. In Study 1, the intra-assay variability was 19.8% (low melatonin) and 22.1% (high melatonin), while the inter-assay variability was 14.7% (low melatonin) and 16.5% (high melatonin). In Study 2, the intra-assay variability was 12.6% (low melatonin) and 16.2% (high melatonin), while the inter-assay variability was 14.7% (low melatonin) and 13.3% (high melatonin).

2.4 | Statistical analysis

Statistical analyses were done using software R (R version 3.3.0; The R Core team, 2013; nlme package). Data about DLMO, sleep timing and activity were analyzed with a mixed model with group (control/intervention) and time (week of protocol) as fixed factors and ID (participants' identification) as random factor. Weekly (DLMO) and daily (sleep timing and activity) values were entered in the models. In Study 1, we used a 2×3 design, with group as between-subject factor and time as within-subject factor (baseline/week 1/week 2). The first 2 weeks of the study were treated as baseline. In Study 2, we used a 2×4 design, with group as between-subject factor and time as within-subject factor (baseline/week 1/week 2/wash-out week). The first 10 days of the study were treated as baseline. In Study 1, morning light (10-min bin light levels (lux) averaged across 6 hr between 6:00 and 12:00 hr) was analyzed as a covariate to control for the advancing effects that morning light potentially has on phase of entrainment and sleep. In Study 2, evening light (10-min bin light levels (lux) averaged across 6 hr between 18:00 and 0:00 hr) was analyzed as a covariate to control for the delaying effects that evening light potentially has on phase of entrainment and sleep.

When the interaction effect between group and time was significant, the change in the variables of interest during the first intervention week and the second intervention week (and the wash-out week in Study 2) relative to baseline was analyzed comparing the two groups (control/intervention) with one-tailed independent *t* tests (a-priori prediction for the one-tailed *t* test: DLMO, sleep timing and CoG_{act} of the participants in the intervention group are earlier during the intervention weeks compared to baseline; Bonferroni correction was applied for multiple comparisons). In addition, in Study 2 the changes in bedroom light intensities and in sleep quality (PSQI) between baseline and during/after the intervention were compared between the two groups with a Mann–Whitney-Wilcoxon test. Standard deviations (*SD*) are reported in the text and in the Tables, while standard errors of the mean (*SEM*) are plotted in the figures.

3 | RESULTS

The results from Study 1 (less blue light in the evening) and Study 2 (more light in the morning) are described in the following paragraphs.

3.1 | Study 1—Decreasing blue light at the end of the internal day

The demographics of the participants are reported in Table 1. The groups were not significantly different according to sex, age, chronotype and SJL (Independent *t* tests for age: $t\ (36) = -0.116$, $p = 0.909$; chronotype: $t\ (36) = -1.312$, $p = 0.198$; SJL: $t\ (36) = -0.334$, $p = 0.740$).

The analysis of the variables assessed with the daily sleep diaries revealed significant changes in sleep timing on workdays, but not on work-free days, in individuals that wore blue-light-blocking glasses. In all analyses, morning light (assessed at the wrist between 6:00 and 12:00 hr) was analyzed as a covariate. In particular, we found that sleep onset on workdays (SO_w) changed across the protocol in the intervention group, while it did not vary in the control group (group-time interaction effect; $F_{2,498} = 3.514$, $p = 0.031$). We therefore explored this difference between groups over time (separating week 1 and week 2; Figure 3a,b). SO_w advanced (relative to baseline) in the intervention group (by 36 ± 45 min) after week 1, while it did not change in the control group (delay by 2 ± 45 min; $t\ (36) = -2.606$, $p = 0.013$; Cohen's $d = 0.87$). According to Cohen's guidelines, this was a large effect (Cohen, 1988). After week 2, SO_w remained earlier relative to baseline (advance by 18 ± 50 min) in the intervention group and stable in the control group (advance by 30 s ± 44 min). However, the difference between the groups (week 2 relative to baseline) was no longer statistically significant, $t\ (35) = -1.136$, $p = 0.264$.

The changes in DLMO across the protocol and between groups mirrored the changes in SO_w (group-time interaction effect; Figure 3c,d; $F_{2,59} = 2.872$, $p = 0.065$). Compared to the controls, the intervention group showed a significant advance in DLMO (by 32 ± 37 min relative to baseline DLMO) during week 1, $t\ (24) = -2.402$, $p = 0.024$; Cohen's $d = 0.98$, but not during week 2, $t\ (22) = -0.388$, $p = 0.702$. The phase angle difference between DLMO and SO_w also changed only for the intervention group and only during week 1 (group-time interaction effect; Figure 3e,f; $F_{2,59} = 6.622$, $p = 0.003$). While it remained stable across the protocol for the controls, the intervention group showed

TABLE 1 Demographic characteristics of 38 (23 females) participants in Study 1 randomly assigned to control and intervention groups

	Control group		Intervention group	
Parameter	**Average (*SD*)**	**Range**	**Average (*SD*)**	**Range**
Age	23.6 (7.0)	19–47	23.8 (3.7)	19–33
Chronotype (MSF_{sc}, hr:min)	5:01 (62′)	3:10–7:12	5:29 (69′)	3:30–8:24
Social Jetlag (hr:min)	2:04 (30′)	1:27–3:27	2:07 (28′)	1:34–3:27
Sleep onset on workdays (hr:min)	23:42 (50′)	22:25–1:15	23:52 (68′)	22:10–2:40
Sleep end on workdays (hr:min)	7:11 (48′)	5:45–8:30	7:37 (72′)	5:10–9:25
Sleep duration on workdays (hr:min)	7:28 (52′)	5:10–9:25	7:45 (52′)	5:15–9:19
Sleep onset on work-free days (hr:min)	1:03 (65′)	22:55–3:15	1:27 (75′)	23:40–4:10
Sleep end on work-free days (hr:min)	9:58 (55′)	8:00–12:00	10:16 (76′)	8:15–13:00
Sleep duration on work-free days (hr:min)	8:55 (40′)	7:25–10:04	8:49 (54′)	7:10–10:30

Note. The two groups did not significantly differ in relation to their demographic and sleep characteristics at baseline. Data concerning chronotype, sleep onset and sleep end refer to local clock time.

a smaller phase angle difference (on average 31 ± 27 min smaller) during week 1, $t\ (21) = 2.103$, $p = 0.048$. To better understand why the intervention was effective only during week 1, we looked at the changes in DLMO of each individual participant that wore the blue-light-blocking glasses during week 1 and week 2 (Supporting Information Figure S3). Three groups of responders could be identified: (a) those who progressively advanced or showed a stable earlier phase of entrainment (advancing/stabilizing group); (b) those who advanced during week 1 and delayed during week 2 (advancing/reverting group); (c) those who delayed during both weeks (delaying group). We then plotted the light exposure of these three groups of participants for each week of the protocol (Supporting Information Figure S4). While for the advancing/stabilizing group the light exposure across the weeks of the protocol was quite similar, the light exposure of the advancing/reverting group varied during week 2 compared to week 1 and to baseline. In absolute numbers, the participants were exposed to higher amounts of light approximately between 16:00 and 19:00 hr, which, for most of them, corresponded to the time before wearing the glasses. Light exposure, however, was not statistically significant different between the study weeks.

Sleep end and sleep duration on workdays (SE_w and SD_w, respectively) did not change between groups and across time (group-time interaction effects; SE_w: $F_{2,500} = 0.796$, $p = 0.452$; SD_w: $F_{2,497} = 0.995$, $p = 0.371$). There was a trend indicating that the intervention group slept longer (by on average 17 ± 34 min) compared to the control group during week 1 relative to baseline, $t\ (36) = 1.873$, $p = 0.069$. SJL also failed to change between groups and across time (group-time interaction effect; $F_{2,80} = 0.376$, $p = 0.688$). The same was true for sleep timing and duration on work-free days. Finally, center of gravity (CoG_{act}) did not change, neither on work- nor on free days (group-time interaction effects; workdays: $F_{2,490} = 0.368$, $p = 0.692$; work-free days: $F_{2,230} = 0.807$, $p = 0.448$). The weekly averages together with the standard deviations (*SD*) of the parameters assessed are reported in Supporting Information Table S1.

Overall, participants were exposed to comparable light levels (morning light between 6:00 and 12:00 hr: $F_{2,788} = 1.069$, $p = 0.344$; evening light between 18:00 and 00:00 hr: $F_{2,760} = 1.224$, $p = 0.295$; Supporting Information Figure S5), and therefore any change observed in the variables of interest is likely to be related to wearing the blue-light-blocking glasses and not to differences in light exposure between groups and across the protocol.

3.2 | Study 2—Increasing bedroom light at the beginning of the internal day

The demographics of the participants are reported in Table 2. In the control group, there were 11 females and nine males. In the intervention group, there were nine females and 11 males. Independent *t* tests were run to confirm that control

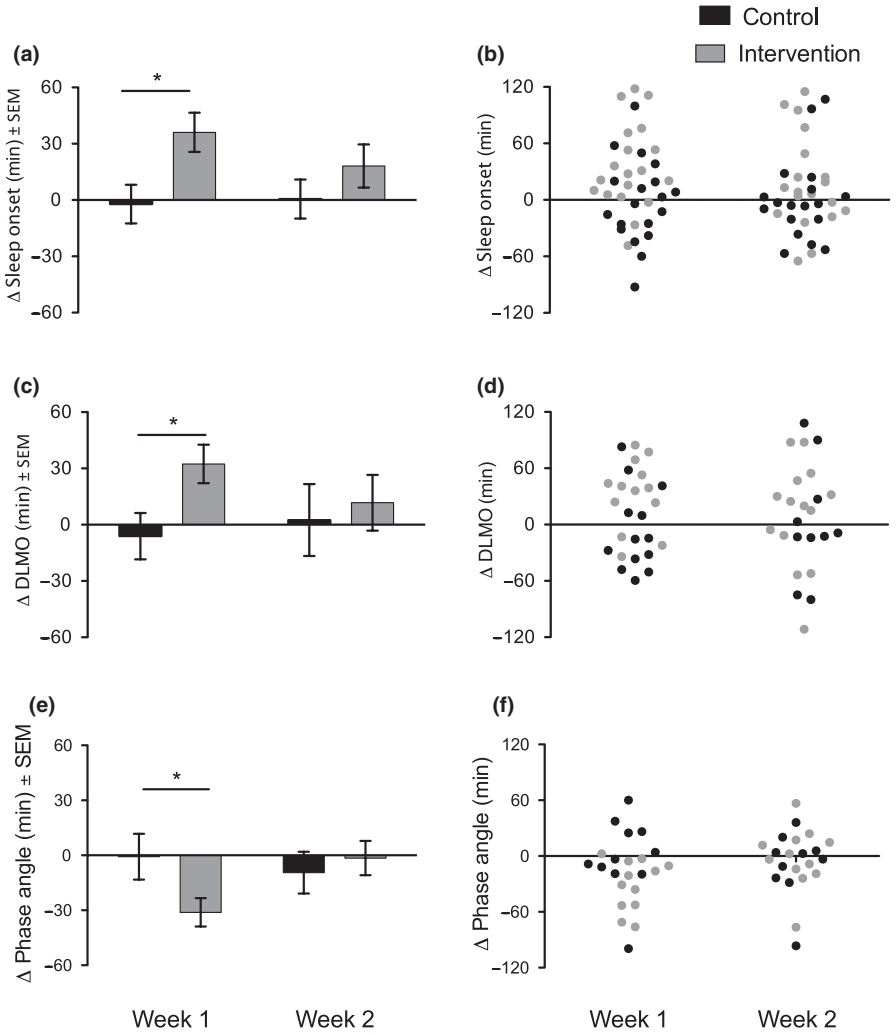

FIGURE 3 Blocking blue light late in the internal day advances both sleep onset on workdays (SO_w) and Dim-Light Melatonin Onset (DLMO), and reduces the phase angle difference between DLMO and SO_w. Control and intervention (blue-light-blocking glasses) group averages with standard error of the mean (*SEM*) (a–c–e) and individual data points (b–d–f) are plotted separately for the control (black) and the intervention (grey) groups. (a and b) The changes in SO_w expressed in minutes (Δ Sleep onset [min]) during week 1 and week 2 are plotted relative to baseline. Positive values represent phase advances and negative values phase delays. During week 1, the intervention group significantly advanced SO_w (on average by 36 ± 45 min) compared to the control group (*$p < 0.05$ with Bonferroni correction). (c and d) The changes in DLMO expressed in minutes (Δ DLMO [min]) during week 1 and 2 are plotted relative to baseline. Positive values represent phase advances and negative values phase delays. During week 1, the intervention group significantly advanced DLMO (on average by 32 ± 37 min) compared to the control group (*$p < 0.05$ with Bonferroni correction). (e and f) The changes in phase angle difference between DLMO and SO_w during week 1 and week 2 are plotted relative to baseline. Positive values represent an increase and negative values a decrease in phase angle difference between DLMO and SO_w. During week 1, the intervention group significantly decreased the phase angle difference between DLMO and SO_w (by 31 ± 27 min) compared to the control group (*$p < 0.05$ with Bonferroni correction)

and intervention groups did not differ at baseline in terms of age, chronotype and SJL (age: t (35) = −0.287, $p = 0.776$; chronotype: t (35) = −0.536, $p = 0.595$; SJL: t (35) = 0.389, $p = 0.699$).

We first compared the light intensities in the bedrooms during the intervention weeks (relative to baseline) between the two groups. Sleeping with open curtains significantly increased the morning light levels (first two hours after dawn) in the bedrooms of the intervention group compared to the control group ($U = 42$, $z = -3.787$, $p < 0.0001$). This increase was however mainly associated with changes in the bedroom light intensities of only four participants. The intervention did not negatively affect subjective sleep quality assessed with the PSQI ($U = 106.5$, $z = 0.420$, $p = 0.686$). The two groups did not, however, differ in terms of morning light exposure (assessed at the wrist between 6:00 and 12:00 hr) across the weeks ($F_{3,744} = 1.331$, $p = 0.263$; Supporting Information Figure S6). The exposure to light in the evening (assessed at the wrist between 18:00

TABLE 2 Demographic characteristics of 38 (20 females) participants in Study 2 randomly assigned to control and intervention groups

	Control group		Intervention group	
Parameter	**Average (*SD*)**	**Range**	**Average (*SD*)**	**Range**
Age	22.7 (3.5)	19–35	22.9 (2.7)	19–29
Chronotype (MSF_{sc}, hr:min)	5:22 (31′)	4:42–6:24	5:36 (42′)	4:42–7:48
Social Jetlag (hr:min)	1:40 (39′)	0:37–2:57	1:40 (39′)	0:42–2:55
Sleep onset on workdays (hr:min)	23:47 (37′)	22:34–0:42	0:15 (38′)	23:04–1:45
Sleep end on workdays (hr:min)	8:00 (51′)	7:00–10:00	8:10 (31′)	7:30–9:00
Sleep duration on workdays (hr:min)	8:12 (50′)	6:17–9:45	7:55 (40′)	7:09–9:55
Sleep onset on work-free days (hr:min)	1:13 (33′)	0:30–2:18	1:33 (52′)	0:04–4:00
Sleep end on work-free days (hr:min)	9:54 (49′)	9:00–12:00	10:14 (55′)	9:00–12:30
Sleep duration on work-free days (hr:min)	8:41 (49′)	7:19–10:30	8:41 (45′)	7:15–9:58

Note. The two groups did not significantly differ in relation to their demographic and sleep characteristics at baseline. Data concerning chronotype, sleep onset and sleep end refer to local clock time.

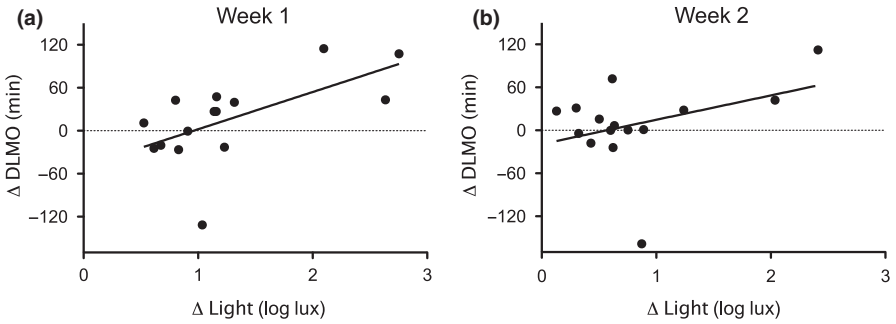

FIGURE 4 Correlation between individual changes in Dim-Light Melatonin Onset (DLMO) and bedroom light intensity. The correlation between the shift in DLMO and the change in morning bedroom light intensity (assessed with the HOBOs) during week 1 (a) and week 2 (b) relative to baseline is plotted for the intervention group only. There was a positive significant correlation (only during week 1, $p < 0.05$, $R^2 = 0.36$), indicating that the participants who experienced a greater increase in morning bedroom light intensity showed a greater advance in their DLMO

and 00:00 hr) also did not differ between the groups ($F_{3,748} = 0.781$, $p = 0.505$; Supporting Information Figure S6). Similarly, we did not find a change between groups in all the variables assessed with the sleep diaries and actigraphy (CoG_{act}) both on workdays and on work-free days. SJL and DLMO did also not vary between groups across the study (SJL: $F_{3,58} = 1.991$, $p = 0.125$; DLMO: $F_{3,53} = 0.856$, $p = 0.470$). However, the interaction effect between the change in bedroom light intensities and time on DLMO was significant ($F_{3,38} = 3.241$, $p = 0.033$). The shift in DLMO during week 1 (relative to baseline) correlated with the change in bedroom light intensity in the intervention group ($b = 0.866$, t (13) = 2.741, $p = 0.0168$, $R^2 = 0.37$; Figure 4a). In other words, the strength of the intervention (increase in morning bedroom light intensity) correlated positively with greater advances in the timing of DLMO in participants whose bedrooms received more morning light during the intervention. A similar correlation was found during the second intervention week, which was, however, not significant ($b = 0.567$, t (13) = 1.439, $p = 0.174$; Figure 4b). The weekly averages together with the standard deviations (*SD*) of the parameters assessed are reported in Supporting Information Table S2.

4 | DISCUSSION

We modified evening and morning light exposure in young adults to test whether these in-home protocols can shift phase of entrainment and thereby sleep timing. We wish to develop protocols to reduce SJL, a condition that has been associated with several health risks. We chose to use light interventions because light is the strongest zeitgeber for entrainment of human behaviour (Duffy & Wright, 2005; Roenneberg & Foster, 1997; Roenneberg & Merrow, 2007). In addition, light has been shown to have important direct effects on the regulation of brain areas that are associated with mood and learning (LeGates, Fernandez, & Hattar, 2014). Light interventions therefore have the potential to become the first choice treatment for clinical cases of comorbidity between circadian/sleep and psychiatric disorders.

One protocol involved wearing blue-light-blocking glasses at the end of the internal day (Study 1) and the second protocol involved sleeping with open curtains, thus increasing light at the beginning of the internal day (Study 2). While Study 1 was able to advance both phase of entrainment (estimated via DLMO, only on workdays) and sleep timing (only on workdays), Study 2 led to changes neither in DLMO nor in sleep timing. Notably, the phase advances achieved in Study 1 did not reduce SJL. There are two possible explanations for this. First, we only observed phase advances on workdays, which would even increase SJL, if weekend sleep times remained stable. The lack of a significant sleep-time advancing effect through our intervention on work-free days might in part result from a lack of statistical power: participants had less work-free days (on average 2 per week), and the variance of their sleep times on work-free days was higher (see Supporting Information Table S1). Second, SJL was relatively low at baseline in both studies (on average, <2 hr) and subjects slept on average 7.5 hr on workdays, which lies in the normal range for sleep duration in young adults (7–9 hr; Hirshkowitz et al., 2015), indicating limited room for improvement.

4.1 | Study 1—Decreasing blue light at the end of the internal day

Despite numerous publications describing the negative effects of evening (blue) light exposure on sleep timing and alertness (Chang, Aeschbach, Duffy, & Czeisler, 2015; Chellappa et al., 2013; Wahnschaffe et al., 2013; Wood, Rea, Plitnick, & Figueiro, 2013), few studies describe interventions to decrease evening light exposure. Similar protocols to those used here have been shown to reduce light-dependent melatonin suppression and subjective alertness before bedtime (Sasseville, Paquet, Sevigny, & Hebert, 2006; van der Lely et al., 2014) and—in clinical settings—to improve symptoms of bipolar disorder and insomnia (Phelps, 2008; Shechter, Kim, St-Onge, & Westwood, 2018). In line with our results, Esaki et al. (2016) found that DLMO and sleep onset advanced in patients with delayed sleep phase syndrome after 2 weeks of wearing blue-light-blocking glasses in the evening (Esaki et al., 2016). Another study found a similar advance by using blue-light-blocking glasses in conjunction with supplementary light in the morning, but also enforced earlier bedtimes (in contrast to our protocol that allowed subjects to freely choose when they retired to bed; Appleman et al., 2013). To the best of our knowledge, this is the first study showing that wearing blue-light-blocking glasses in the evening alone advances DLMO and sleep timing in healthy individuals. Since these glasses also decrease light intensity by 50%, the observed effects are probably a result of changes in both light spectrum composition and intensity.

Surprisingly, our blue-light suppressing intervention only led to advances in the first but not in the second week of intervention. This could be due to adaptation (e.g., via increased light sensitivity), which has been shown for continuously wearing orange contact lenses, based on increased melatonin suppression (Giménez, Beersma, Bollen, van der Linden, & Gordijn, 2014). However, this explanation seems unlikely because our participants only wore blue-blockers in the evening. It is important to note that what we observed at the group level (advance of DLMO and sleep timing during week 1 but not week 2) was true only for half of the participants (Supporting Information Figure S3). By looking at the individual light exposure we noticed that this was different in the participants who advanced and stabilized their DLMO during week 1 and 2 and in the participants who advanced their DLMO during week 1 but delayed it during week 2. The latter subjects appeared in fact to be exposed to more light during the hours preceding the intervention (wearing the blue-light-blocking glasses) in week 2 compared to week 1 and to baseline (Supporting Information Figure S4). If we also consider that the participants advanced their phase of entrainment during week 1, it is likely that they were exposed to more light at a sensitive phase of their biological evening, leading to a consequent delay in DLMO during week 2.

4.2 | Study 2—Increasing bedroom light at the beginning of the internal day

The light intervention of Study 2 (sleeping in a room with more morning light) changed neither DLMO nor sleep timing. The beneficial effects of morning light in relation to sleep and depressive disorders have been described (Rosenthal et al., 1990; Terman, Terman, Lo, & Cooper, 2001), but there is lack of data from field studies on the effects of morning light on the sleep-wake cycle in healthy individuals. In a controlled laboratory study, maximal melatonin phase shifts were obtained

with 500 lux (Zeitzer et al., 2000). However, in field studies, where participants are exposed to light during the day, stronger light intensities are probably necessary. A study was run during winter in the Antarctic, showing that an hour of morning bright light (maximal light exposure 4,775 lux) advanced both sleep and melatonin rhythms (Corbett, Middleton, & Arendt, 2012). Other studies investigating the effects of artificial dawn on sleep have not found advances in DLMO or sleep timing (Giménez et al., 2010; Tonetti et al., 2015). In these studies, the maximal light levels emitted by the lamps prior to awakening were 250 lux. In our study, the average light levels (recorded at the wrist level) during the 30 min prior to awakening (in participants who slept with open curtains) were 150 lux. Thus, the light intensities achieved in these studies (via natural dawn filtered through window glass or artificial dawn) seem insufficient to advance phase of entrainment. However, it should be noted that there were substantial inter-individual differences in the increase of bedroom morning light during the intervention. In particular, the bedroom light levels increased more than 100 times only in four participants who also showed the greatest advance in DLMO (Figure 4). It is therefore possible that different bedrooms (window size and orientation, distance between bed and window, etc.) produced a large variance in the strength of the intervention, leading to different shifts of the circadian phase. It should also be mentioned that retinal illumination (especially for short wavelengths) is greatly reduced (up to 97%) when the eye lids are closed (Kantermann & Roenneberg, 2009). This suggests that sleeping with open curtains would be an effective intervention only if people were awakened by the light and opened their eyes to be directly exposed to light.

5 | CONCLUDING REMARKS

In conclusion, based on the protocols used in these studies, decreasing exposure to blue light at the end of the internal day is more effective in advancing sleep timing and phase of entrainment (DLMO) than increasing exposure to light through windows (i.e., no supplemental artificial light sources) at the beginning of the internal day. Future studies should investigate individual differences (i.e., age, sex, health (e.g., cataract), chronotype, lifestyle factors) in response to light at different times of day. Epidemiological studies based on the MCTQ (Roenneberg et al., 2015) suggest that young adults are not as sensitive to changing chronotype through light exposure, thus these interventions may be best suited for a particular stage of life. Further, due to inherent differences in chronotype and the genetic bases therein, we suggest that for some individuals a light intervention in the morning could be more effective than in the evening or vice versa. Future experiments should also investigate more long-term effects of such interventions to explore how the circadian system adapts to new light regimes and whether late chronotypes could find a stable, earlier phase of entrainment. Similar studies should be also repeated in participants suffering from extreme SJL to assess whether SJL can be decreased with light and whether this would lead to better health outcomes.

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CONFLICT OF INTEREST

The authors declare no competing interests.

DATA ACCESSIBILITY

Data analyzed in this study is available from the corresponding authors on request.

AUTHOR CONTRIBUTIONS

Both studies were designed by G.Z., T.K., and M.M. G.Z. collected and analyzed the data and prepared the figures under the supervision of T.K. and M.M. G.Z. wrote the manuscript under the supervision of M.M. and both T.K. and M.M. reviewed the manuscript.

ORCID

Giulia Zerbini https://orcid.org/0000-0002-5348-9212

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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