

# Resetting the late timing of 'night owls' has a positive impact on mental health and performance

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DOI:

[10.1016/j.sleep.2019.05.001](https://doi.org/10.1016/j.sleep.2019.05.001)

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*Document Version*

Peer reviewed version

*Citation for published version (Harvard):*

Facer-Childs, E, Middleton, B, Skene, D & Bagshaw, A 2019, 'Resetting the late timing of 'night owls' has a positive impact on mental health and performance', *Sleep Medicine*, vol. 60, pp. 236-247.  
<https://doi.org/10.1016/j.sleep.2019.05.001>

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1 **Title: Resetting the late timing of ‘night owls’ has a positive impact on**  
2 **mental health and performance**

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27 **Highlights:**

28 Here we took a group of ‘night owls’ (i.e. people with extreme late sleeping and waking patterns) and  
29 attempted to shift their habitual late timings earlier in a real-world setting using simple, practical non-  
30 pharmacological interventions. We show that by using this intervention we can:

- 31
- Achieve a phase advance of around two hours
  - Decrease self-reported ratings of depression and stress
  - Reduce sleepiness in the morning
  - Significantly improve simple indices of cognitive and physical performance
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38 **Abstract**

39 There is conflict between living according to our endogenous biological rhythms and our external  
40 environment, with disruptions resulting in negative consequences to health and performance. This is  
41 often documented in shift work and jet lag, but ‘societal norms’ e.g. typical working hours, can create  
42 profound issues for ‘night owls’, people whose internal biological timing predisposes them to follow  
43 an unusually late sleep-wake cycle. Night owls have also been associated with health issues, mood  
44 disturbances, poorer performance and increased mortality rates. This study used a randomized control  
45 trial design aimed to shift the late timing of night owls to an earlier time (phase advance), using non-  
46 pharmacological, practical interventions in a real-world setting. These interventions targeted light  
47 exposure (through earlier wake up/sleep times), fixed meals times, caffeine intake and exercise.  
48 Overall, participants demonstrated a significant advance of ~2 h in sleep/wake timings as measured by  
49 actigraphy and circadian phase markers (dim light melatonin onset and peak time of the cortisol  
50 awakening response), whilst having no adverse effect on sleep duration. Importantly, the phase  
51 advance was accompanied by significant improvements to self-reported depression and stress, as well  
52 as improved cognitive (reaction time) and physical (grip strength) performance measures during the  
53 typical ‘suboptimal’ morning hours. Our findings propose a novel strategy for shifting clock timing  
54 towards a pattern that is more aligned to societal demands that could significantly improve elements  
55 of performance, mental health and sleep timing in the real world.

56

57 **Keywords:** Late circadian phenotypes; chronotype; actigraphy; dim light melatonin onset; cortisol  
58 awakening response; non-pharmacological interventions; phase advancing; depression; stress;  
59 performance

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## 64 **Introduction**

65 There is often little regard for the impact of sleep and circadian disruptions in society's attitude  
66 towards the organisation of our typical working day. Disturbances to the sleep/wake system that  
67 impair daily functioning leading to reduced health are prevalent, with around two thirds of the UK's  
68 adult population (67%) reporting some sort of sleep issue [1, 2]. It is well documented that restricted  
69 sleep and disrupted circadian rhythmicity result in changes to many physiological processes such as  
70 endocrine regulation [3] and core body temperature (CBT) [4], as well as being linked with a variety  
71 of health issues, including mood disturbances [5], increased morbidity and mortality rates [6], and  
72 declines in cognitive and physical performance [7]. Disruption to circadian and sleep/wake processing  
73 represents a substantial economic burden on society, primarily through loss of productivity,  
74 absenteeism and poor performance [8], and increases the risk of occupational accidents [9]. A major  
75 factor influencing these outcomes is a lack of appreciation for individual differences in vulnerability  
76 to sleep disruption and circadian misalignment, and a lack of awareness of the extent to which an  
77 individual's circadian timing may not align with the normal 09:00 h - 17:00 h working day.

78 Individual differences in biological rhythms are influenced by physiological [10, 11], genetic [12] and  
79 behavioural [13] factors. These differences allow the categorisation of individuals according to their  
80 circadian timing, with particularly early and late timings often referred to as 'larks' and 'night owls'  
81 (termed Early and Late circadian phenotypes, ECP/LCP, in this study). At their most extreme these  
82 differences can result in clinical diagnoses of the circadian rhythm sleep-wake disorders (CRSWDs),  
83 Advanced Sleep-Wake Phase Disorder (ASWPD) and Delayed Sleep-Wake Phase Disorder  
84 (DSWPD), which are more prevalent in older and younger subjects, respectively. The extent to which  
85 these clinical disorders overlap in terms of mechanisms with extreme circadian phenotypes in the  
86 healthy population remains unknown.

87 DSWPD is often associated with mood disorders such as depression [14], and this group of  
88 individuals also tend to be restricted by social factors such as work/school routines which shorten  
89 sleep resulting in an accumulation of 'sleep debt'. This causes excessive sleepiness during the day and  
90 impairment of cognitive functioning [15]. While clinical assessment is needed to diagnose DSPWD,

91 many of its symptoms are shared with ‘night owls’ (LCPs). LCPs are categorized based on late  
92 sleep/wake timings, a delay in dim light melatonin onset (DLMO) and/or defective sleep homeostasis  
93 [16]. LCPs have been associated with higher scores for depression [17], decreased morning cognitive  
94 performance, excessive daytime sleepiness [18], as well increased morbidity and mortality risks [6].  
95 Diurnal variations in both cognitive and physical performance measures have also been shown to vary  
96 between circadian phenotypes [19], with LCPs often having difficulties fitting into traditional working  
97 hours. Since around 50% of a given population would fall into a ‘Late type’ category (waking after  
98 8:18 h) [20], one could propose that these individuals are compromised by having delayed circadian  
99 timing and could benefit by being shifted towards an earlier pattern.

100 Resetting biological clocks can be achieved using behavioural methods, pharmacological methods or  
101 a combination of the two. The human circadian system is most responsive to light, which allows  
102 sleep/wake activity and physiology to adapt to the 24 h light dark cycle. As a result, light, or lack of  
103 light, is a major target to try and reset biological clocks through a process called photic entrainment.  
104 Bright light has been shown to shift circadian phase depending on time and duration of light  
105 administered (phase response curve) [21, 22]. Exposure in the early morning phase advances the  
106 circadian system causing DLMO to peak earlier and sleep onset to become advanced [23].  
107 Conversely, light exposure during the biological night creates a phase delay shown by a later DLMO  
108 [24, 25].

109 Non photic forms of entrainment have also been researched to try and shift circadian phase [26].  
110 These behavioural targets i.e. non-pharmacological interventions, include altering sleep/wake cycles  
111 [27], timed physical exercise [28] and timed feeding [29]. Timed feeding has been shown to shift  
112 peripheral clocks in mice without affecting the SCN clock [30]. Furthermore, timed feeding has been  
113 shown to regulate peripheral metabolic rhythms with a 5-hour delay in meal timings delaying rhythms  
114 of plasma glucose and adipose PER2 clock gene expression [29]. An alternative circadian zeitgeber  
115 that has been explored is targeted physical exercise. Timed exercise can alter the rhythm of core body  
116 temperature [31] and melatonin [32]. A recent paper has further supported these findings, showing

117 that exercise in the morning and early afternoon elicits a phase advance, whereas scheduled evening  
118 exercise causes a phase delay [33].

119 The majority of our society has stringent work and schooling hours requiring attendance between the  
120 hours of 09:00 h and 17:00 h. Despite these traditional imposed social clock requirements, there has  
121 been some shift towards understanding biological constraints by allowing flexibility of working hours  
122 [34], as well as attempts to move school start times to fit to adolescents' notoriously late running  
123 biological clocks [35]. However, despite awareness of the consequences, there is still a long way to go  
124 to directly translate research outcomes and affect change in our rapidly evolving 'round the clock'  
125 society.

126 Although attempting a phase advance (shifting the clock earlier) using some of these methods has  
127 previously been shown in laboratory studies [28, 36, 37], field studies are lacking. Furthermore,  
128 investigating the impact on mental health and diurnal variations in performance have not yet been  
129 attempted in real world settings. Here we propose a novel intervention strategy for 'night owls'  
130 (LCPs), many of whom suffer from chronic circadian misalignment or disrupted sleep homeostasis.  
131 Using simple, practical lifestyle changes, we aimed to phase advance sleep/wake timings, DLMO and  
132 time of peak cortisol awakening response. We hypothesised that if a phase advance is achieved this  
133 would improve self-rated measures of mental health (depression, anxiety and stress) as well as shift  
134 the timing of peak performance earlier, and thus improve simple indices of cognitive (reaction time)  
135 and physical (grip strength) performance at non-optimum times of day.

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142 **Methods**

143 **Participants**

144 The study received a favourable ethical opinion from the University of Birmingham Research Ethics  
145 Committee, was performed in accordance with the Declaration of Helsinki and participants gave  
146 written informed consent before involvement. A total of 178 individuals completed the Munich  
147 ChronoType Questionnaire (MCTQ, paper version [38]) to calculate corrected mid-sleep on free days  
148 ( $MSF_{sc}$ ). Participants classified as Late chronotypes using an age and gender matched MCTQ  
149 database were invited to take part in the study ( $n = 49$ ). Individuals were screened for no diagnoses of  
150 sleep or neurological disorders via self-report and were not taking any medications that affected sleep,  
151 melatonin and cortisol rhythms. A total of 29 individuals agreed to take part in the study, of which  
152 five were excluded based on medical history and two dropped out prior to starting the study.

153 The final sample consisted of 22 healthy individuals (15 female, aged  $21.3 \pm 3.3$  years,  $MSF_{sc}$   $06:52 \pm$   
154  $00:17$  h). The study used a randomized control trial design and was conducted over six weeks for each  
155 participant which took place between April and June 2016 (sunrise range  $06:42$  h to  $04:40$  h, sunset  
156 range  $19:41$  h to  $21:32$  h, latitude  $52^\circ 29' 22.0956''$  N). Participants were randomly assigned to the  
157 experimental ( $n = 12$ , 9 female) or control ( $n = 10$ , 6 female) groups at the start of the study. Two  
158 weeks of acclimatisation was used to assess habitual sleep patterns using actigraphy and gather  
159 questionnaire data at baseline (pre-intervention). Following this period, participants were asked to  
160 provide saliva samples for melatonin and cortisol in their home environment (details below) before  
161 attending the laboratory for testing sessions at  $14:00$  h,  $20:00$  h and  $08:00$  h. To simulate a ‘real  
162 world’ setting, participants were able to leave the laboratory between testing sessions. Participants  
163 were then given a schedule to follow for the next three weeks (intervention) before returning to repeat  
164 all testing sessions, physiological sampling and questionnaires (Figure 1). Participants completed the  
165 test sessions on the same day pre- and post-intervention. Summary details of participants’ data pre-  
166 intervention for experimental and control groups to confirm accurate matching can be found in  
167 Supplemental Table 1.

168

169 **Non-Pharmacological Interventions**

170 At the final pre-intervention testing session, the experimental group were given an intervention  
171 schedule to follow for a period of three weeks. These interventions followed standard sleep hygiene  
172 suggestions and targeted appropriately timed light exposure, sleep, meals, caffeine and exercise  
173 (summarised in Table 1). The control group were given a placebo single instruction to ‘eat lunch at  
174 the same time every day’ with the assumption that there would be no differences in sleep timings and  
175 hence no effect on circadian phase. Adherence to the intervention was monitored through self-report.  
176 Meal timings pre- and post-intervention were collected as part of a diet questionnaire which enquired  
177 about food intake habits over the prior 2 weeks. Timing of naps were monitored through daily sleep  
178 diaries. A feedback questionnaire was administered at the end of the study where participants were  
179 asked whether they adhered to the intervention schedule on a scale of 0 (not at all) to 10 (completely).  
180 At each testing session participants answered an online questionnaire to record timing of external  
181 variables prior to/between sessions such as caffeine intake, exercise and meal times.

182

183 **\*\*\*INSERT TABLE 1\*\*\***

184 **Table 1.** Details of intervention schedule given to participants in the experimental group. The control  
185 group were given a single instruction (shown in **bold**). Method of monitoring adherence (in addition to a  
186 feedback questionnaire administered post-intervention) is given for each intervention target.  
187

<b>Intervention target</b>	<b>Instructions given</b>	<b>How adherence was monitored</b>
Wake up time	Participants were asked to try and wake up 2-3 hours before habitual wake up time. Participants were asked to maximise outdoor light exposure during the mornings.	Continuous monitoring pre- and post-intervention through actigraphy and sleep diaries.
Sleep/wake timings	Participants were asked to try and keep sleep/wake times fixed (within 15/30mins) between workdays and free days.	Continuous monitoring pre- and post-intervention through actigraphy and sleep diaries.

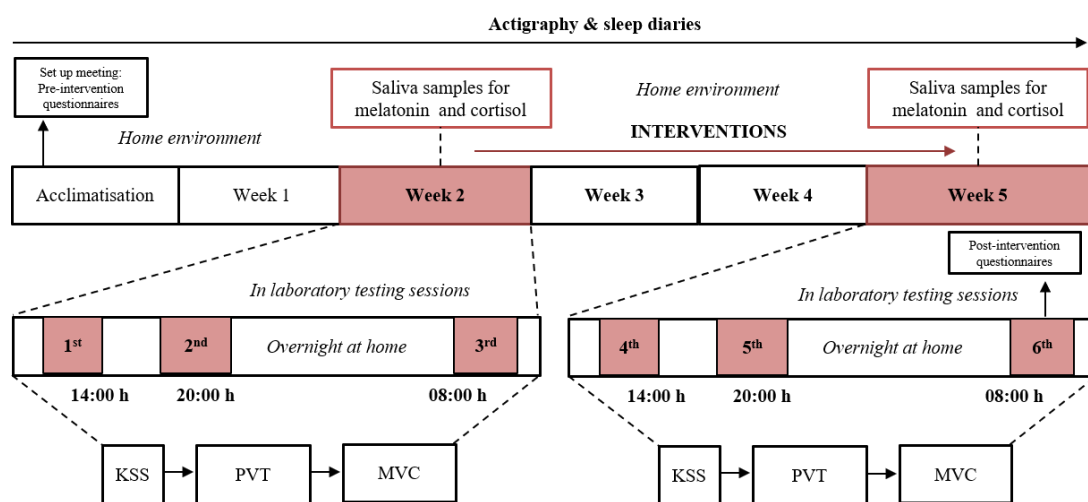


Sleep onset	Participants were asked to try and go to sleep 2-3 hours before habitual bedtime. Participants were asked to limit light exposure during the evenings.	Continuous monitoring pre- and post-intervention through actigraphy and sleep diaries.
Diet/nutrition	Participants were asked to keep a regular schedule for daily meals. Participants were asked to have breakfast as soon after wake up as possible. <b>Participants were asked to eat lunch at the same time every day.</b> Participants were asked not to have dinner after 19:00 h.	A diet questionnaire was administered pre- and post-intervention. An online questionnaire was completed at all testing sessions to record time since last meal.
Caffeine intake	Participants were asked not to drink any caffeine after 15:00 h.	An online questionnaire was completed at all testing sessions to record time since caffeine intake.
Power naps	Participants were asked not to nap after 16:00 h.	Napping was recorded through self-reported daily sleep diaries.
Exercise	If exercise was part of an individual's usual routine they were asked to schedule this during the morning.	An online questionnaire was completed at all testing sessions to record time since exercise.

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203 **Figure 1. Schematic illustration of experimental protocol.** Actigraphy combined with sleep diaries were  
204 completed for the duration of the study as well as physiological sampling for melatonin and cortisol  
205 measurements prior to attending testing sessions during weeks 2 and 5 (pre- and post-intervention). At each  
206 testing session participants completed cognitive (psychomotor vigilance task, PVT) and physical (maximum  
207 voluntary contraction, MVC, of isometric grip strength) performance testing coupled with subjective sleepiness  
208 ratings using the Karolinska Sleepiness Scale (KSS).  
209

210 **Physiological Data**

211 All participants underwent training in how to collect saliva samples in their home environment  
212 following strict protocols. During the sampling period, participants were asked to refrain from  
213 cleaning their teeth, drinking caffeinated drinks, alcoholic drinks or any drinks that contained artificial  
214 colouring. Each individual was provided with a sample record collection form in order to report the  
215 exact times that samples were given and report any factors that could have affected the sampling  
216 period e.g. exposure to light, disruption to sampling. Participants provided saliva samples during one  
217 morning and one evening during pre-intervention (week 2) and post-intervention (week 5). Samples  
218 for melatonin were collected whilst seated in dim lighting conditions i.e. no overhead lights, no  
219 electronic devices and curtains closed, every 30 minutes from between three and four hours prior to  
220 habitual bedtime until one hour after habitual bedtime. Morning samples for cortisol were collected  
221 over a period of 3 hours from wake-up time (the first five samples every 15 minutes and the  
222 remaining four samples every 30 minutes). Exact sampling times for each individual were recorded.

223 Radioimmunoassays (RIA) of melatonin and cortisol in human saliva were performed (Stockgrand  
224 Ltd, University of Surrey) using an Iodine<sup>125</sup> radioactive labelled tracer and solid phase separation  
225 [39]. Individual DLMOs were calculated with a linear response function using the mean of the  
226 individual pre-intervention concentration values plus two standard deviations of the mean. The time  
227 of highest cortisol concentration recorded during the sampling period was used as an indicator of peak  
228 cortisol awakening response. Due to insufficient or contaminated samples paired DLMO values (pre-  
229 and post-intervention) could not be computed for three subjects in the experimental group and five  
230 subjects in the control group.

231  
232 **Behavioural Data**

233 *Sleep Analysis:* Actigraphs (Actiwatch® Light, 2006, Cambridge Neurotechnology Ltd), combined  
234 with daily sleep diaries, were worn on the non-dominant wrist for the entire duration of the study  
235 (weeks 1-5) to monitor actigraphic sleep and rest-activity patterns (1-minute epochs) in the home  
236 environment and analysed with the manufacturer's software (Sleep Analysis 7.23, Cambridge

237 Neurotechnology Ltd). Due to incorrect wearing of the devices, actigraphic data from two individuals  
238 (one in the experimental group and one in the control group) were not usable.

239 **Questionnaires:** A set of questionnaires were completed by each participant during a set up meeting  
240 pre-intervention and repeated at the end of the final testing session (post-intervention). Questionnaires  
241 included the MCTQ, paper version [38], Epworth Sleepiness Scale (ESS) [40], Pittsburgh Sleep  
242 Quality Index (PSQI) [41], Profile of Mood States (POMS) [42], Depression, Anxiety and Stress  
243 Scale (DASS) [43], and a Diet Questionnaire [29]. Due to insufficient completion of questionnaires,  
244 three individuals' results were not recorded for POMS, two for DASS and two for the Diet  
245 Questionnaire.

246 **Sleepiness:** Daytime subjective sleepiness, measured using the Karolinska Sleepiness Scale (KSS)  
247 [44], was assessed at each testing session before the cognitive and physical tasks were performed.

248 **Reaction time:** Cognitive testing consisted of a two-minute visual psychomotor vigilance task (PVT)  
249 [45]. The PVT was conducted on a desktop computer (DQ67OW, Intel® Core™ i7-2600 processor,  
250 4GB RAM, 32-bit Windows 7) with a standard keyboard and mouse. The same set up was used  
251 throughout the study for each participant and each testing session. Participants also performed three  
252 trial tests during the acclimatisation phase to familiarise themselves with the set up and minimise  
253 learning effects. Milliseconds were recorded for each trial, then a mean response time was taken over  
254 the number of trials.

255 **Grip strength:** To obtain a simple measure of physical performance an electronic hand dynamometer  
256 (EH101, CAMRY) was used to perform a six second maximum voluntary contraction (MVC) test of  
257 isometric grip strength [46]. Participants stood with the elbow extended at 180° and used their  
258 dominant hand in a pronated position to apply as much grip pressure as possible. Raw scores were  
259 recorded in kg. Three trials were completed with two minutes rest between each trial and the highest  
260 recorded value was used in the subsequent analysis. A set script was used to motivate the participants  
261 due to the influence of motivation on performance [47].

262

## 263 **Statistical Analysis**

264 Statistical comparisons were performed in GraphPad Prism (version 7.00), using linear regression  
265 analysis and two-way repeated measures ANOVA with post hoc tests corrected for multiple  
266 comparisons, adding intervention group (experimental/control), assessment period (pre- vs. post-  
267 intervention) or time of day (08:00 h, 14:00 h and 20:00 h) as factors. Diurnal variations in  
268 performance and sleepiness variables were plotted using second degree polynomial regression curves.  
269 Due to data collection occurring at 14:00 h through to 08:00 h, the model is constrained to this time  
270 period.

271 The raw scores for the performance measurements (reaction time in milliseconds from the PVT and  
272 grip strength in kilograms from the MVC test) were normalised by converting to percentages relative  
273 to each individual's time of peak performance. For example, the testing session where fastest reaction  
274 time and strongest grip strength was recorded was designated as 100% for that participant. The  
275 subsequent scores were calculated relative to this. Higher percentages always relate to better  
276 performance achieved (faster reaction time and stronger grip strength). This was to allow diurnal  
277 variations to be quantified in a standardised way across individuals and across different measures of  
278 performance (Facer-Childs et al. 2018). These data were normalised relative to each individual in the  
279 pre- and post-intervention conditions separately. Test statistics are given to one significant figure.  
280 Significance levels are displayed as ns = not significant,  $p < 0.05 = *$ ,  $p < 0.01 = **$ ,  $p < 0.001 = ***$   
281 and  $p < 0.0001 = ****$ . Values are represented as the mean  $\pm$  standard error of the mean (SEM) unless  
282 specified otherwise (age and BMI values are given with standard deviations). Exact p values are given  
283 to two significant figures, apart from when significance is identified as less than 0.0001, in which case  
284  $p < 0.0001$  is reported. The 08:00 h test is described as morning, 14:00 h as afternoon and 20:00 h as  
285 evening. Reaction time (measured using the PVT) will be referred to as a simple index of attentional  
286 cognitive performance and isometric grip strength (measured using an MVC test) as a simple index of  
287 physical performance.

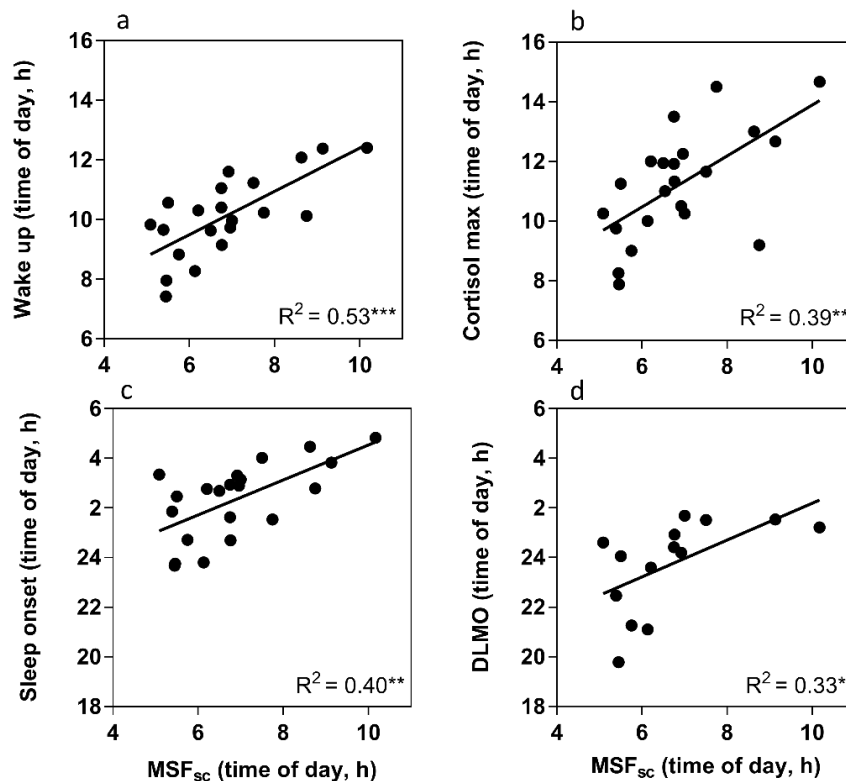
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289 **Results**

290 To confirm that the study groups were evenly matched according to the range of variables discussed  
291 below, all data were initially compared pre-intervention and no significant differences were found in  
292 any of the parameters measured (Supplemental Table 1). Experimental and control groups were of  
293 similar age ( $21.7 \pm 2.8$  and  $20.9 \pm 3.9$  years), BMI ( $22.9 \pm 3.2$  and  $22.6 \pm 2.1$ ) and  $MSF_{sc}$  ( $07:15 \text{ h} \pm$   
294  $00:27$  and  $06:02 \text{ h} \pm 00:14$ ). At baseline (pre-intervention) significant linear relationships were  
295 observed between  $MSF_{sc}$  and wake up time ( $R^2 = 0.53$ ,  $F = 21.21$ ,  $p = 0.0002$ ), sleep onset ( $R^2 = 0.40$ ,  
296  $F = 12.69$ ,  $p = 0.0021$ ), peak time of the cortisol awakening response ( $R^2 = 0.39$ ,  $F = 12.65$ ,  $p =$   
297  $0.002$ ), and DLMO ( $R^2 = 0.33$ ,  $F = 5.98$ ,  $p = 0.03$ ) (Figure 2). These results support and validate the  
298 classification of participants as LCPs through actigraphic analyses and biological phase markers  
299 following the original identification as Late chronotypes from the MCTQ.

300 **\*\*\*INSERT FIGURE 2\*\*\***

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**Figure 2. Linear relationships between pre-intervention corrected  $MSF_{sc}$  and biological phase markers to validate circadian phenotyping.** a) Wake up time (h), b) Time of peak cortisol awakening response (h), c) Sleep onset (h), d) Dim light melatonin onset (DLMO) (h). Corrected mid-sleep on free days ( $MSF_{sc}$ ) is displayed as time of day (h) on the x axis. Statistical analysis was carried out using linear regression analysis. Asterisks represent significant relationships (\* =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ ) and  $R^2$  value is shown in the bottom right corner.

### 330 **Adherence to Interventions**

331 Overall, the experimental group reported on average  $7.8 \pm 0.7$  adherence to the interventions in the  
332 feedback questionnaire. Adherence to interventions targeting sleep/wake and dietary variables  
333 (monitored through actigraphy and a diet questionnaires) were confirmed with an advance in timings  
334 (see below). Avoidance of naps after 16:00 h was confirmed using self-reported sleep diaries. Results  
335 from the online questionnaire at the evening testing session confirm an advance in self-reported  
336 timing of caffeine intake, exercise and last meal for the experimental group post-intervention.  
337 Average self-reported caffeine intake before the 20:00 h testing session was on average 4 hours earlier  
338 post-intervention in the experimental group ( $5.9 \pm 1.7$  h pre-intervention and  $10.3 \pm 1.5$  h post-  
339 intervention), meaning this advanced from 14:00 h to 10:00 h. Average self-reported hours since  
340 exercise advanced from  $6.8 \pm 1.7$  h before the evening test session pre-intervention to  $7.8 \pm 1.8$  h  
341 before the evening test session post-intervention, as did hours since last meal from  $2.4 \pm 0.4$  h pre-  
342 intervention to  $3.8 \pm 0.8$  hrs post intervention. By contrast, the control group had a slight delay in  
343 timings of exercise and meal time relative to pre-intervention ( $6.0 \pm 1.9$  h to  $4.8 \pm 1.8$  h and  $3.5 \pm 1.0$   
344 h to  $2.8 \pm 0.8$  h respectively) and a slight advance in hours in caffeine from  $6.1 \pm 2.3$  h to  $8.7 \pm 2.6$  h.

345

### 346 **Phase Advance**

347 Compared to pre-intervention, a clear phase advance of around 2 h was observed post-intervention in  
348 the experimental group, as measured by the MCTQ, actigraphy and circadian phase markers (Figure 3  
349 and Table 2).  $MSF_{sc}$  was shifted significantly earlier by  $2.57 \pm 0.32$  h ( $p < 0.0001$ ). This advance was  
350 confirmed with actigraphic analysis showing a significant advance of  $1.73 \pm 0.28$  h for sleep onset  
351 and  $1.92 \pm 0.26$  h for wake-up time (both  $p < 0.0001$ ), with no significant changes in sleep duration,  
352 sleep efficiency or sleep latency. DLMO was advanced by  $1.96 \pm 0.63$  h ( $p = 0.018$ ), and time of peak  
353 cortisol awakening response by  $2.22 \pm 0.50$  h ( $p = 0.0005$ ). There were no significant changes in  
354 phase angle (time between sleep onset and DLMO). Average self-reported breakfast time in the  
355 experimental group shifted significantly earlier by  $1.11 \pm 0.39$  h compared to pre-intervention ( $p =$

356 0.022). Similarly, average self-reported lunch and dinner times also advanced by  $0.75 \pm 0.27$  h (lunch,  
357  $p = 0.023$ ) and  $1.44 \pm 0.49$  h (dinner,  $p = 0.021$ ). In the control group there was a significant delay of  
358  $1.16 \pm 0.34$  h in sleep onset ( $p = 0.0067$ ) and  $1.24 \pm 0.32$  h in wake-up time ( $p = 0.0021$ ) compared to  
359 pre-intervention. By contrast to the experimental group, no other variables were significantly different  
360 following the control intervention.

361

### 362 **Impact of Interventions on Mental Well-Being**

363 Subjective ratings of depression and stress significantly decreased following the interventions in the  
364 experimental group (Figure 4 and Table 2). Overall DASS score decreased by  $8.7 \pm 2.4$  points from  
365 19.8 to 11.2 (pre-intervention). Splitting DASS into depression, anxiety and stress scores separately  
366 revealed a significant effect of intervention ( $F(1,11) = 13.28$ ,  $p = 0.0039$ ), and significant decreases in  
367 the depression and stress elements but not anxiety ( $p = 0.37$ ). Depression was reduced from  $5.5 \pm 1.0$   
368 to  $2.3 \pm 1.2$  ( $p = 0.025$ ), and stress from  $9.5 \pm 2.2$  to  $5.7 \pm 1.9$  ( $p = 0.0061$ ). There were no significant  
369 differences found for the control group in any parameters measured. In both study groups no  
370 significant differences were observed for POMS, PSQI or ESS (Figure 4 and Table 2).

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381 **Table 2.** Summary of main variables and statistical analysis for the experimental group and control  
 382 group pre- and post-intervention.<sup>1</sup>  
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Variable measured	Experimental group		Control group		Interaction (intervention group and assessment period)	Main Effect of Intervention Group (experimental vs. control)	Main Effect of Assessment Period (pre- vs post-intervention)
	Pre- intervention	Post- intervention	Pre- intervention	Post- intervention			
MCTQ Score (hh:mm)	07:15 ± 00:27	04:40 ± 00:15	06:02 ± 00:14	07:10 ± 00:18	F (1,20) = 50.8****	F (1, 20) = 3.9 <sup>ns</sup>	F (1, 20) = 14.8**
<b>Nutrition related variables</b>							
Average days per week eating breakfast (days)	4.1 ± 0.6	5.4 ± 0.5	4.7 ± 0.8	4.4 ± 0.8	F (1, 19) = 4.1 <sup>ns</sup>	F (1, 19) = 0.04 <sup>ns</sup>	F (1, 19) = 1.6 <sup>ns</sup>
Average breakfast time (hh:mm)	10:33 ± 00:25	09:24 ± 00:24	10:01 ± 00:34	10:41 ± 00:20	F (1, 18) = 9.2**	F (1, 18) = 0.4 <sup>ns</sup>	F (1, 18) = 0.6 <sup>ns</sup>
Average lunch time (hh:mm)	14:36 ± 00:30	13:51 ± 00:27	13:27 ± 00:17	13:39 ± 00:19	F (1, 18) = 6.4*	F (1, 18) = 1.6 <sup>ns</sup>	F (1, 18) = 2.1 <sup>ns</sup>
Average dinner time (hh:mm)	20:07 ± 00:45	18:41 ± 00:14	18:49 ± 00:17	19:06 ± 00:20	F (1, 15) = 6.5*	F (1, 15) = 0.7 <sup>ns</sup>	F (1, 15) = 3.0 <sup>ns</sup>
<b>Mental well-being variables</b>							
Pittsburgh Sleep Quality Index (PSQI)	4.8 ± 0.7	4.2 ± 0.7	5.3 ± 0.8	4.8 ± 0.5	F (1, 20) = 0.02 <sup>ns</sup>	F (1, 20) = 0.4 <sup>ns</sup>	F (1, 20) = 1.1 <sup>ns</sup>
Profile of Mood States (POMS)	10.3 ± 6.2	-2.9 ± 4.5	8.5 ± 5.7	7.1 ± 6.1	F (1, 17) = 2.2 <sup>ns</sup>	F (1, 17) = 0.3 <sup>ns</sup>	F (1, 17) = 3.4 <sup>ns</sup>
Epworth Sleepiness Scale (ESS)	7.1 ± 1.2	6.3 ± 1.1	9.0 ± 1.0	8.7 ± 0.7	F (1, 20) = 0.2 <sup>ns</sup>	F (1, 20) = 2.8 <sup>ns</sup>	F (1, 20) = 0.7 <sup>ns</sup>
Depression Anxiety and Stress Scale (DASS)	19.8 ± 3.4	11.2 ± 3.1	13.8 ± 3.7	13.6 ± 5.0	F (1, 18) = 2.6 <sup>ns</sup>	F (1, 18) = 2.0 <sup>ns</sup>	F (1, 18) = 5.2*
<b>Actigraphy variables</b>							
Sleep Onset (hh:mm)	02:46 ± 00:26	01:03 ± 00:18	01:37 ± 00:30	02:47 ± 00:27	F (1, 18) = 42.7****	F (1, 18) = 0.3 <sup>ns</sup>	F (1, 18) = 1.7 <sup>ns</sup>
Wake Up Time (hh:mm)	10:31 ± 00:23	08:36 ± 00:15	09:37 ± 00:29	10:51 ± 00:29	F (1, 18) = 59.6****	F (1, 18) = 1.7 <sup>ns</sup>	F (1, 18) = 2.8 <sup>ns</sup>
Sleep Duration (h)	7.75 ± 0.20	7.55 ± 0.20	7.8 ± 0.2	7.9 ± 0.1	F (1, 18) = 0.9 <sup>ns</sup>	F (1, 18) = 0.6 <sup>ns</sup>	F (1, 18) = 0.2 <sup>ns</sup>
Sleep Efficiency (%)	76.80 ± 1.48	75.40 ± 1.25	78.3 ± 1.9	77.2 ± 1.5	F (1, 18) = 0.03 <sup>ns</sup>	F (1, 18) = 0.7 <sup>ns</sup>	F (1, 18) = 1.8 <sup>ns</sup>
Sleep Latency (hh:mm)	00:27 ± 00:04	00:28 ± 00:02	00:21 ± 00:03	00:22 ± 00:03	F (1, 18) = 0.07 <sup>ns</sup>	F (1, 18) = 2.0 <sup>ns</sup>	F (1, 18) = 0.2 <sup>ns</sup>
<b>Physiological variables</b>							
Dim Light Melatonin Onset (hh:mm)	00:02 ± 00:37	22:04 ± 00:21	23:18 ± 00:54	22:54 ± 00:45	F (1, 12) = 2.2 <sup>ns</sup>	F (1, 12) = 0.005 <sup>ns</sup>	F (1, 12) = 5.0*
Cortisol Peak Time (hh:mm)	11:19 ± 00:31	09:06 ± 00:19	11:05 ± 00:36	11:19 ± 00:32	F (1, 20) = 11.1**	F (1, 20) = 2.7 <sup>ns</sup>	F (1, 20) = 7.2*

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<sup>1</sup> Statistical analysis was done with two-way repeated measures ANOVA with Sidak's post hoc tests corrected for multiple comparisons. Intervention group (experimental/control) and assessment period (pre- and post-intervention) are used as factors in the statistical analysis. Ns = not significant, \* = p < 0.05, \*\* = p < 0.01, \*\*\*\* = p < 0.0001. Values are shown as mean ± SEM unless specified.



386 \*\*\*INSERT FIGURE 3\*\*\*

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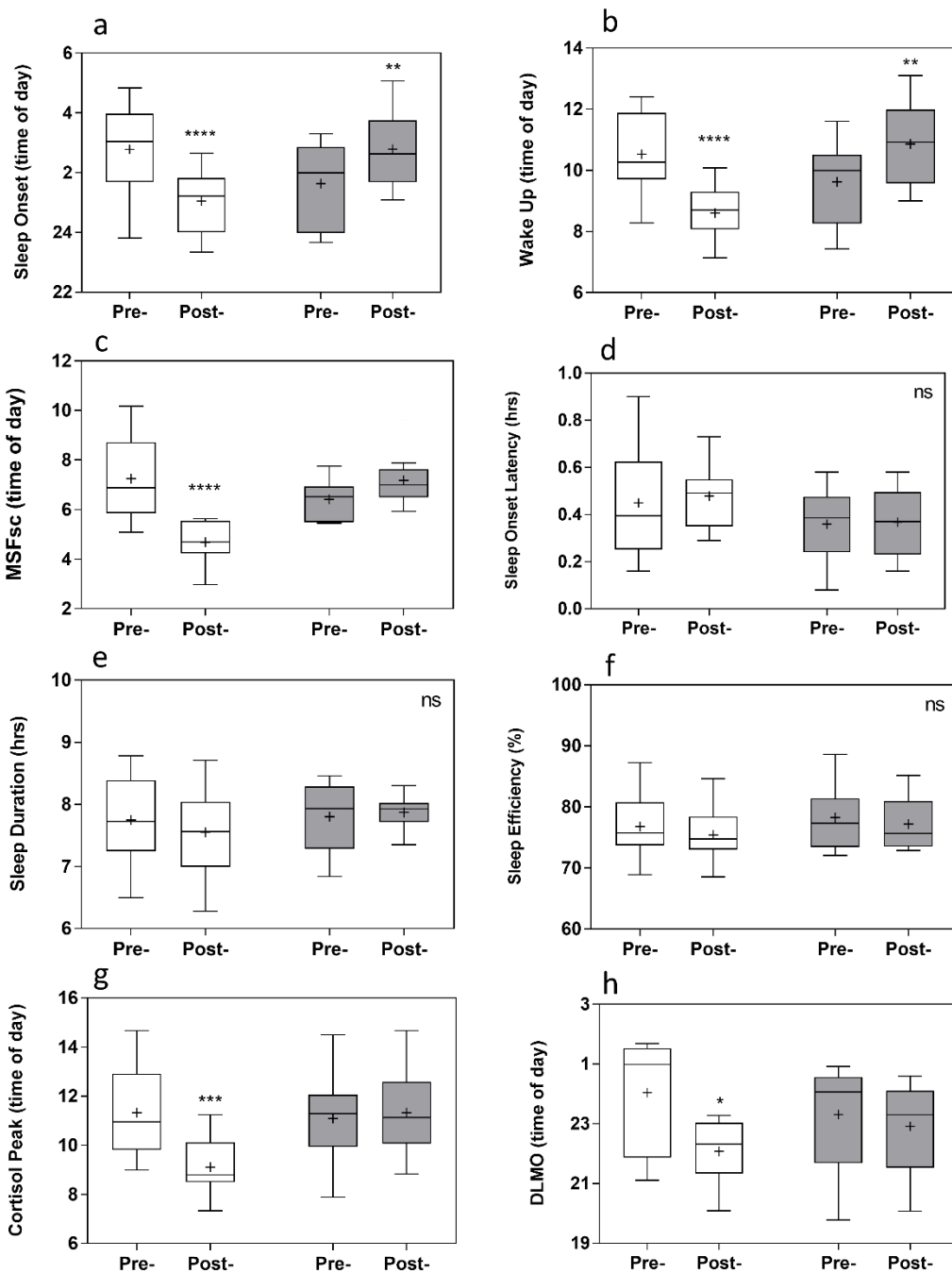
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**Figure 3.** Actigraphy, MCTQ and physiological data pre-intervention (pre-) and post-intervention (post-) for experimental (white) and control (light grey) groups. a) Sleep onset, b) Wake up time, c) Corrected mid-sleep on free days (MSF<sub>sc</sub>), d) Sleep onset latency, e) Sleep duration, f) Sleep efficiency, g) Time of cortisol maximum during the cortisol awakening response, H) Dim light melatonin onset (DLMO). Data are shown as Tukey box-plots; the line in the box indicates the median, the mean value is shown by the + symbol. Asterisks represent significant differences pre- and post-intervention. Ns = not significant, \* =  $p < 0.05$ , \*\*\* =  $p < 0.001$ , \*\*\*\* =  $p < 0.0001$ .

414 \*\*\*INSERT FIGURE 4\*\*\*

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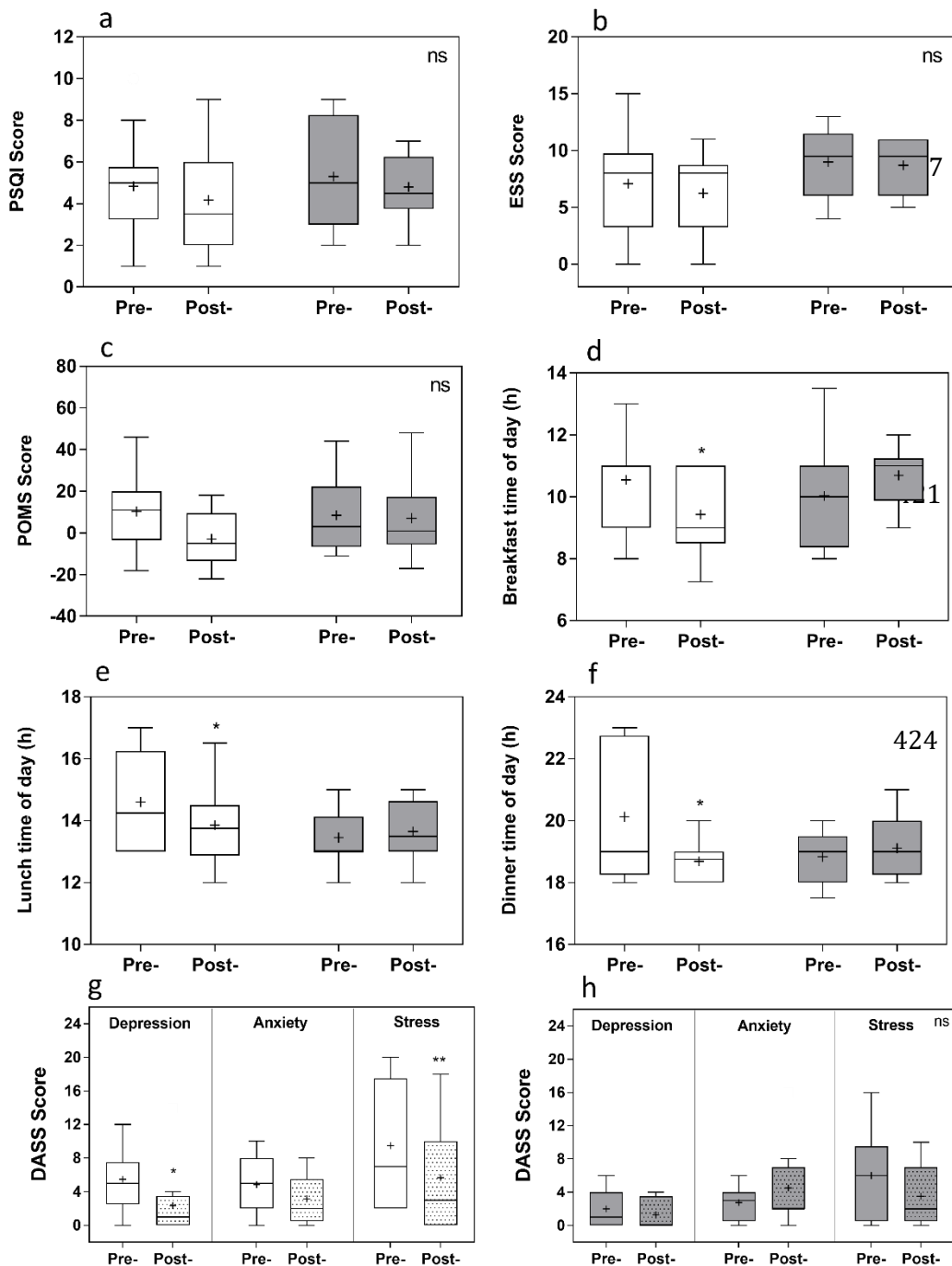
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432 **Figure 4.** Sleep and mental well-being data pre-intervention (pre-) and post-intervention (post-) for  
433 experimental (white) and control (light grey) groups. a) Pittsburgh Sleep Quality Index (PSQI), b) Epworth  
434 Sleepiness Scale (ESS), c) Profile of Mood States (POMS), d) Breakfast time of day (h), e) Lunch time of  
435 day (h), f) Dinner time of day (h). Depression, Anxiety and Stress Scale (DASS) data for experimental (g)  
436 and control (h) groups and shown with a clear pattern (pre-interventions) and a dotted fill pattern (post-  
437 intervention). Data are shown as Tukey box-plots; the line in the box indicates the median, the mean value  
438 is shown by the + symbol. Asterisks represent significant differences pre- and post-intervention. Ns = not  
439 significant, \* =  $p < 0.05$ , \*\* =  $p < 0.01$ .

## 440 **Impact of Interventions on Performance and Sleepiness**

441 Using second order polynomial regression analysis, peak performance and sleepiness times were  
442 identified from best fit diurnal variation curves (Figure 5). Within the constraints of the model (08:00  
443 h to 20:00 h), sleepiness was highest at 08:00 h for both the experimental and control groups pre- and  
444 post-intervention. At the pre-intervention testing, strongest grip strength in the experimental group  
445 occurred at the 20:00 h testing session which advanced to 15:21 h post-intervention. In the control  
446 group, timing of peak grip strength was delayed from 17:12 h to 20:00 h post-intervention. The same  
447 was seen for the PVT with fastest reaction time advancing in the experimental group from 20:00 h to  
448 12:30 h and delaying in the control group from 15:48 h to 19:48 h.

449 There was a significant reduction in inter-individual variation of performance in the experimental  
450 group but no significant changes in the control group. During pre-intervention testing, average grip  
451 strength varied by 14.2% in the experimental group, which was reduced to 7.2% post-intervention ( $p$   
452 = 0.0024). The same was seen for reaction time with average inter-individual differences reduced  
453 from 13.0% pre-intervention to 4.4% post-intervention ( $p$  = 0.028).

454 A significant interaction of time of day and intervention was found for sleepiness in the experimental  
455 group ( $F(2,22) = 3.44$ ,  $p = 0.049$ ) as well as main effects of time of day ( $F(2,22) = 11.41$ ,  $p = 0.0004$ )  
456 and interventions ( $F(2,11) = 5.36$ ,  $p = 0.041$ ). Following interventions, sleepiness was lower at 08:00  
457 h ( $4.6 \pm 0.6$  vs  $6.3 \pm 0.3$ ) and 14:00 h ( $3.6 \pm 0.5$  vs  $4.7 \pm 0.5$ ) but these differences were only  
458 significant at 08:00 h ( $p = 0.0061$ ). The experimental group also showed a significant main effect of  
459 time of day on grip strength performance ( $F(2,22) = 21.73$ ,  $p < 0.0001$ ), as well as a significant main  
460 effect of interventions ( $F(1,11) = 4.94$ ,  $p = 0.048$ ) and an interaction effect ( $F(2,22) = 9.19$ ,  $p =$   
461  $0.0013$ ). Post hoc tests revealed that grip strength at both 08:00 h and 14:00 h significantly improved  
462 following interventions ( $p = 0.015$  and  $p = 0.0075$  respectively). For PVT performance, there was a  
463 main effect of time of day ( $F(2,22) = 3.85$ ,  $p = 0.037$ ) but not interventions. The interaction was found  
464 to be significant, however ( $F(2,22) = 7.93$ ,  $p = 0.0026$ ). Reaction time at 08:00 h was significantly  
465 faster after interventions ( $p = 0.017$ ) but there was no change at 14:00 h or 20:00 h.

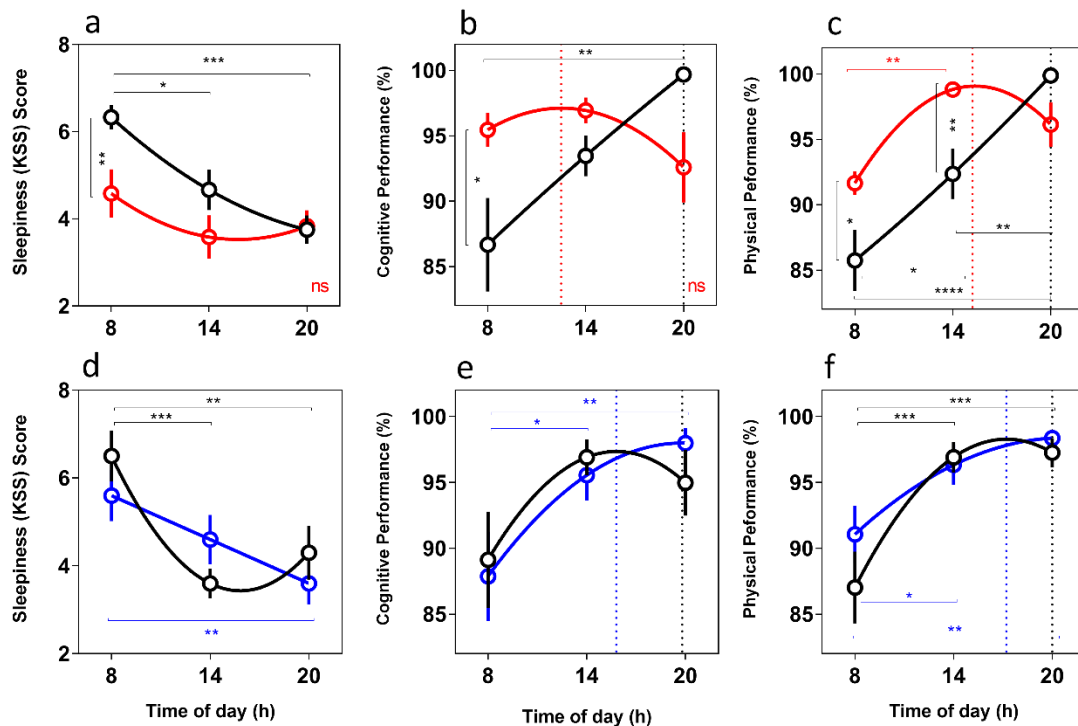
466 In the control group, a significant main effect of time of day was found for sleepiness ( $F(2,18) = 8.86$ ,  
 467  $p = 0.0021$ ), MVC ( $F(2,18) = 14.73$ ,  $p = 0.0002$ ) and PVT ( $F(2,18) = 3.63$ ),  $p = 0.048$ ) performance,  
 468 but not for interventions or the interaction. Post hoc tests did not show any significant changes from  
 469 pre- to post-intervention in the control group for any parameters.

470

471 **\*\*\*INSERT FIGURE 5\*\*\***

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476 **Figure 5. Nonlinear regression curves to show diurnal variations in sleepiness, cognitive and physical**  
 477 **performance pre-intervention (black) and post-intervention in experimental (red) and control (blue)**  
 478 **groups.** (a,d) Subjective sleepiness measured with the Karolinska Sleepiness Scale (KSS). (b,e) Psychomotor  
 479 vigilance task (PVT) performance (average percentage of individual maximum), (c,f) Grip strength performance  
 480 (average percentage of individual maximum). Higher percentages relate to better performance e.g. 100% is  
 481 fastest reaction time and strongest grip strength. Dashed lines represent the time of peak performance in each  
 482 condition (pre-intervention is black in both groups and post-intervention is shown in red for the experimental  
 483 group and blue for the control group). Clock time of test (h) is shown on the x-axis for each parameter. Ns = not  
 484 significant, \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ .

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494 **Discussion**

495 Researchers, clinicians and industry experts are constantly seeking ways to better understand how we  
496 can improve mental health, well-being and performance. One factor that seems constantly overlooked  
497 is the timing of behaviour e.g. sleeping, eating and working. Here we took a group of ‘night owls’ and  
498 attempted to reset their habitual late timings in behaviour in a real-world setting using simple,  
499 practical, non-pharmacological interventions. We show that a phase advance of around two hours can  
500 be achieved which was accompanied by significant reductions in subjective ratings of depression and  
501 stress. In addition, elements of cognitive (reaction time) and physical (grip strength) performance  
502 significantly improved during ‘non optimal’ times, and diurnal peaks in performance occurred earlier  
503 in the day.

504 **Phase Advance**  
505

506 Actigraphy analysis revealed a significant advance in both actigraphic sleep onset and wake up time  
507 pre- to post-intervention in the experimental group. Sleep duration, latency and efficiency all  
508 remained similar pre- and post-intervention confirming that the earlier sleep onset was not associated  
509 with increased sleep latency and hence a curtailment of sleep duration. The behavioural impact of the  
510 intervention can therefore be attributed specifically to the shifting of sleep timing and not to an  
511 alteration of sleep homeostasis. In support of the actigraphy data, we also found a significant phase  
512 advance in melatonin onset (DLMO) of nearly 2 h (00:02 to 22:04 h). This was coupled with a similar  
513 advance in peak timing of the cortisol awakening response that shifted from 11:19 to 09:06 h. Phase  
514 angle, measured as the time between DLMO and sleep onset, was also consistent pre- and post-  
515 intervention. By using a gold standard circadian phase marker, in addition to objective actigraphy,  
516 these results suggest a true circadian phase advance was observed in the experimental group following  
517 the interventions.

518 As light is the dominant zeitgeber of the circadian system it has been one of the main treatment  
519 options of CRSWDs such as DSWPD [48], and mood disorders e.g. seasonal affective disorder [49].  
520 Although controlled light exposure was not specifically administered in this study, participants were

521 asked to wake up earlier and maximise exposure to morning light, thereby contributing to a phase  
522 advance in the circadian system. Simultaneously, the earlier sleep onset times observed combined  
523 with the instructions to decrease evening light exposure e.g. from room lighting and electronic  
524 devices, could have contributed to the delay in DLMO and sleep onset [50] [51]. Timing of food  
525 intake could also be a factor influencing the phase advance. Meal timing has been suggested to have  
526 an entraining effect on the circadian system, in particular the peripheral clocks involved in  
527 metabolism [29] . Along with the importance of sleep for appetite regulation, studies have found that  
528 a morning carbohydrate rich meal can phase advance CBT [52]. There was a significant advance in  
529 average self-reported breakfast time (10:33 to 09:25 h) and an increase in the number of days/week  
530 breakfast was eaten, although this did not quite reach significance. The same was seen with average  
531 self-reported timing of lunch and dinner, which occurred significantly earlier post-intervention,  
532 allowing us to confirm adherence to the intervention requirement of not eating dinner after 19:00.  
533 These advances in meal times, which were observed in the experimental group but not the control  
534 group, could potentially be contributing to the advance in circadian timing, however, as the phase  
535 shifting effects of food were not measured directly in this study it remains speculative.

536

### 537 **Impact of interventions on mental well-being and performance**

538 The association of a delayed sleep phase with reduced mental health e.g. depression, has been shown  
539 in a number of independent studies [17, 53, 54]. Targeting sleep and circadian phase has also become  
540 a focus in the development of novel treatments in neuropsychological disorders. Following the  
541 interventions, we found a significant decrease in depression and stress score in the experimental  
542 group, indicative of better mental health. This was coupled with a similar trend in mood disturbances,  
543 with POMS score reducing from 10.33 to -2.89, although this did not quite reach statistical  
544 significance. Interestingly, it was the depression and stress elements of the DASS scale that were  
545 reduced significantly, with anxiety score not being affected. Although anxiety and depression are two  
546 separate conditions with different diagnostic criteria, they are often comorbid. These results, however,  
547 suggest each factor is affected independently, indicating separable relationships with sleep timing.

548 This is consistent with the literature suggesting that the temporal relationship between  
549 anxiety/depression and reductions in sleep quality or quantity is also different (i.e. anxiety generally  
550 preceding sleep issues, depression generally following sleep issues [55]). Being able to objectively  
551 explore these factors separately and identify the direction of causality would be an important future  
552 step within this work to determine the potential clinical usefulness of the approach for improving  
553 mental health.

554 Daytime sleepiness, measured here using the KSS, is one of the key factors associated with poor  
555 performance [56] and higher risk of errors [57]. Increased sleepiness, leading to lapses of  
556 concentration and even micro sleeps, has been proposed as a main influence in many of the vehicle-  
557 related incidents recorded annually [58]. Being able to reduce daytime sleepiness remains a leading  
558 motivation in both clinical settings and when considering performance/productivity in the real world  
559 [59-61]. Here we show that the experimental intervention significantly decreased daytime sleepiness  
560 at 08:00 h and at 14:00 h. Sleepiness was still at its highest in the morning, although significantly  
561 lower than pre-intervention. This near two-point difference in the morning means a change from  
562 ‘some signs of sleepiness’ to ‘rather alert’ (score of 6 to 4 on the KSS). There was a loss of significant  
563 diurnal variations in KSS score, similar to what was observed for reaction time and grip strength  
564 measures. The KSS score has previously been shown to correlate significantly with performance  
565 variables such as the PVT [62], as well as objective drowsiness [63]. Therefore, this intervention  
566 could prove useful to those professions that are generally more affected by sleepiness and require high  
567 vigilance such as air traffic control, lorry driving and aviation [64], especially since the risk of  
568 accidents has been shown to exhibit diurnal variation [65].

569 Understanding diurnal variations in performance has allowed some studies to shed light on the reason  
570 behind the high risk of motor accidents at non-optimal times of day [66], whilst others have examined  
571 the effect on performance in athletes [67, 68]. In line with these suggestions, we now show the  
572 potential of manipulating these diurnal variations in night owls (LCPs), producing a phase advance, to  
573 create a profile with peak performance occurring earlier in the day. There were significant  
574 improvements in reaction time (measured using a PVT) and isometric grip strength (measured using

575 an MVC test) at ‘non-optimal’ morning times in the experimental group but not in the control group.  
576 The experimental group also showed a significant decrease in diurnal variations of sleepiness and  
577 performance variables. This reduction in amplitude is in line with previous research which showed a  
578 much larger range in performance differences for night owls (LCPs) compared with morning larks  
579 (ECPs) [67, 69]. The diurnal curves of reaction time and grip strength mirror the advance in sleep and  
580 circadian timings, with peak grip strength being shifted from 20:00 h to 15:21 h, and fastest reaction  
581 time occurring at 12:30 h instead of 20:00 h post-intervention.

## 582 **Limitations**

584 It is important to recognise that since we investigated relatively simple measures of cognitive  
585 (reaction time) and physical (grip strength) performance we should be cautious in over generalising  
586 how this intervention would impact more complex measures. Sleep deprivation studies [70, 71] would  
587 suggest that more complex cognitive processes are likely to be affected, although the impact tends to  
588 be smaller. The PVT is a standard tool used in clinical and research settings to measure sustained  
589 attention, and has been shown to be sensitive to sleep loss and time of day [72], with minimal practice  
590 effects. Here we used a shortened version of the PVT (2-minute vs 10 minute) which could have  
591 reduced the sensitivity to time of day effects, as pointed out by Basner, Mollicone [73]. An  
592 investigation into the validity of a 2 minute and 5 minute PVT, however, showed similar time of day  
593 relationships compared to the 10 minute PVT, although overall reaction times were increased with  
594 task duration, as expected [45]. This can give us confidence that the time of day effects we observed  
595 in our study are reliable. Grip strength is a simple measure of muscle strength, which is frequently  
596 used as an evaluation of muscle function in exercise and clinical settings. MVC of isometric grip  
597 strength offers a robust approach to investigating contributions from central and peripheral  
598 mechanisms because the ability to produce maximal force relies on the capability of the muscle as  
599 well as the activation from the central nervous system [69]. Using isometric grip strength allows us to  
600 provide an insight into how this intervention can impact a simple index of physical performance.  
601 Previous research has correlated measures of muscle strength with sprint and jump performance [74].



602 However, performance itself is multifaceted and cannot be defined by one measure alone, so future  
603 work will need to explore how diurnal variations in different cognitive and physical performance  
604 tasks are influenced by this intervention.

605 We have relatively small sample sizes so further studies will be required to investigate how this  
606 intervention could impact larger cohorts and different populations. This also limits our ability to  
607 perform higher order analysis due to low power, which should be incorporated in future research in  
608 line with the discussion from Bland and Altman [75].

609 Although we were able to partially monitor adherence to the interventions, with the experimental  
610 group reporting 78% adherence ( $7.8 \pm 0.7$  out of 10), this was mostly done by self-report. Since the  
611 control group were only asked to eat lunch at the same time each day, this was confirmed with no  
612 significant changes in timing of lunch reported in the diet questionnaire. A more tightly controlled  
613 experiment would have perhaps allowed more detailed assessment of each individual's behaviour and  
614 adherence to the protocol, however a strength of this study is that we were investigating individuals in  
615 a more realistic setting as opposed to artificial laboratory conditions.

616 Despite the value in using a real-world protocol due to its relative ease of implementation and less  
617 disruption to individuals' daily lives, it does limit the ability to control the many environmental and  
618 social influences that can have an impact. In addition, care should be taken when using these  
619 interventions to ensure that the timings do not risk overlapping with the delay period of the human  
620 phase response curve to light [21, 22]. Constant routine and forced desynchrony protocols allow the  
621 characterisation of a truly endogenous rhythm through removing/minimising the influence of external  
622 cues. The present study, however, was not aimed at finding endogenous components to performance  
623 and mental health measures but looked at the integrated system as a whole. The combination of  
624 endogenous circadian rhythms, sleep homeostasis, environmental cues and social schedules is what  
625 affects daily functioning and diurnal variations in the real world. Therefore, although we cannot  
626 attribute the changes we see strictly to one or other of these influences, we provide evidence that a  
627 practical intervention can phase advance night owls in a real life setting with positive outcomes on  
628 self-reported depression and stress, reaction time and grip strength.

629

## 630 **Conclusions**

631 Here we show the ability of a simple non-pharmacological intervention to phase advance night owls,  
632 reduce negative elements of mental health and sleepiness as well as manipulate peak performance times  
633 in the real world. These findings could yield considerable benefits in a number of different settings.  
634 Within the general population, of which a large proportion are night owls, these findings could offer a  
635 simple strategy to improve mental well-being and performance. Within clinical settings, further  
636 treatments for mental health in depression and stress could be explored specifically targeting circadian  
637 disruption without the need for pharmacological agents. This intervention could also be applied within  
638 more niche settings e.g. industry or sporting sectors, who have a key focus on developing strategies to  
639 maximise productivity and optimise performance. Despite the need for further research, this remains an  
640 exciting prospect for a society that is increasingly suffering from poor health, reduced mental well-  
641 being and under continuous pressure to achieve personal best performance.

642

## 643 **Acknowledgements**

644 This work was supported by funding from the Biotechnology and Biological Sciences Research  
645 Council (BBSRC, BB/J014532/1) and the Engineering and Physical Sciences Research Council  
646 (EPSRC, EP/J002909/1). E.F.C was supported by a Wellcome Trust Institutional Strategic Support  
647 Fund (ISSF) Scheme accelerator fellowship (Wellcome 204846/Z/16/Z). Stockgrand Ltd provided the  
648 reagents for melatonin and cortisol measurements. Our sincere thanks are to all participants and to Dr  
649 Olivia Walch for her contribution to the study.

650

## 651 **Author Contributions**

652 E.F.C. and A.P.B. conceived of and designed the study with contributions from D.J.S. E.F.C collected  
653 and analysed the data. RIA analyses was performed by B.M. E.F.C wrote the manuscript with  
654 contributions from A.P.B and D.J.S. All other authors commented on the manuscript.

655 **Competing Interests**

656 B.M. and D.J.S. are co-directors of Stockgrand Ltd. The authors declare no other competing financial  
657 interests.

658

659 **List of abbreviations**

660 ECP: Early circadian phenotype

661 LCP: Late circadian phenotype

662 CBT: Core body temperature

663 CRSWDs: Circadian rhythm sleep-wake disorders

664 MSF<sub>sc</sub>: Corrected mid-sleep on free days

665 DLMO: Dim light melatonin onset

666 KSS: Karolinska Sleepiness Scale

667 PVT: Psychomotor vigilance task

668 MVC: Maximum voluntary contraction

669

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671

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848 **Supplemental Information**

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850 **Supplemental Table 1 (S1).** Summary of demographic, mental well-being, nutrition related,  
851 actigraphic and physiological details pre-intervention for experimental and control groups.<sup>2</sup>

Variable Measured (mean ± SEM)	Experimental Group	Control Group (Con)	Significance
Sample Size	N = 12	N = 10	n/a
<b>Demographic variables</b>			
Age (years, mean ± SD)	21.7 ± 2.8	20.9 ± 3.9	p = 0.60 <sup>b</sup>
Percentage of Males/Females (%)	M: 25	M: 40	p = 0.65 <sup>c</sup>
	F: 75	F: 60	
BMI (mean ± SD)	22.9 ± 3.2	22.6 ± 2.1	p = 0.81 <sup>a</sup>
MCTQ Score (hh:mm)	07:15 ± 00:27	06:24 ± 00:14	p = 0.12 <sup>a</sup>
<b>Nutrition related variables</b>			
Average days per week eating breakfast (days)	4.09 ± 0.62	4.70 ± 0.84	p = 0.38 <sup>a</sup>
Average breakfast time (hh:mm)	10:33 ± 00:25	10:01 ± 00:34	p = 0.47 <sup>a</sup>
Average lunch time (hh:mm)	14:36 ± 00:30	13:27 ± 00:17	p = 0.10 <sup>b</sup>
Average dinner time (hh:mm)	20:07 ± 00:45	18:49 ± 00:17	p = 0.34 <sup>b</sup>
<b>Mental Well-Being Variables</b>			
Pittsburgh Sleep Quality Index (PSQI)	4.83 ± 0.71	5.30 ± 0.80	p = 0.67 <sup>a</sup>
Profile of Mood States (POMS)	10.33 ± 6.15	8.50 ± 5.74	p = 0.54 <sup>a</sup>
Epworth Sleepiness Scale (ESS)	7.08 ± 1.16	9.00 ± 0.99	p = 0.24 <sup>a</sup>
Depression Anxiety and Stress Scale (DASS)	19.83 ± 3.36	13.78 ± 3.66	p = 0.24 <sup>a</sup>
<b>Actigraphy Variables and Non-Parametric Circadian Rhythm Analysis (NPCRA)</b>			
Bed Time (hh:mm)	02:19 ± 00:25	01:16 ± 00:30	p = 0.15 <sup>a</sup>
Get Up Time (hh:mm)	10:46 ± 00:23	09:54 ± 00:31	p = 0.17 <sup>a</sup>
Sleep Onset (hh:mm)	02:46 ± 00:26	01:37 ± 00:30	p = 0.13 <sup>a</sup>
Wake Up Time (hh:mm)	10:31 ± 00:23	09:37 ± 00:29	p = 0.14 <sup>a</sup>
Sleep Duration (h)	7.75 ± 0.20	7.81 ± 0.20	p = 0.91 <sup>a</sup>
Sleep Efficiency (%)	76.80 ± 1.48	78.26 ± 1.91	p = 0.55 <sup>a</sup>
Sleep Latency (hh:mm)	00:27 ± 00:04	00:21 ± 00:03	p = 0.34 <sup>a</sup>
Fragmentation Index	34.86 ± 3.63	30.47 ± 2.27	p = 0.48 <sup>b</sup>
Inter-daily Stability	0.38 ± 0.03	0.38 ± 0.05	p = 0.26 <sup>b</sup>
Intra-daily Variability	0.85 ± 0.05	0.79 ± 0.06	p = 0.25 <sup>a</sup>
L5 Onset (hh:mm)	03:57 ± 00:27	03:03 ± 00:34	p = 0.40 <sup>a</sup>

<sup>2</sup> Values are shown as mean ± SEM unless specified. Significance is shown with <sup>a</sup>unpaired two sample t-tests, <sup>b</sup>non-parametric Mann-Whitney or <sup>c</sup>Fisher's exact test. Phase angle is calculated by the interval time between dim light melatonin onset and sleep onset.

M10 Onset (hh:mm)	12:43 ± 00:37	12:14 ± 00:36	p = 0.96 <sup>a</sup>
Relative Amplitude	0.83 ± 0.03	0.82 ± 0.03	p = 0.26 <sup>b</sup>
<b>Physiological Variables</b>			
Dim Light Melatonin Onset (DLMO) (hh:mm)	00:02 ± 00:34	23:18 ± 00:54	p = 0.97 <sup>a</sup>
Phase Angle (h)	2.94 ± 0.29	2.47 ± 0.72	p = 0.30 <sup>b</sup>
Peak Melatonin Concentration (pg/ml)	26.89 ± 3.98	21.02 ± 5.85	p = 0.22 <sup>a</sup>
Peak Time of Melatonin (hh:mm)	02:06 ± 00:28	02:01 ± 00:33	p = 0.73 <sup>a</sup>
Cortisol Peak Time (hh:mm)	11:19 ± 00:31	11:05 ± 00:36	p = 0.78 <sup>a</sup>
Peak Cortisol Concentration (nmol/l)	23.31 ± 2.39	22.64 ± 3.57	p = 0.79 <sup>b</sup>
Cortisol Awakening Response (%)	113.16 ± 33.71	112.37 ± 45.28	p = 0.64 <sup>b</sup>
Area Under the Curve (total time)	98.83 ± 10.47	104.41 ± 14.01	p = 0.75 <sup>a</sup>

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