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The impact of daylight-saving time (DST) on patients with delayed sleep-wake phase disorder (DSWPD)

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Abstract

Due to time zones, sun time and local time rarely match. The difference between local and sun time, which we designate by Solar Jet Lag (SoJL), depends on location within a time zone and can range from zero to several hours. Daylight saving time (DST) simply adds 1 h to SoJL, independently of the location. We hypothesised that the impact of DST is particularly problematic in patients with delayed sleep-wake phase disorder (DSWPD), worsening their sleep debt. DSWPD is characterised by a chronic misalignment between the internal and social timing, reflected by an inability to fall asleep and wake-up at conventional or socially acceptable times. We analysed the clinical records of 162 DSWPD patients from a sleep medicine centre in Lisbon, Portugal (GMTzone), and separated them into two groups: the ones diagnosed across DST or across Standard Time (ST). We included 82 patients (54.9% male; age: median [Q_1, Q_3] 34.5 [25.0, 45.3]; range 16–92; 54 in DST and 28 in ST) who had Dim Light Melatonin Onset (DLMO) measured as a marker for the circadian phase and sleep timing (onset, SO, mid-point, MS and end, SE) self-reported separately for work- and work-free days. Differences between ST and DST were compared using Mann–Whitney or Student's *t*-tests. On a weekly average, patients in DST slept less (difference between medians of 37 min. $p < .01$), mainly due to sleep on workdays (SD_w, $p < .01$), which also correlated with SoJL ($r_{sp} = .38$, $p < .01$). While the time from DLMO to SO was similar in those in ST or those in DST, the time from DLMO to SE was significantly shorter for those in DST. The average duration between DLMO and sleep end was close to 10.5 h in ST, the biological night length described in the literature. Our results favour perennial ST and suggest assigning time-zones close to sun time to prevent social jetlag and sleep deprivation.

KEYWORDS
chronotype, DLMO, DSWPD, local time, phase angle, solar time

1 | INTRODUCTION

Daylight saving time (DST) is a political decision to advance local time by 1 h in spring and return to standard time (ST) in the autumn. Depending on the location within the time zone, differences between local and (apparent) sun time which we termed as "Solar JetLag" (SoJL) may occur even during ST, but in locations on the west of the time-zone meridian, DST increases these differences by 1 h. Chronobiologists and sleep researchers have highlighted the potential negative effects of DST on health,[1–3] both linked to the DST transition itself (acute) and to the time spent in DST (chronic). DST affects a quarter of the world's population. Its acute consequences have been analysed in several studies excellently summarised in Zhang et al.[3] The Zhang study investigated a health database (150 million) and estimated that DST transitions increase relative risks for many diseases by as much as 10%.[3] Workplace injuries were also reported to increase (5.7%) and workers had 67.6% more sick days due to injuries on the day after DST change than on non-phase change days[4] as well as more fatal traffic accidents (6% in the United States with the spring DST).[5] While our knowledge about the acute effects is growing, those elicited chronically during DST are much less explored.

Most organisms use a circadian clock to predict daily environmental changes and adapt physiology accordingly—from gene expression to behaviour. In mammals, the sleep-wake cycle is the most obvious of these adaptations. Circadian clocks can only reliably predict a rhythmic environment if they are synchronised, which they do actively (entrainment) using so-called zeitgebers (rhythmic environmental cues). By far, the most important zeitgeber is the light:dark cycle.[6,7] The production of melatonin, sometimes called the hormone of the night, is under circadian control but melatonin is also light-degradable. The onset of its production is routinely measured under dim-light conditions (Dim Light Melatonin Onset, DLMO) and many studies also measure its production offset. The melatonin production duration can be used to quantify the biological night. The duration of the biological night is reported to be approximately 10:30 h under highly controlled conditions.[8] One would expect the "biological night" to adapt to the season in the field, but that is surprisingly not the case: Illnerova et al.[9] found it to be around 9:15 h in both winter and summer, although the entire biological night was delayed by approximately 1:30 h in winter. This delay is comparable to sleep questionnaire results.[10]

During entrainment, circadian clocks adapt a specific phase relationship to the zeitgeber (e.g., to the light–dark cycle—sun clock). This "phase of entrainment" varies in individuals. These so-called "chronotypes" from a distribution ranging from extremely early "larks" to extremely late "owls", with the majority ("doves") falling between these two extremes.[11] Over the course of industrialisation, most human clocks have become later due to decreased zeitgeber strength (low light levels in buildings compared to outside during the day and lack of darkness during the night due to artificial light). With social schedules still being rather early, the circadian clocks of late chronotypes are commonly misaligned with the social clocks. This misalignment, also known as "social jetlag" (SJL), has been associated with health deficits.[12] Extreme late chronotypes are often diagnosed with "Delayed Sleep-Wake Phase Disorder" (DSWPD), one of the most common circadian sleep-wake disorders. Its symptoms (as reported by the patient or caregiver), are a chronic (i.e., ≥3 months) inability to fall asleep and wake-up at conventional or socially acceptable times.[13]

Since the spring change of DST is simply a convention to begin work an hour earlier, we hypothesise that DST affects particularly DSWPD patients, who would maintain their late internal DLMO which would reflect in a decrease in the average weekly sleep duration. The biological clock uses light to entrain, with morning light advancing and evening light delaying the clock. Although getting up earlier in summer is accompanied by more exposure to advancing morning light, people get home earlier from work during DST and thus expose themselves to even more delaying evening light. As a consequence, clocks may retain their phase of entrainment during DST (in reference to midnight or noon) or even get later rather than adapting to the advanced social schedule.

2 | MATERIAL AND METHODS

2.1 | Participants

Our sample consisted of 162 DSWPD patients who came to a Portuguese sleep medicine centre (CENC) between 2012 and 2017 and who were diagnosed by board-certified sleep physician (neurologist) according to the ICSD3 criteria.[13] The clinic keeps a database for DSWPD patients.[14]

Saliva was collected in 117 patients, and we were able to assess the melatonin phase (DLMO) in 82 patients (70.1%); in 24 patients, melatonin was already high (>3 pg/mL) in the first sample, and in 11 patients melatonin was low across all timepoints (<1 pg/ml). Thus, only 82 patients were included in this study (54.9% male; the median age of 34.5 ([25.0, 45.3]; range 16–92). 28 patients (34.1%) came first to the clinic during ST and 54 (65.9%) during DST.

2.2 | Procedures

Due to the retrospective, cross-sectional nature of this study using anonymized records, informed consent was not required. The Ethical Committee of the Lisbon Medical School and the Portuguese National Data Protection Board approved the study (Nr – 73/17; 91.359.930).

Socio-demographic data, number of workdays, date of appointment, date of melatonin collection and DLMO phase, as well as sleep timings for work ($_w$) and work-free ($_f$) days, were retrieved from the clinical records. We were able to reconstruct the Munich ChronoType Questionnaire (MCTQ) variables[15] from the self-reports stored in the clinical records[16]: sleep onset time on work- (SO_w) and work-free days (SO_f); mid-sleep timing on workdays (MS_w) and mid-sleep timing on work-free days (MS_f); mid-sleep timing on free-days corrected for the oversleep accumulated over the workweek (MSF_{sc}); sleep end time on work- (SE_w) and work-free days (SE_f). We additionally computed sleep durations (SE-SO) on work- (SD_w) and work-free days (SD_f), weekly sleep duration (SD_{week}) and social jet lag (SJL; quantified by the difference between MS_f and MS_w).[17]

The saliva samples for DLMO assessment were collected at the patient's home, using a melatonin collection protocol commonly used in research and clinical settings.[18] Blue-wavelength-light blocking glasses were distributed to patients who were instructed to start using them from 2 h before the first sampling through to the last collection. Patients were also instructed to refrain from using toothpaste or consuming coffee, tea, alcohol, energetic drinks, chocolate, and banana during saliva collections. Sampling times were defined according to reported average bedtime; 3 h, 2 h, and 1 h before bedtime, bedtime and 1 h after regular bedtime. For the extreme late individuals (bedtime after 04:00 h), collection started at 0:00 h and finished 1 h after regular bedtime. A range of 5–10 collections was obtained from patients. We used three different methods for DLMO estimation: 3 pg/mL, fixed threshold, two standard deviations from the baseline and the hockey-stick method.[19] We opted for the DLMO estimated using the fixed 3 pg/mL threshold for sample size reasons (insufficient points to calculate baseline for some individuals). Results were similar using the other methods.

2.3 | Statistical analyses

Normality was assessed using Shapiro–Wilk test. Categorical variables were described as absolute frequency and percentage. Since the distribution of some of the sleep timing variables were skewed, we decided to represent them as median and interquartile range and used Wilcoxon–Mann–Whitney test for comparisons between patients in ST versus patients in DST. For significant associations, the effect size was estimated with the R and was defined as small 0.1, medium 0.3, and large 0.5.[20] Phase angle differences were normally distributed and therefore represented as mean (±SD) and Student's *t*-tests were used for comparisons (ST vs. DST). For these normally distributed data the effect size was estimated with Cohen's *d* test and was defined as small 0.2, medium 0.5, and large 0.8.[20] All these comparisons and descriptive statistics are shown in Table 1 and Figure 1 (for further reference, Table S1 shows both means and medians). We also used generalised estimating equation (GEE) models adjusted for age and sex to assess the association between (i) *local time* (ST vs. DST), (ii) *work* (workdays vs. work-free days) and (iii) *local time*work* interaction and the dependent variables: (i) *SO*, (ii) *SE* and (iii) *SD*. We used robust estimation of standard errors and specified AR-1 as the correlation structure (GEE).[21] When selecting the model, we used Quasi Information Criterion (QIC) for determining the distribution of the dependent variable and link function to be used: normal-identity in the case of SO and SD_w gamma-log in the case of SE.

As central result variables we established the phase angle differences between DLMO and the sleep phases (SO_w, SO_f, MS_w, MS_f, SE_w, SE_f, and MSF_{sc},). Since all DLMO measurements were performed during the workweek, we used these DLMO phases for both workday and free-day comparisons.

The difference in hours between local and (apparent) solar time, which we termed "solar jetlag" (SoJL) was computed using the R package *solartime*[22] that calculates solar noon at the respective geolocation and calendar day. Daylength was computed using the same package. These calculations were performed for the day of DLMO collection. For the distribution of patients coming to the clinic over the years and the respective distance between local and solar time, see Figure S1.

Spearman's correlation was calculated for sleep timing and DLMO with daylength for each group (ST and DST), as well as for sleep duration with daylength for each group (ST and DST).

All tests were considered statistically significant when $p < .05$. Analyses were performed with SPSS v.27 and R v. 4.1.1 and 4.2.2 (packages: *tableOne*,[23] *geepack*,[21] *tidyr*[24]). We plotted data using *ggplot2*[25] and *ggeffects*.[26]

3 | RESULTS

DSWPD patients during DST, despite the advance in local time for 1 h in reference to solar time, show no difference in the time of DLMO (Table 1). They also have

TABLE 1 Summary of demographic data, sleep, and biological phases of DSWPD in standard time (ST) and daylight-saving time (DST).

	DST	ST	*p*	Effect size
Sample size	54	28		
Age (years; median [IQR]	34.00 [25.75–43.50]	36.00 [23.25, 50.00]	.777	
Sex: male; *n* (%)	33 (61.1)	12 (42.9)	.180	
Workdays per week (*n*; median [IQR])	5.00 [5.00, 5.00]	5.00 [5.00, 5.00]	.985	
Employed (yes) – *n* (%)	43 (79.6)	22 (78.6)	.911	
Sleep-wake behaviour				
SO_w (h; median [IQR])	3.00 [2.29–3.81]	3.21 [2.37–4.00]	.663	
MSW (h; median [IQR])	6.00 [5.00–7.16]	7.21 [5.50–8.50]	.092	
SE_w (h; median [IQR])	9.00 [8.00–11.00]	11.00 [8.50–12.38]	**.020**	$R = 0.26$
SD_w (h; median [IQR])	6.29 [5.00–7.50]	7.00 [6.13–8.90]	**.003**	$R = 0.33$
SO_f (h; median [IQR])	4.00 [3.00–5.00]	4.00 [2.67–5.06]	.914	
MSF (h; median [IQR])	7.86 [6.95–8.85]	8.38 [7.06–9.12]	.343	
SE_f (h; median [IQR])	12.00 [10.38–13.00]	12.00 [11.00–13.69]	.143	
SD_f (h; median [IQR])	7.75 [6.69–8.58]	8.59 [7.00–9.00]	.092	
MSF_{sc} (h; median [IQR])	7.54 [6.32–8.65]	8.25 [6.36–9.14]	.226	
Weekly sleep duration (h; median [IQR])	6.77 [5.70–7.50]	7.39 [6.81–9.00]	**.004**	$R = 0.31$
Social jetlag (h; median [IQR])	1.38 [0.00–2.52]	0.75 [0.00–2.06]	.373	
Circadian phase				
DLMO (h; median [IQR])	1.92 [0.18–2.55]	1.13 [−0.28, 2.21]	.197	
Phase angles				
SO_w − DLMO [mean (SD)]	1.68 (1.28)	2.08 (1.59)	.224	
SO_f – DLMO [mean (SD)]	2.50 (1.38)	2.82 (1.81)	.374	
SE_w – DLMO [mean (SD)]	7.87 (1.89)	9.53 (2.14)	**<.001**	$d = 0.84$
SE_f – DLMO [mean (SD)]	10.03 (2.15)	11.11 (1.90)	**.027**	$d = 0.53$
MSW − DLMO [mean (SD)]	4.84 (1.33)	5.80 (1.68)	**.005**	$d = 0.67$
MSF − DLMO [mean (SD)]	6.18 (1.57)	7.05 (1.74)	**.025**	$d = 0.53$
MSF_{sc} − DLMO [mean (SD)]	5.89 (1.56)	6.72 (1.81)	**.045**	$d = 0.50$

Note: Mann–Whitney, *t*-test or Chi-square according to data distribution. Effect size is given by *R* (small 0.1, medium 0.3, and large 0.5) and Cohen's *d* (small 0.2, medium 0.5, and large 0.8). For MSF_{sc} the samples are $n = 25$ (ST) and 49 (DST) due to the use of alarm clock on free days; All time values presented are given in local time (decimal values).

Abbreviations: DSWPD, delayed sleep-wake phase disorder; DLMO, dim light melatonin onset; IQR, interquartile range (reported as Q1 and Q3); MSF_{sc}, mid-point of sleep on free-days sleep corrected; MSF, mid-sleep point on free-days; MSW, mid-sleep point on workdays; SD_f, sleep duration on free days; SD_w, sleep duration on workdays; SE_f, sleep end on free days; SE_w, sleep end on workdays; SO_f, sleep onset on free days; SO_w, sleep onset on workdays.

similar DLMO times when those are transformed into apparent solar time. (Figure 1). See Figure S2 for individual distribution of DLMO values from both groups (ST and DST), according to local time and solar time.

The times between DLMO and sleep onset were not different in patients under the different conditions, ST and DST (SO_w-DLMO and SO_f-DLMO respectively), while the times between DLMO and sleep end were longer for those in ST than for those in DST (SE_w-DLMO: $p < .001$; SE_f-DLMO: $p = .027$; Table 1).

SoJL median [Q1–Q3] was −1.58 [−1.67 to −1.55] in DST and −0.77 [−0.84 to −0.46] in ST, representing a higher misalignment between local and solar time during DST (Mann–Whitney $U = 1512$, $p < .001$).

As shown before,[10] sleep timing (onset, midpoint, end) was correlated with photoperiod only during ST (Figures S3 and S4). This is another piece of evidence of entrainment to solar time since ST is the one with fewer **So**lar **J**et**L**ag (SoJL; the difference between local and solar time; Figure 2).

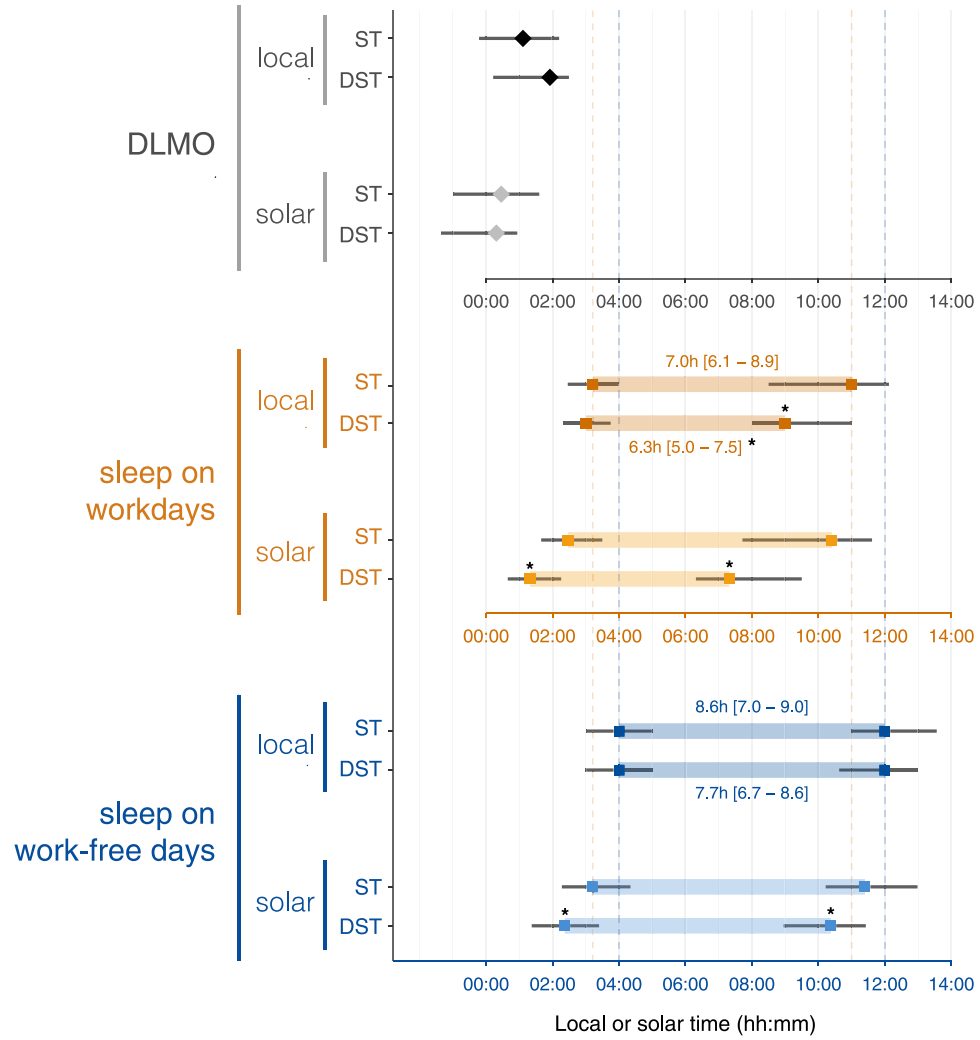

FIGURE 1 Sleep timing and duration on workdays and work-free days according to date of data collection (daylight saving time [DST] vs. standard time [ST]). Dim light melatonin onset (DLMO) was collected once on a weekday and group medians in different contexts are represented as diamonds; whiskers represent Q_1–Q_3. Sleep onsets and offsets are represented as squares; whiskers represent Q_1–Q_3. Sleep duration values are shown on the top/bottom of *local-time* bars as median [Q_1–Q_3] (durations are shown only for local time since they are identical to those in solar time). Please note that despite different median values for sleep durations, the medians of onset and offset on work-free days are at the same times. This means that many people shorten their sleep in DST by i) going to bed later or ii) getting up earlier or iii) both. *$p < .05$ as compared to ST, Wilcoxon–Mann–Whitney test (ST vs. DST).

DSWPD patients during DST slept shorter than those in ST (weekly average; Mann–Whitney $U = 1045$, $p = .004$, Table 1), which was driven by workday sleep (SD_w) (Mann–Whitney $U = 1059.5$, $p = .003$); the DST-ST difference was not significant on work-free days (Mann–Whitney $U = 928.50$, $p = .09$, Figure 1). SD_w also correlated with SoJL ($r_{sp} = .38$, $p < .01$). The association between SD_w and SoJL grouped by month can be seen in Figure 2.

Using GEE, we could not detect a significant interaction effect of *local time*work* but we found a significant association between both *local time* (DST vs. ST) and *work* (work- vs. work-free days) with sleep duration (dependent variable), suggesting that sleep duration may be independently influenced by both variables. SO and SE were significantly associated with *work*, while there was no interaction or effect of *local time*. Sleep end was earlier during DST (see Figure 3), but without reaching significance (DST vs. ST) in the GEE model ($p < .10$) (Table 2).

4 | DISCUSSION

In DSWPD patients, DLMO time is the same for those under standard time (ST) or under daylight saving time (DST) in either local or solar time. This suggests that DST poses a challenge by increasing the distance between local and solar time. However, we cannot rule out or

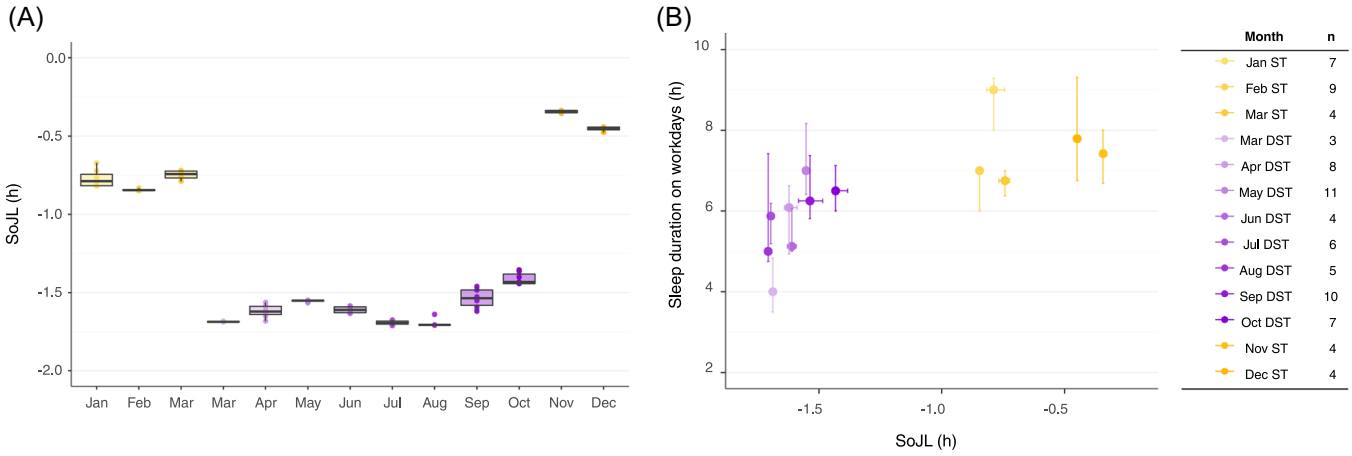

FIGURE 2 (A) Difference between local and solar time – Solar Jet Lag (SoJL) by month throughout the year; there are two entries for March since there are individuals for ST and DST, the same was not the case for the DST-ST transition. (B) Relationship between sleep duration on workdays and the SoJL. Values are presented by month and data are shown as median [Q_1–Q_3]. Purple colour for daylight saving time (DST) and yellow for standard time (ST); SoJL: solar jet lag. The number of subjects per month and condition (ST-DST) are shown on the right.

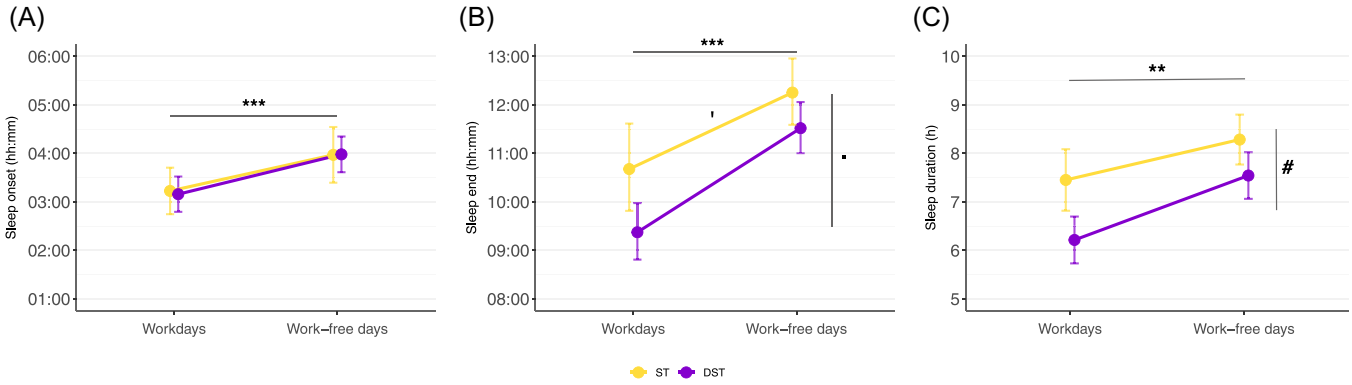

FIGURE 3 Estimated marginal means (95% CI) with age held at 37.1 from GEE models having as dependent variable: (A) sleep onset, (B) sleep end, (C) sleep duration. Asterisks indicate a significant effect of *work* (workdays vs. work-free days; *$p < .05$, **$p < .01$, ***$p < .001$). Sharp indicates a significant effect of *local time* (DST vs. ST; #$p < .05$, $p < .10$). We could not falsify our hypothesis of the interaction of *DST*work* ('$p = .09$). CI, confidence interval; DST, daylight saving time; GEE, generalised estimating equation.

TABLE 2 Association of local time, work, and their interaction with sleep duration, sleep timings (sleep onset and sleep end).

Dependent variable	Local time (DST vs. ST)	Work(workdays vs. work-free days)	Local time*work
Sleep duration (SD)	−0.74 (0.37)*	−0.83 (0.26)**	−0.50 (0.35)
Sleep onset (SO)	0.01 (0.35)	−0.74 (0.18)***	−0.08 (0.23)
Sleep end (SE)	−0.06 (0.04)	−0.14 (0.03)***	−0.07 (0.04)

Note: Generalised estimating equation models results adjusted for age and sex; coefficients (standard errors) are shown. Please note that the distribution of the dependent variable-link function are: normal-identity in the case of SD and SO, and gamma-log in the case of SE.

Abbreviations: DST, daylight saving time; ST, standard time.

*$p < .05$; **$p < .01$; ***$p < .001$.

quantify dependencies of entrainment to one or another (local and solar time), a matter that should be better explored with a larger sample and with a longitudinal design.

Due to social constraints, we would expect that sleep-wake behaviour of the patients in DST would adapt to the change in local time, which in part is the case for these patients, despite their circadian clock not responding to

the change: in a sun-time-centric analysis, sleep-wake behaviour of DSWPD patients is significantly earlier (Figure 1). In a study that recorded actimetry in people (i.e., from the general population) across the DST change for several weeks: while early types adjusted behaviourally to DST, late types did not.[10] Here, we take this finding a step further by showing similar behaviour in DSWPD patients but validating their inability to change with DLMO measurements. Kantermann et al also showed that sleep-wake behaviour correlates with dawn during ST but not in DST. Our findings support this: sleep-wake behaviour in DSWPD patients correlates with photoperiod (daylength) while it does not in the DST. These results again suggest an influence of solar time: by moving local time away from solar time, we may be burdening those with clocks more resistant to adjustments. The discrepancy between local and solar time (SoJL) is larger for DST than ST (Figure 2).

A large cross-sectional study measuring the 24 h peak of cortisol across the year supports the tight coupling between internal time and solar time: instead of the expected change of 1 h between ST and DST, they report only a 2 min difference.[26] A large study of the German population ($N = 21\,600$) showed sleep-wake behaviour progressively delaying from East to West, in line with the sun's progression.[7]

In the present study, the entrained phase of the biological clock (measured by dim-light melatonin onset, DLMO), was unchanged, when corrected for sun time (Figure 1). This means, DLMO should be later when using clock time, which it actually was (though not reaching significance). Our results contrasts those published by Illnerova and colleagues, who found DLMO to be advanced by about 90 min in summer (July) compared to winter (January),[9] which were similar to what Zerbini et al.[27] reported recently (advance by 82 min on workdays and 1 h on free days, comparing June/July with December).

Our results validate those reported by Stothard et al.,[28] who also found DLMO to be later (in local time) in the summer. Although Stothard shows shorter biological nights in summer compared to winter, the DLMO phase does not change in relationship to mid-melatonin. This may reflect mixed light effects, where changing morning light (advancing) is compensated by the equally changing evening light (delaying). This explanation would also explain the lack of differences between the two patient groups. The advancing effects of more morning light (in DST) are compensated both by being exposed to more evening light when coming home earlier in DST, amplified by a higher light sensitivity in the evening, especially for late chronotypes.[29] In addition, extreme DSWPD patients show a decreased light sensitivity in comparison to controls for up to 9 h in the morning (after awakening).[30] Like previously reported in the introduction section, circadian formalism predicts that all chronotypes—except for the extreme larks—should actually get later under DST due to the mentioned increase in advancing light but also an increase in delaying light.[31,32] The altered sensitivities of DSWPD patients would predict an amplification of this effect, which is supported by our results.

Our sample is unique for the disease severity of the DSWPD patients. Their median DLMO was around 1.30 h, and their median sleep onset time was around 3.00 h on workdays and 4.00 h on free days. Other studies of DSWPD report earlier timings for DLMO and sleep onset. In an Australian study, DLMO was earlier than 23.00 h, and the sleep onset was around midnight on workdays, and 1.00 h on free days.[33] Wilson et al.[34] found a sleep onset closer to the present study (3 h 26 min), although the average DLMO was more than 1 h earlier (00 h 18 min). Therefore, our clinical sample represents an opportunity to understand the strain under which very late types are in DST, despite the limitation of not being longitudinal. Nevertheless, sociodemographic characteristics of comparison groups (DST vs. ST) are similar, even if with different sample sizes.

Over the last years, two different phenotypes of DSWPD patients have been reported in the literature, one with a SO-DLMO phase angle in the normal range (~2 h) and another with a larger phase angle.[33,35,36] In our comparison of DSWPD patients in DST and in ST, these two phenotypes are apparently equally distributed in both conditions (DST and ST), since no differences were found in SO-DLMO phase angle on work- ($p = .224$) or work-free days ($p = .374$) between groups. Moreover, the phase angle (SO-DLMO) distribution for both groups is similar to values reported for healthy entrained subjects, in which DLMO was −4.5 h to 0.55 h to bedtime.[32] Further studies are needed to uncover the mechanisms explaining variability in phase angle and respective DSWPD phenotypes.

DSWPD patients sleep longer in ST than those in DST, mainly due to later sleep ends rather than earlier sleep onsets; they start their sleep at the same time relative to DLMO in ST and DST but wake later in ST than in DST, especially on workdays (11 h vs. 9 h). Unfortunately, we cannot rule out that these differences in wake-up time are driven by different work schedules between groups, since this information was not available from the clinical records. However, there were no differences in frequency of unemployment and number of working days between patients in DST versus ST. The time between DLMO and sleep end in ST (workdays 9.53 h; free days 11.11 h) was close to the biological night

length described in the literature.[8] In DST, DSWPD patients are woken on workdays even earlier in their biological night than those in ST.

Under a local-time-centric view, people go to work at the same time in ST and DST but dawn and dusk occur an hour later during DST. However, dawn and dusk only change with natural photoperiod, so under a sun-time-centric view, people go to work an hour earlier during DST. The earlier wakeup during DST shortens sleep by a whole hour compared to those in ST and thereby increases sleep pressure (especially on workdays). Thus, the fact that DSWPD patients sleep earlier during DST when viewed sun-time-centrically (same time under local-time-centric view) may be due to a higher sleep pressure rather than an adaptation to DST. In this paper, we introduced the concept of **So**lar **J**et**L**ag (SoJL), which represents the differences between solar and local time, i.e., quantifies how displaced people have to live from solar time. Notably, SoJL negatively correlates with sleep duration.

During DST, DSWPD patients do not sleep only shorter on workdays, as to be expected in view of earlier work schedules, but also on work-free days (despite nonsignificant in bivariate analysis), which is in accordance with published results.[27] Our statistical model shows that both local time (DST vs ST) and work schedule (work- vs free days) contribute independently to shortening sleep. This suggests that patients on free days, sleep more, independently of being in ST or DST, and that in ST, patients also sleep more independently of being on work or free days. It remains to be seen whether with a larger sample of DSWPD patients interaction would be present.

To our knowledge, this is the first study showing that DSWPD patients under DST have significantly shorter sleep, in comparison to those under ST. A longitudinal study in university students showed a sleep reduction of 25 min for the acute transition from ST to DST,[37] which is less than what we show here for the chronic effects of DST on DSWPD patients (a difference between medians of about 37 min and between averages of 70 min).

In summary, we suggest that DSWPD patients do not adapt to DST similarly to what has been suggested for normal chronotypes.[26]

Although patients were diagnosed in a clinical setting, this study was performed under real-life conditions leading to several limitations: (1) due to its cross-sectional nature, results from ST and DST relate to different patients, precluding intra-individual analyses; (2) saliva samples (for DLMO measurements) were collected at home rather than under controlled laboratory conditions, precluding light measurements before and during collections (for patient instructions, see Section 2.2 Procedures); (3) we only measured DLMO, precluding calculations of melatonin mid- and offset phases; (4) DLMO was only measured for workdays. As so, the phase angle between sleep onset and DLMO for free days was calculated using DLMO values from workdays. Thus, the difference in the phase angle between DLMO and sleep on workdays and free days could be smaller if DLMO from free days had been used. Zerbini et al.,[27] for instance, reported that DLMO on free days was later than on workdays in a sample of young adults. However, our main result is the relationship between SoJL and sleep duration, which was assessed only with sleep data of workdays (5). This study was conducted in an urban environment, thereby confounding behaviour and circadian phase, unlike in the natural light (camping) studies.[28,31]

5 | OUTLOOK

Ideally, this study would be performed under natural light (camping) conditions for the same patients in ST and DST but adding alarm clocks that impose local time. This would eliminate individual electric-light confounders. In addition, it would be important to measure the full melatonin profile. Our study shows how DSWPD patients are affected by DST, which is probably just an amplification of how most chronotypes are affected. For public health reasons, our results recommend ST throughout the year as already recommended by the Sleep and Chronobiology Scientific Societies (European Biological Rhythms Society [EBRS], European Sleep Research Society [ESRS] and Society for Research on Biological Rhythms [SRBR])[1,38] and even suggest reassigning states or regions to their appropriate time zones to reduce internal misalignment and sleep deprivation. This is especially important for DSWPD patients, reducing DSWPD incidence and helping these individuals cope with society (e.g., maintaining their jobs).

AUTHOR CONTRIBUTIONS

Cátia Reis, Till Roenneberg, and Achim Kramer contributed to the design of the study. Cátia Reis and Teresa Paiva sample collection. Luísa K. Pilz and Cátia Reis performed the statistical analysis. Cátia Reis, Luísa V. Lopes and Luísa K. Pilz wrote the manuscript. All authors revised the manuscript and approved the submitted version.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

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

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