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Authors

Wang, Yumeng
Chen, Caihan Tony
Deboer, Tom
[et al.](#)

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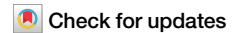
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Daytime illumination shapes sex differences in sleep and circadian responses to nighttime light



Yumeng Wang¹, Caihan Tony Chen¹, Tom Deboer², Gene D. Block¹, Ketema N. Paul³✉ & Christopher S. Colwell¹✉

Light is the primary environmental cue regulating circadian rhythms and sleep–wake behavior, yet daylight intensity is rarely standardized or reported. This variability may contribute to inconsistent findings on sex differences in sleep regulation. In prior work, we observed no sex differences in sleep–wake architecture under relatively bright daylight, suggesting that daytime illumination may be a critical but underappreciated determinant of sex difference in sleep regulation. Here, we tested whether daylight intensity modulates sex differences in sleep–wake architecture and vulnerability to dim light at night (DLaN). Male and female C57BL/6 J mice were exposed to acute (one night) or chronic (two weeks) DLaN (10-lux) under daytime light intensities of 50-, 100-, or 300-lux. Sleep was assessed using electroencephalographic measures of vigilance states and slow wave activity (SWA). Dim daylight (50-lux) revealed marked sex differences in dark phase that were absent under brighter daylight (300-lux). Acute DLaN reduced early-night wakefulness in both sexes under low daytime light but had little effect under bright daylight conditions. Following chronic DLaN, males showed reduced wakefulness and dampened rhythm amplitude, whereas females exhibited larger phase shifts, rhythm attenuation, and altered SWA timing under 50- and 100-lux, and bright daytime light largely prevented these effects.

Sex differences in sleep are robustly documented in humans but remain surprisingly unresolved in animal models. In humans, women more frequently report insomnia symptoms, poorer subjective sleep quality, and greater difficulty maintaining sleep¹, yet objective measures show that women obtain longer total sleep time on average than men². These convergent findings suggest a strong influence of biological sex on sleep regulation in humans. In rodents, however, the evidence is inconsistent, even within the same mouse strain^{3,4}. Some studies report that males spend less time awake and more time asleep than females^{3,5–8}, whereas others detect no sex differences in sleep or wakefulness^{4,9}. The source of this variability remains unclear. Notably, most studies do not report or systematically control the daytime lighting environment, despite the central role of light in shaping circadian and sleep–wake regulation.

Our previous work provides a potential clue: under relatively bright daytime light (300 lux), male and female C57BL/6J mice showed no detectable sex differences in sleep architecture⁴. Bright daytime light strengthens circadian rhythms in humans and other species^{10,11}, and because

sleep–wake organization is strongly governed by the circadian system¹², these findings raise the possibility that daytime light intensity itself may mask or reveal sex differences in sleep. To date, however, this hypothesis has not been directly tested.

Light exposure at night further complicates this picture. Dim light at night (DLaN) is increasingly recognized as a risk factor for sleep disruption, circadian misalignment, cardiometabolic dysfunction, and mood disorders^{13–16}. Human studies suggest that a “bright day & dark night” lighting profile is associated with improved sleep and health outcomes¹⁰. Yet rodent studies of DLaN report highly variable outcomes, with some demonstrating pronounced sleep and circadian disruption and others finding minimal effects. A striking but underappreciated feature of this literature is the wide range of daytime light intensities used across studies.

Here, we directly test whether daytime light intensity shapes sex differences in sleep and vulnerability to nighttime light exposure. By systematically manipulating daytime illumination and nighttime light exposure in male and female mice, we test two related hypotheses: first, that sex

¹Department of Psychiatry & Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA, USA. ²Laboratory for Neurophysiology, Department of Cell and Chemical Biology, Leiden University Medical Center, Leiden, The Netherlands. ³Department of Integrative Biology and Physiology, University of California Los Angeles, Los Angeles, CA, USA. ✉e-mail: ketema.paul@ucla.edu; ccolwell@mednet.ucla.edu

differences in sleep are contingent on daytime light intensity; and second, that brighter daytime light buffers the effects of nighttime light on sleep and circadian regulation. Together, this work provides a unified framework for reconciling inconsistencies in literature and offers new insight into how lighting environments shape sleep health.

Results and discussion

Daytime light intensity alters sleep quantity but not sleep quality in male and female mice

Daytime light intensity did not significantly affect sleep or wakefulness during the light phase across any lighting condition (Fig. 1). In contrast, daytime light intensity strongly influenced the amount of sleep and wakefulness during the dark phase, where sex differences were most pronounced under dim daytime light and progressively diminished with increasing daytime illumination. Across the 24-hour cycle, sex differences were primarily restricted to the dark phase, with no significant differences detected during the light phase. Under 50 lux daytime light condition, female mice exhibited significantly more wakefulness and less sleep during the dark period compared with males, particularly around zeitgeber time (ZT) 18–20 for wakefulness and ZT19–20 for non-rapid eye moment (NREM) sleep. A significant interaction between ZT and sex was observed for wakefulness and NREM sleep, with sex differences evident across all vigilance states (Fig. 1A–C; Supplementary Table 1).

Under 100 lux and 300 lux daytime light conditions, hourly sleep–wake distributions did not differ significantly between sexes (Fig. 1D–I); however, sex differences in overall dark-phase wakefulness and NREM sleep

remained detectable under 100 lux light condition (Fig. 1D, E; Supplementary Table 1). In contrast, under bright daytime light (300 lux), only a main effect of ZT was observed, with no significant sex effect or interaction, consistent with our previous findings (Fig. 1G–I; Supplementary Table 1). Quantification of sleep–wake percentages across light and dark phases confirmed these findings. Under 50 lux daytime light, female mice spent significantly more time awake and less time in NREM and rapid eye moment (REM) sleep during the dark phase compared with males (Fig. 1J–L). Although hourly differences were not detected under 100 lux, females still exhibited increased dark-phase wakefulness and reduced NREM sleep relative to males. No sex differences were observed under 300 lux daytime light (Fig. 1J–L; Supplementary Table 1).

Notably, daytime light intensity exerted a strong effect on dark-phase sleep–wake distribution in female mice. Dim daytime light was associated with increased nocturnal wakefulness, whereas brighter daytime light progressively increased NREM and REM sleep during the dark phase (Fig. 1J–L). In contrast, male mice exhibited minimal sensitivity to daytime light intensity. Together, these findings indicate that sex differences in sleep–wake architecture are driven primarily by increased nocturnal wakefulness in females under dim daytime light conditions.

Overall, these findings indicate that daytime light intensity shapes nighttime sleep–wake structure in a sex-dependent manner. In particular, increasing daytime light intensity reduced the sex differences observed under dim daytime light conditions, while males showed little sensitivity to changes in daytime light intensity across the range tested.

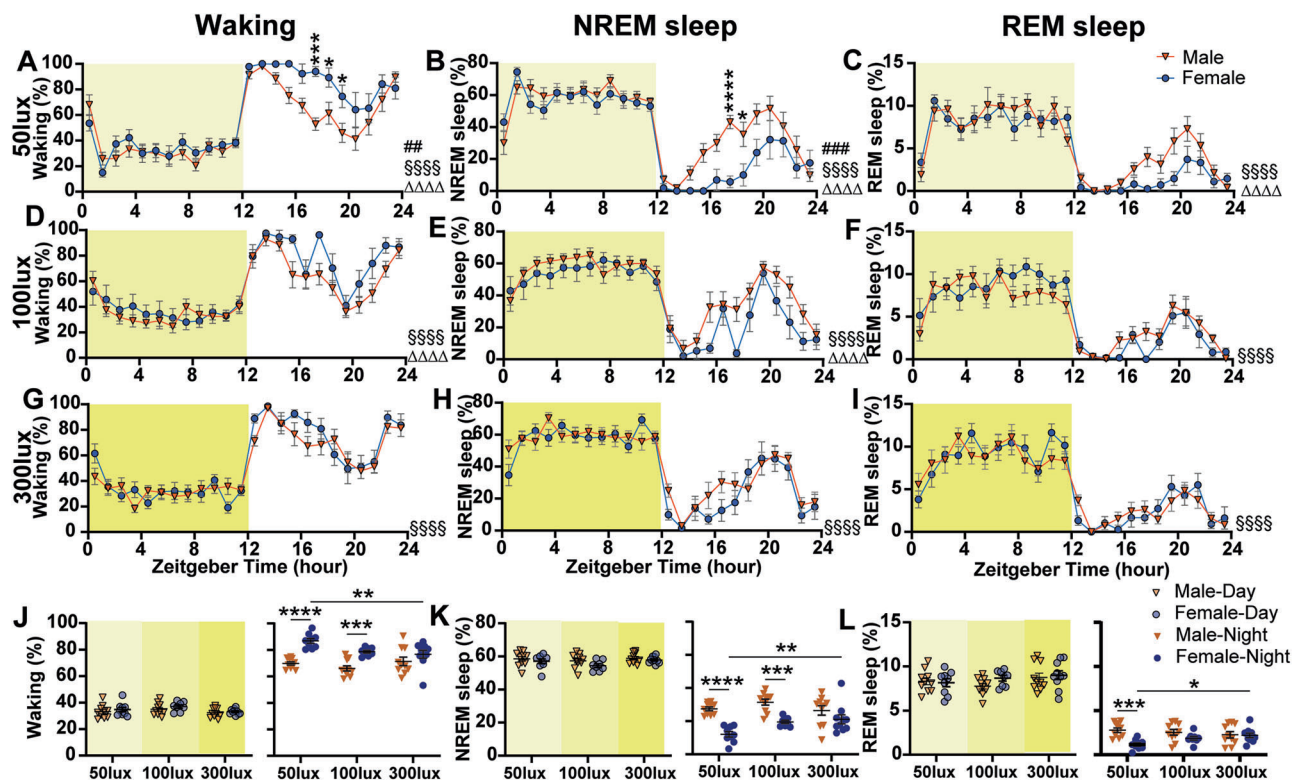


Fig. 1 | Male and female mice exhibit distinct sleep architecture under different daytime light intensities. A–I 24-hour distribution of time spent in waking (A, D, G), NREM sleep (B, E, H), and REM sleep (C, F, I) in male (orange triangles) and female (blue circles) mice under 50 lux (A–C), 100 lux (D–F), and 300 lux (G–I) daylight conditions. Pound signs (#) indicate a significant interaction between the factors zeitgeber time and sex (two-way repeated-measures ANOVA; time as the repeated-measures factor, all tests were two-sided with $\alpha = 0.05$, $\# p < 0.01$, $\#\# p < 0.001$). Main effects of zeitgeber time and sex are indicated by § and Δ, respectively (§§§§ $p < 0.0001$; ΔΔΔΔ $p < 0.0001$). Asterisks indicate significant post hoc differences between male and female mice (Bonferroni multiple comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$). J–L Daytime (left) and nighttime (right) distribution of time spent in waking, NREM sleep, and REM sleep. Data was analyzed using a two-way repeated-measures ANOVA and all tests were two-sided with $\alpha = 0.05$. Asterisks indicate significant post hoc comparisons (Bonferroni multiple comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$). Detailed P values for each panel are provided in Supplementary Table 1. Different shades of yellow represent different daytime light intensities. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 10$ males and $n = 8$ females; 300 lux, $n = 10$ males and $n = 10$ females. Data are presented as mean $\pm$ SEM.

$p < 0.05$, *** $p < 0.001$, **** $p < 0.0001$). J–L Daytime (left) and nighttime (right) distribution of time spent in waking, NREM sleep, and REM sleep. Data was analyzed using a two-way repeated-measures ANOVA and all tests were two-sided with $\alpha = 0.05$. Asterisks indicate significant post hoc comparisons (Bonferroni multiple comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$). Detailed P values for each panel are provided in Supplementary Table 1. Different shades of yellow represent different daytime light intensities. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 10$ males and $n = 8$ females; 300 lux, $n = 10$ males and $n = 10$ females. Data are presented as mean $\pm$ SEM.

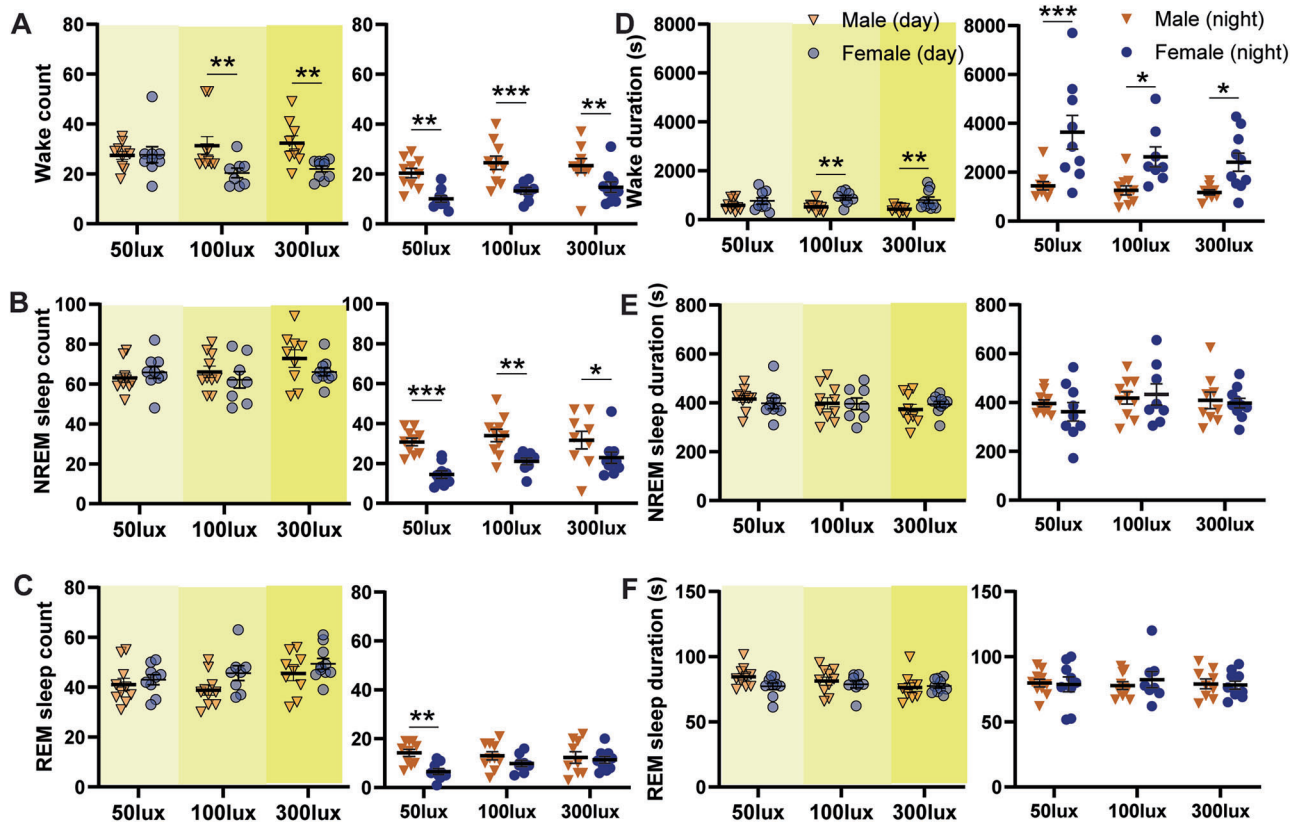


Fig. 2 | Bout number and episode duration in male and female mice under different daytime light intensities. A–C Number of bouts of wakefulness, NREM sleep, and REM sleep during the day (left) and night (right) in male and female mice exposed to 50, 100, or 300 lux daytime light. D–F Episode duration of wakefulness, NREM sleep, and REM sleep during the day (left) and night (right) in male and female mice exposed to 50, 100, or 300 lux daytime light. Data was analyzed using a two-way ANOVA and all tests were two-sided, with $\alpha = 0.05$. Asterisks indicate

significant differences across daytime light intensity or between sexes (Bonferroni multiple-comparisons test after two-way ANOVA; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$). Detailed P values for each panel are provided in Supplementary Table 2. Different shades of yellow represent different daytime light intensities. Data are presented as mean $\pm$ SEM. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 10$ males and $n = 8$ females; 300 lux, $n = 9$ males and $n = 10$ females.

To determine whether daytime light intensity affected sleep quality, we analyzed episode counts and durations for each vigilance state (Fig. 2; Supplementary Table 2). A main effect of sex was observed for wake episode counts, with males exhibiting more wake episodes, consistent with more fragmented wakefulness compared with females (Fig. 2A). For NREM and REM sleep, episode counts were influenced primarily by the dark phase rather than daytime light intensity, with males exhibiting higher counts (Fig. 2B). A sex difference in the number of REM episodes was detected only under 50 lux daytime light condition (Fig. 2C). No significant effects of daytime light intensity were detected on episode counts for any vigilance state (Fig. 2A–C). Similarly, wake episode duration differed by sex, with females exhibiting longer wake bouts than males (Fig. 2D), whereas NREM and REM episode durations were not significantly affected by sex or daytime light intensity (Fig. 2E, F). Together these data indicate that female mice exhibit more consolidated wakefulness than male mice and vigilance state episode consolidation is largely independent of daytime light intensity.

Effects of dim light at night on sleep–wake architecture under different daytime light intensities

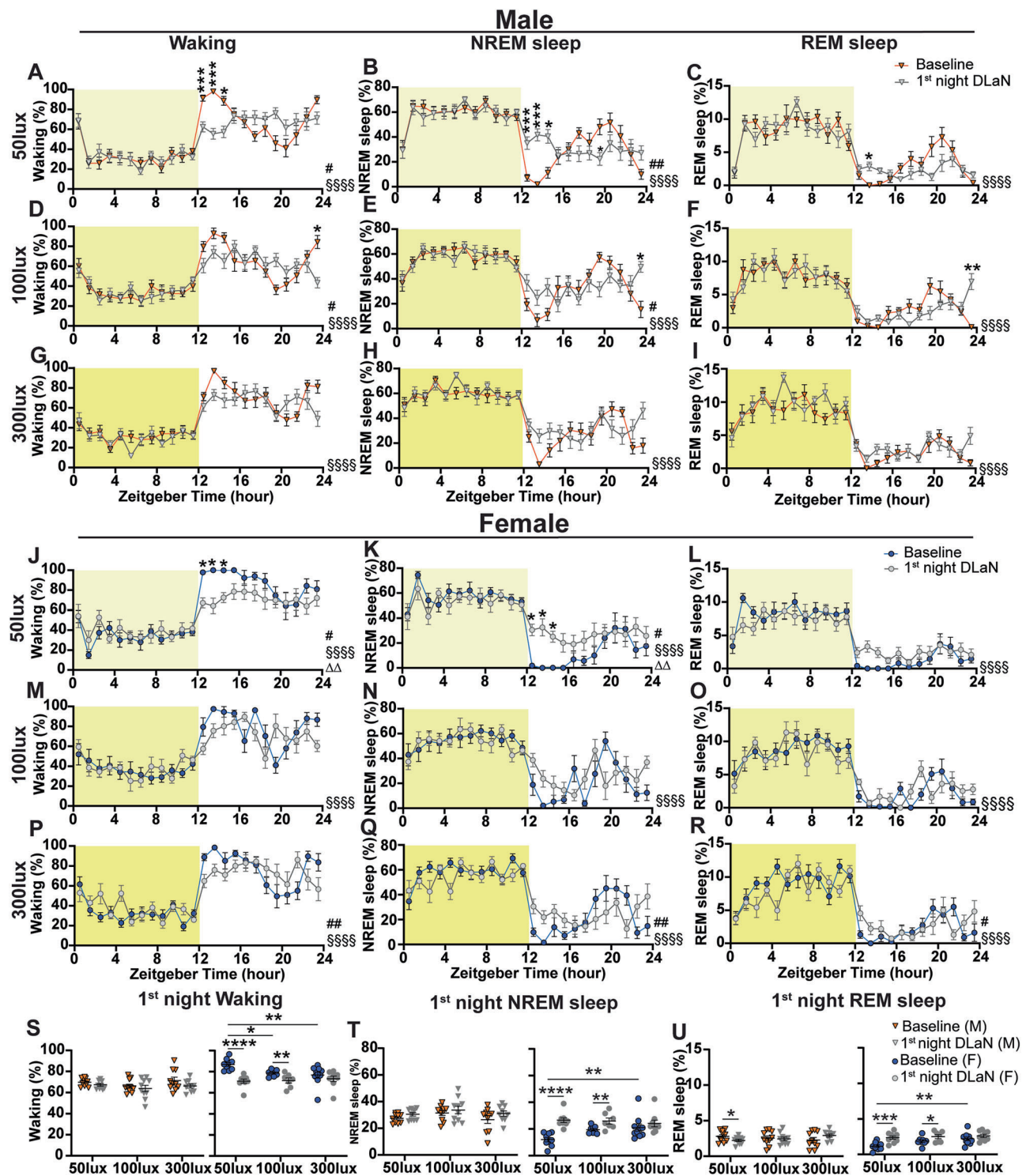
Sleep architecture was evaluated over a 24-hour period in male and female mice housed under dim (50 lux), moderate (100 lux), or bright (300 lux) daytime light levels and examined in acute DLaN (Fig. 3) and after chronic DLaN exposure for two weeks (Fig. 4). The baseline data showed in Figs. 3 and 4 are the same data from Fig. 1.

Acute effects of dim light at night on sleep–wake architecture under different daytime light intensities

As anticipated, the effects of the first night of DLaN were strongly dependent on the daytime light intensity (Fig. 3). Mice housed under 50 lux and 100 lux daytime lighting exhibited pronounced changes in sleep–wake architecture, whereas those housed under 300 lux daytime light showed relatively modest alterations. This pattern was observed in both sexes, although the magnitude and structure of the response differed between males and females.

In males, acute DLaN exposure induced time-specific changes that varied with daytime light intensity. Under 50 lux daytime light, males exhibited reduced wakefulness and increased NREM sleep during the early dim light phase (ZT13–15, Fig. 3A–C). Under 100 lux, males showed increased sleep at the end of the dim light phase (ZT24; Fig. 3D–F). Under 300 lux daytime light, effects of acute DLaN were limited (Fig. 3G–I). Females displayed a distinct response to acute DLaN exposure. Under 50 lux daytime light, females showed reduced wakefulness and increased NREM sleep early in the dim light phase (ZT13–15; Fig. 3J–L). Under 100 lux, no time points were affected by acute DLaN (Fig. 3M–O). Under 300 lux, changes were modest and restricted to the interaction between “zeitgeber time” and “acute DLaN” (Fig. 3P–R).

When sleep–wake states were quantified across the entire 12-hour dim light phase, first-night DLaN had a main effect on nighttime wakefulness and NREM sleep in males, but no main effect of daytime light intensity was detected (Fig. 3S, T). However, a significant interaction between first night DLaN exposure and daytime light intensity was observed for REM sleep (Fig. 3U). Specifically, under 50 lux daytime light, male mice exhibited a



modest but significant reduction in REM sleep following the first night of DLaN exposure. In contrast, females exhibited robust sensitivity to both daytime light intensity and acute DLaN exposure. Significant interactions between DLaN exposure and daytime light intensity were observed for wakefulness and NREM sleep, along with a main effect of DLaN across all vigilance states (Fig. 3S–U; Supplementary Table 3). Under both 50 and 100 lux daytime light, acute DLaN exposure markedly reduced wakefulness and increased both NREM and REM sleep across the dim light phase, effects that were not observed under 300 lux daytime light.

Because light at night is commonly associated with sleep fragmentation, we next examined sleep-wake episode structure following acute DLaN exposure. Although acute DLaN increased the number of NREM and REM sleep bouts, these changes could largely reflect the overall increase in sleep time (Supplementary Fig. 1A–F). Therefore, we further analyzed episode duration, which provides a better measure of sleep-wake consolidation. Compared with females, males exhibited a more pronounced reduction in NREM and REM sleep duration across all three lighting groups, indicating greater sleep fragmentation in response to acute DLaN. In contrast, females showed little or no change in episode duration, suggesting females preserved

Fig. 3 | Sleep architecture of male and female mice during the first night of DLaN under different daytime light intensities. A–I 24-hour distribution of time spent in wakefulness (A, D, G), NREM sleep (B, E, H), and REM sleep (C, F, I) in male mice under baseline conditions (orange triangles) and during the first night of DLaN (gray triangles) following exposure to 50, 100, or 300 lux daytime light. J–R 24-hour distribution of time spent in wakefulness (J, M, P), NREM sleep (K, N, Q), and REM sleep (L, O, R) in female mice under baseline conditions (blue circles) and during the first night of DLaN (gray circles) following exposure to 50, 100, or 300 lux daytime light. Pound signs (#) indicate a significant interaction between zeitgeber time and first night DLaN (two-way repeated-measures ANOVA or mixed-effects model with Geisser–Greenhouse correction, time and 1st night DLaN as the repeated-measures factors, all tests were two-sided with $\alpha = 0.05$; # $p < 0.05$, ## $p < 0.01$). Main effects of zeitgeber time and first-night DLaN are indicated by \$ and Δ , respectively

(\$\$\$ $p < 0.0001$; $\Delta\Delta p < 0.01$). Asterisks denote significant post hoc differences between baseline and first night DLaN (Bonferroni multiple-comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$). S–U Nighttime distribution of time spent in wakefulness, NREM sleep, and REM sleep under baseline conditions and during the first night of DLaN in male (left, triangle) and female mice (right, circle) under different daytime light intensities. Data was analyzed using a mixed-effects model and all tests were two-sided with $\alpha = 0.05$. Asterisks indicate significant post hoc comparisons (Bonferroni multiple-comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$). Detailed P values for each panel are provided in Supplementary Table 3. Different shades of yellow represent different daytime light intensities. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 10$ males and $n = 8$ females (first-night DLaN: 7 females); 300 lux, $n = 10$ males and $n = 10$ females (first-night DLaN: 9 males and 9 females). Data are presented as mean $\pm$ SEM.

sleep-wake consolidation even under acute DLaN (Supplementary Fig. 1G–L).

Together, these findings indicate that daytime light intensity modulates acute sensitivity to light at night. Brighter daytime light substantially attenuated the sleep-wake effects of acute DLaN, whereas mice maintained under 50 or 100 lux exhibited more pronounced alterations in sleep-wake organization. Females showed larger changes in the overall amounts of sleep and wakefulness, whereas males exhibited greater sleep fragmentation, indicating that acute responses to DLaN depend on both daytime light intensity and sex.

Chronic effects of dim light at night on sleep-wake architecture under different daytime light intensities

Because the effects of acute DLaN were highly dependent on daytime light intensity and differed by sex, we next examined whether these responses persist, adapt, or worsen following chronic DLaN exposure. After 2 weeks of DLaN exposure, sleep-wake alterations became more pronounced and diverged markedly from the acute responses observed after a single night of DLaN. As with acute exposure, chronic DLaN did not significantly affect sleep-wake organization during the light phase; instead, the most prominent alterations occurred during the dim light phase (Fig. 4).

In male mice, chronic DLaN elicited a highly consistent and reproducible response across all three daytime light intensities. Compared with baseline light-dark (LD) conditions, males exhibited increased sleep and reduced wakefulness at both the beginning and end of the dim light phase under 50, 100, and 300 lux daytime light (Fig. 4A–I). Significant interactions between zeitgeber time and DLaN exposure were detected for wakefulness, NREM sleep, and REM sleep across all daytime light conditions (Supplementary Table 4), indicating a robust and light-intensity-independent response to chronic DLaN. This response pattern closely mirrors previous reports of chronic DLaN exposure in male rodents, including a 14-day DLaN paradigm in rats¹⁷.

In contrast, female mice exhibited a strikingly different and unexpected response to chronic DLaN exposure. Unlike males, females did not show reduced wakefulness or increased NREM sleep at the onset of the dim light phase, with no significant differences observed at ZT13 or ZT14 under any daytime light condition (Fig. 4J–R). Instead, the temporal structure of the DLaN response in females depended strongly on daytime light intensity. Under 50 lux daytime light, wakefulness was reduced during the middle and end of the dim light phase (ZT20), accompanied by increases in both NREM and REM sleep. Under 100 and 300 lux daytime light conditions, reductions in wakefulness and increases in NREM sleep were largely restricted to the end of the dim light phase (ZT23; Fig. 4J–R). Notably, unlike male mice, females also exhibited DLaN-induced alterations during the light phase. Under 100 lux daytime light, chronic DLaN reduced REM sleep during the mid-light phase (ZT 9; Fig. 4O). Significant interactions between time and DLaN exposure were detected across all three daytime light intensities in females (Supplementary Table 4), indicating a temporally dynamic but context-dependent response to chronic DLaN. Regardless of sex, both sexes

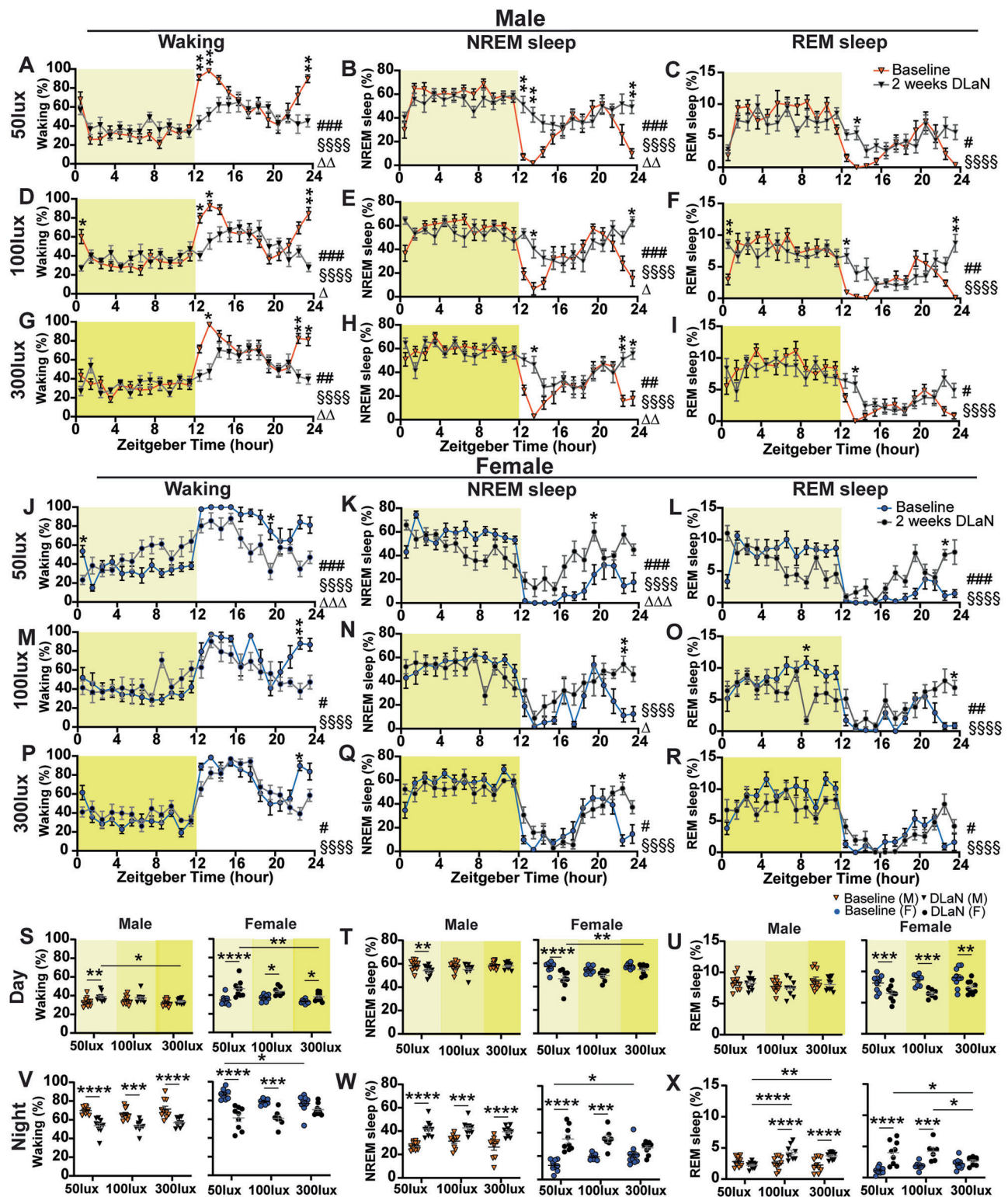
exhibited a similar anticipatory pattern prior to lights on (ZT 0) under baseline LD conditions, characterized by increased wakefulness, consistent with anticipation of the light phase. Across all light conditions, this increase in wakefulness was markedly attenuated under chronic DLaN exposure, indicating a loss of anticipation of the start of light phase in both sexes.

To assess total amount of sleep and wakefulness, we next quantified total 24-hour amounts of vigilance states under baseline and chronic DLaN conditions. Male mice displayed a consistent response across all three daytime light intensities, characterized by decreased wakefulness and increased NREM sleep, with relatively minor effects on REM sleep (Supplementary Fig. 2A–C). The female mice housed under 50 and 100 lux daytime light conditions also showed similar overall patterns, whereas females housed under 300 lux daytime light conditions did not exhibit significant changes in total wakefulness or NREM sleep. In contrast to males, REM sleep responses in females were strongly modulated by daytime light intensity, increasing under 50 lux but decreasing under 300 lux conditions (Supplementary Fig. 2D–F).

Because the largest effects were observed during the dark phase, we further analyzed sleep-wake distribution separately for the light and dark/dim light phases. During the light phase, male mice exhibited modest increases in wakefulness and corresponding decreases in NREM sleep under 50 lux daytime light, with no significant effects on REM sleep under any lighting condition (Fig. 4S–U). These limited light-phase effects in males are consistent with prior DLaN studies. Female mice, however, showed robust light-phase responses to chronic DLaN across all daytime light intensities, characterized by increased wakefulness and reduced REM sleep (Fig. 4S, U). In contrast, dark-phase responses were more similar between sexes. Male mice showed decreased wakefulness and increased NREM sleep across all three daytime light conditions, whereas females exhibited comparable dim light-phase changes under 50 and 100 lux but not under 300 lux daytime light (Fig. 4V–X). The absence of significant dim light-phase effects in females under bright daytime light suggests that higher daytime illumination partially protects against DLaN-induced sleep disruption.

Effect of dim light at night on quality of sleep and wakefulness under different daytime light intensities

We next examined sleep-wake episode structure after two weeks of DLaN exposure. Consistent with the acute effects observed during the first night of DLaN, bout numbers remained elevated during the dim-light phase, likely reflecting DLaN-induced changes in the distribution of sleep and wakefulness (Supplementary Fig. 3A–F). To assess whether chronic DLaN also altered sleep-wake consolidation, we analyzed episode duration. Chronic DLaN selectively shortened wake episodes during the dim-light phase, whereas the durations of NREM and REM sleep episodes were unchanged (Supplementary Fig. 3G–I). Notably, females exposed to 300 lux during the daytime did not exhibit a significant reduction in wake episode duration, suggesting that brighter daytime light may preserve wake consolidation during chronic DLaN exposure. Finally, analysis of vigilance-state transitions revealed sex-specific and phase-dependent changes during both the



light and dark/dim-light phases, indicating that DLaN alters sleep–wake dynamics beyond simple changes in vigilance-state occupancy (Supplementary Fig. 4).

Chronic DLaN produced robust but sex-dependent changes in sleep–wake architecture. The responses of males were largely independent of daytime light intensity, whereas females were strongly affected by daytime light intensity, with brighter daytime light attenuating DLaN induced sleep changes and preserving sleep–wake consolidation.

Effects of acute and chronic dim light at night on electroencephalogram slow wave activity in NREM sleep in male and female mice under different daytime light intensities

During the first night of DLaN exposure (Fig. 5A–F), neither male nor female mice exhibited a significant interaction between DLaN and ZT, indicating minimal acute effects on NREM sleep electroencephalogram (EEG) slow wave activity (SWA, EEG power density between 0.5–4.0 Hz, an important marker for quality and depth of sleep; Supplementary Table 5). In

Fig. 4 | Sleep architecture of male and female mice following 2 weeks of DLaN under different daytime light intensities. A–I 24-hour distribution of time spent in wakefulness (A, D, G), NREM sleep (B, E, H), and REM sleep (C, F, I) in male mice under baseline conditions (orange triangles) and after 2 weeks of DLaN exposure (black triangles) following exposure to 50, 100, or 300 lux daytime light. J–R 24-hour distribution of time spent in wakefulness (J, M, P), NREM sleep (K, N, Q), and REM sleep (L, O, R) in female mice under baseline conditions (blue circles) and after 2 weeks of DLaN exposure (black circles) following exposure to 50, 100, or 300 lux daytime light. Pound signs (#) indicate a significant interaction between zeitgeber time and DLaN (two-way repeated measures ANOVA or mixed-effects model with Geisser–Greenhouse correction, time and DLaN as the repeated-measures factors, all tests were two-sided with $\alpha = 0.05$; # $p < 0.05$, ## $p < 0.01$, ### $p < 0.001$). Main effects of zeitgeber time and DLaN are indicated by § and Δ, respectively

(§§§§ $p < 0.0001$; Δ $p < 0.05$, ΔΔ $p < 0.01$, ΔΔΔ $p < 0.001$). Asterisks denote significant post hoc differences between baseline and DLaN (Bonferroni multiple-comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$). S–X Daytime and nighttime distribution of time spent in wakefulness, NREM sleep, and REM sleep under baseline conditions and after 2 weeks of DLaN exposure in male (left, triangle) and female mice (right, circle) under different daytime light intensities. Data was analyzed using a mixed-effects model and all tests were two-sided with $\alpha = 0.05$. Asterisks indicate significant post hoc comparisons (Bonferroni multiple-comparisons test; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$). Detailed *P* values for each panel are provided in Supplementary Table 4. Different shades of yellow represent different daytime light intensities. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 10$ males and $n = 8$ females (DLaN: 7 females); 300 lux, $n = 10$ males and $n = 10$ females (DLaN: 9 males and 9 females). Data are presented as mean ± SEM.

male mice housed under 100 lux daytime light, acute DLaN exposure was associated with transient increases in SWA at late dark-phase time points (ZT 21, 23). In contrast, under 300 lux daytime light, no significant main effects or interactions with DLaN were observed in either sex. Together these findings suggest that brighter daytime light attenuates acute DLaN-induced alterations in SWA across sexes.

Following two weeks of DLaN exposure, sex- and light intensity-dependent differences in NREM SWA emerged. In male mice, a significant interaction between DLaN exposure and ZT was observed under 100 lux daytime light (Fig. 5H), whereas no significant interaction or main effect of DLaN was detected under 50 or 300 lux conditions (Fig. 5G, I). Notably, under 50 lux daytime light, males exhibited a localized increase in SWA at ZT20 (Fig. 5G). In contrast, female mice exhibited more robust and widespread SWA responses to chronic DLaN exposure across all daytime light intensities. Under 50 lux daytime light, females showed a pronounced increase in SWA during the light phase, particularly between ZT 9 and ZT 12 (Fig. 5J). Significant interactions between DLaN exposure and ZT were also observed under both 100 lux and 300 lux daytime light conditions (Fig. 5K, L), indicating temporally reorganized SWA dynamics rather than isolated changes in magnitude.

Power spectral analysis of NREM sleep further supported these sex-specific effects. In male mice, chronic DLaN exposure produced a significant increase in delta power. In female mice, a significant main effect of DLaN on NREM spectral power was observed, although no frequency-specific changes were detected (Supplementary Fig. 5), consistent with a redistribution of SWA timing rather than a selective enhancement of delta-band activity.

Acute DLaN produced only modest changes in SWA, and these effects were largely buffered by brighter daytime light in both sexes. In contrast, chronic DLaN produced pronounced sex-dependent effects: males exhibited increased SWA, whereas females showed a redistribution of SWA across the sleep–wake cycle. Brighter daytime light attenuated the increase in SWA in males and helped preserve its temporal organization in females, indicating that daytime light intensity modulates the effects of nighttime light on sleep homeostasis in a manner dependent on both sex and duration of exposure.

Daily timing and amplitude of the sleep–wake rhythm under baseline and DLaN conditions

As slow wave activity during NREM sleep was strongly influenced by the endogenous circadian clock, and in our female mice, we observed a strong interaction between time and DLaN. We next examined whether the daily timing and amplitude of vigilance states were altered under DLaN conditions. To address this, we applied JTK_CYCLE analysis to baseline and DLaN datasets, allowing us to assess changes in the daily peak time and amplitude of wakefulness, NREM sleep, and REM sleep under different daytime light intensities.

Surprisingly, male mice did not exhibit any significant changes in the peak timing of wakefulness, NREM sleep, or REM sleep under DLaN conditions across all daytime light intensities (Fig. 6A–I). In contrast, female mice showed significant phase advances compared with baseline under

dimmer daytime light conditions. Under 50 lux daytime light, females exhibited a marked phase advance around 4 h in wakefulness, NREM sleep and REM sleep (Fig. 6S, V, Y). Similarly, under 100 lux daytime light conditions, female mice showed significant phase advances around 3 h compared with baseline in wakefulness and NREM sleep (Fig. 6T, W). Thus, under both 50 and 100 lux conditions, a phase advance of approximately 3–4 h emerged as a consistent feature of the female response to chronic DLaN. This advance likely explains why female mice did not exhibit the expected decrease in wakefulness or increase in sleep at the beginning of the active phase after two weeks of DLaN exposure: their peak in waking had shifted to earlier ZT. Notably, under 300 lux daytime light conditions, neither the timing of the vigilance state peaks nor overall sleep–wake amounts were significantly altered, suggesting that brighter daytime light may protect against DLaN induced phase shifts and their downstream effects on sleep architecture in female mice.

We next analyzed the amplitude of daily oscillations in vigilance states. In male mice, DLaN exposure led to significant reductions in oscillation amplitude for wakefulness (Fig. 6J–L), NREM sleep (Fig. 6M–N), and REM sleep (Fig. 6P–Q) across all conditions. In female mice, decreases in amplitude were observed for wakefulness (Fig. 6AB–AC), NREM sleep (Fig. 6AE–AF), and REM sleep (Fig. 6AH–AI) under 50 and 100 lux daytime light conditions. However, female mice housed under 300 lux daytime light did not exhibit significant amplitude reductions, further supporting the notion that brighter daytime light may protect against DLaN induced disruptions in both the timing and strength of daily sleep–wake rhythms, particularly in females. Together these data show that chronic DLaN differentially affects timing and amplitude of sleep–wake rhythms in males and females with females showing more susceptibility to DLaN.

Together, these findings demonstrate that chronic DLaN reorganizes daily sleep–wake rhythms in a sex-dependent manner. Males primarily exhibited a reduction in rhythm amplitude, whereas females showed phase advances under dim daytime light conditions. Notably, increasing daytime light intensity to 300 lux prevented the phase advance in females, suggesting that brighter daytime light enhances circadian robustness and may buffer against DLaN-induced disruption.

Daytime light shapes sex differences in sleep–wake architecture

Light is a fundamental regulator of circadian and sleep–wake behavior, yet the influence of daytime light intensity has been studied primarily in humans and in male laboratory animals¹⁸. In diurnal species, brighter daytime light strengthens circadian rhythms and improves sleep consolidation¹¹. For example, in a recent study, exposure of diurnal male Nile grass rats to bright daytime light (1000 lux) increased wakefulness, reduced NREM sleep, and increased delta EEG power compared with dimmer daytime light⁸. In nocturnal rodents brighter daytime light has often been assumed to be detrimental, based largely on studies using constant illumination or extremely high light intensities (>1000 lux) in albino strains, conditions that are likely to induce retinal pathology rather than physiological circadian responses^{19,20}. In contrast, there is little evidence that moderately brighter daytime light impairs sleep or circadian regulation under stable light–dark conditions.

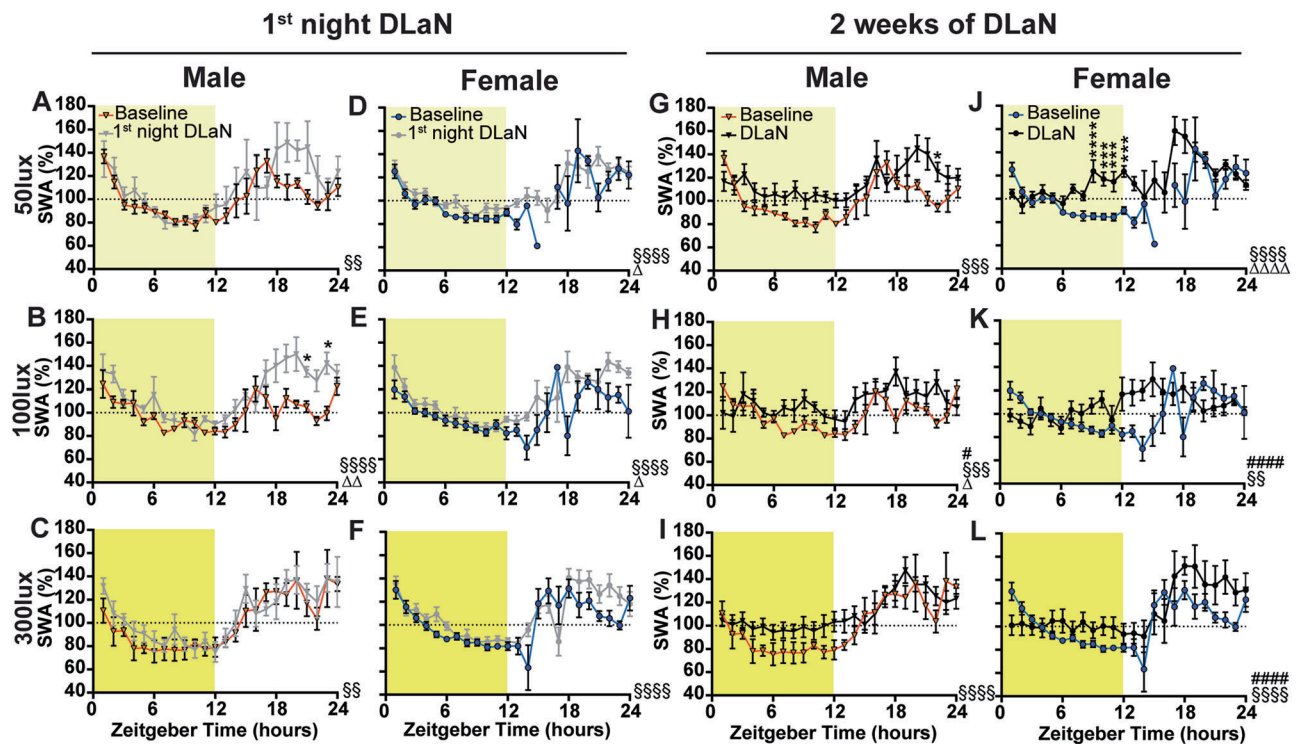


Fig. 5 | NREM sleep slow-wave activity in male and female mice exposed to 1st night of DLaN and 2 weeks of DLaN under different daytime light intensities. A–F 24-hour distribution of NREM sleep slow-wave activity in male (A–C; triangles) and female (D–F; circles) mice following exposure to 50, 100, or 300 lux daytime light, during baseline (orange or blue) and the first night of DLaN (gray). G–L 24-hour distribution of NREM sleep slow-wave activity in male (G–I; triangles) and female (J–L; circles) mice following exposure to 50, 100, or 300 lux daytime light, during baseline and after 2 weeks of DLaN exposure (black). Statistical analysis was performed using a mixed-effects model with Geisser–Greenhouse correction, time and 1st night DLaN or DLaN as the repeated-measures factors, all tests were two-sided with $\alpha = 0.05$. Due to missing time points in the 50lux female baseline, only main effects were analyzed in panels (D) and (J); Bonferroni’s multiple-comparisons

test was performed following a mixed-effects model, with ZT13–ZT19 excluded from the analysis. Pound signs (#) indicate a significant interaction between zeitgeber time and DLaN ($\#p < 0.05$, $\#p < 0.01$, $\#p < 0.001$, $\#p < 0.0001$). Main effects of zeitgeber time and DLaN are indicated by \$ and Δ, respectively ($\$p < 0.01$, $\$p < 0.001$, $\$p < 0.0001$; $\Delta p < 0.05$, $\Delta p < 0.01$, $\Delta p < 0.001$, $\Delta p < 0.0001$). Asterisks denote significant post hoc differences between baseline and DLaN conditions (Bonferroni multiple-comparisons test; $*p < 0.05$, $***p < 0.001$, $****p < 0.0001$). Detailed *P* values for each panel are provided in Supplementary Table 5. Different shades of yellow represent different daytime light intensities. Sample sizes: 50 lux, $n = 10$ males and $n = 9$ females; 100 lux, $n = 9$ males (DLaN: 8) and $n = 8$ females (1st night DLaN: 6, DLaN: 7); 300 lux, $n = 8$ males and $n = 10$ females (1st night DLaN: 9, DLaN: 9). Data are presented as mean $\pm$ SEM.

Whether commonly used daytime light intensities differentially affect sleep-wake regulation across sexes has remained unclear. Across the rodent literatures, reported sex differences in sleep-wake architecture are inconsistent, and daytime light intensity is rarely reported, complicating comparisons across species. We systematically examined sleep-wake architecture in male and female mice housed under 50, 100 and 300 lux daytime light intensity and found that sex differences in sleep-wake behavior depend critically on light intensity: female mice exhibited increased wakefulness under dim daytime light (50 and 100 lux), whereas no sex differences were observed under brighter daytime light (300 lux), consistent with our previous study conducted under the 300 lux conditions⁴. These sex differences were driven primarily by increased wakefulness in females under dim daytime light rather than by changes in males, while sleep quality was largely preserved across all conditions, indicating that daytime light modulates the amount of sleep and wakefulness rather than its quality.

Interestingly, a recent study in Nile grass rat reported sex-dependent differences in NREM and REM sleep under brighter daytime light conditions (1000 lux), whereas our results showed that brighter daytime light largely attenuated sex differences in nocturnal mice⁸. In a separate study using piezoelectric sleep monitoring, daytime light intensity (1000 versus 50 lux) did not significantly affect the percentage of time spent asleep during either the day or night in either sex. However, male Nile grass rats exhibited longer sleep bouts under brighter daytime light, suggesting improved sleep consolidation²¹. Although direct comparisons should be made cautiously because of differences in species, light exposure protocols, and sleep-

assessment methods, these findings raise the possibility that diurnal and nocturnal rodents respond differently to daytime light intensity. Such differences could reflect variation in retinal photic processing, circadian organization, or the neural circuits regulating sleep and arousal across diurnal and nocturnal species.

Sex-specific resilience and vulnerability to light at night

Acute and chronic DLaN exert distinct effects on sleep-wake behavior that are strongly sex- and daytime light intensity-dependent. A single night of DLaN produced rapid but temporally restricted changes in sleep continuity and fragmentation in both sexes, indicating that even brief nighttime light exposure can increase sleep and reduce sleep quality. These acute effects were broadly conserved: males and females showed transient increases in sleep, with subtle differences in magnitude and temporal distribution. In humans, similar single-night dim light exposure increases sleep duration though daytime light was not controlled^{22,23}. Prolonged DLaN, in contrast, induced more persistent alterations in sleep-wake architecture, including dampened daily amplitude, disrupted anticipation of lights-on, and sex-specific changes in the timing and distribution of vigilance states. Males maintained a largely conserved temporal structure with reduced daily amplitude, exhibiting delayed wake onset and increased sleep at both the beginning and end of the dim light phase, similar patterns previously reported in rodents^{17,24}. Females, however, showed pronounced phase advances and redistribution of sleep and wakefulness across the 24-hour cycle, with reduced wakefulness and increased sleep late in the dim light

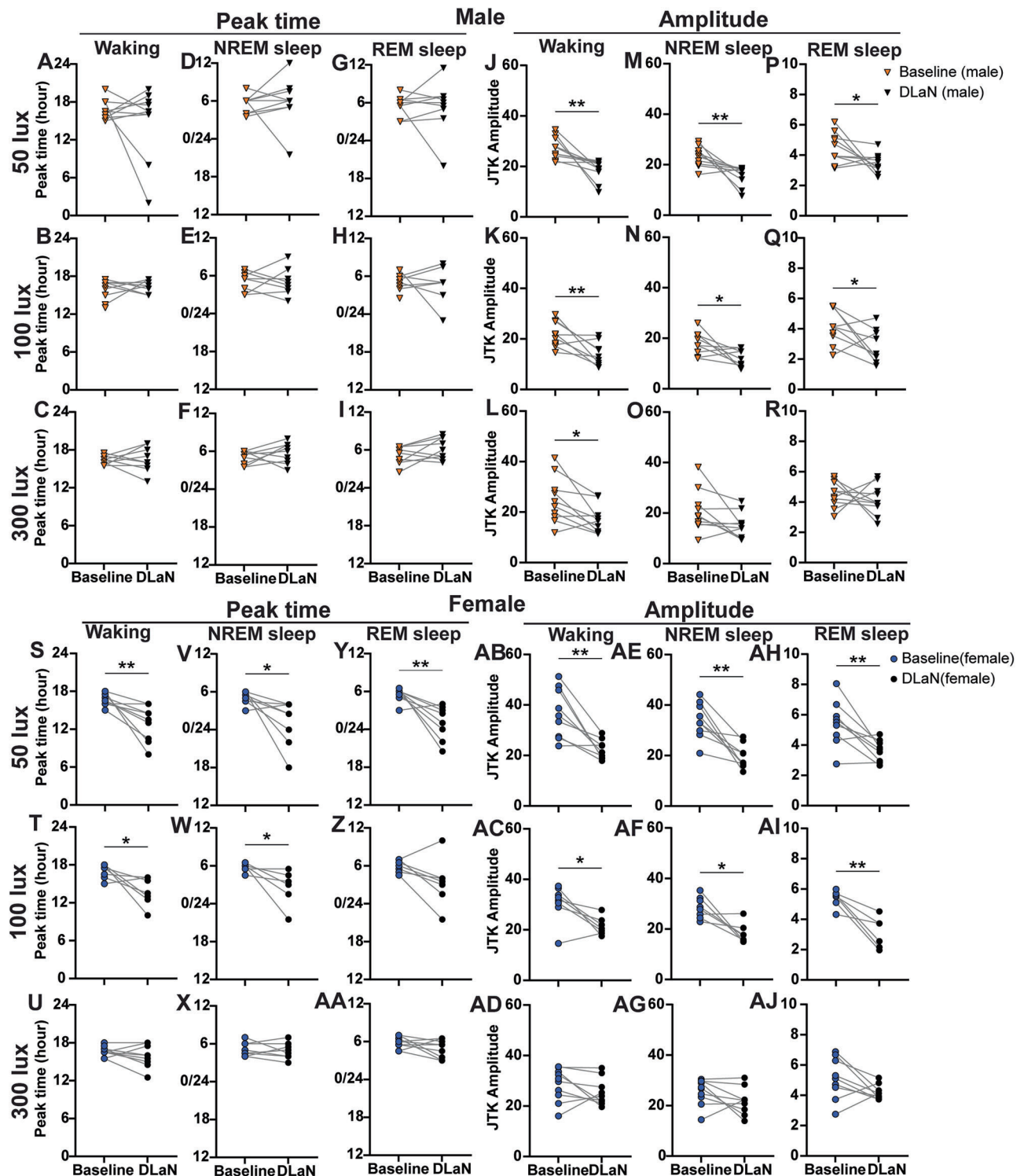


Fig. 6 | Peak timing and amplitude of wakefulness, NREM and REM sleep in male and female mice following 2 weeks of DLaN under different daytime light intensities. A–I Peak timing of wakefulness (A–C), NREM sleep (D–F), and REM sleep (G–I), identified using JTK-Cycle, in male mice exposed to 50 lux (A, D, G), 100 lux (B, E, H), or 300 lux (C, F, I) daytime light under baseline conditions (orange triangles) and after 2 weeks of DLaN exposure (black triangles). J–R Amplitude of wakefulness (J–L), NREM sleep (M–O), and REM sleep (P–R), identified using JTK-Cycle, in male mice exposed to 50 lux (J, M, P), 100 lux (K, N, Q), or 300 lux (L, O, R) daytime light under baseline conditions (orange triangles) and after 2 weeks of DLaN exposure (black triangles). S–AA Peak timing of wakefulness (S–U), NREM sleep (V–X), and REM sleep (Y–AA), identified using JTK-Cycle, in female mice exposed to 50 lux (S, V, Y), 100 lux (T, W, Z), or 300 lux (U, X, AA) daytime

light under baseline conditions (blue circles) and after 2 weeks of DLaN exposure (black circles). AB–AJ Amplitude of wakefulness (AB–AD), NREM sleep (AE–AG), and REM sleep (AH–AJ), identified using JTK-Cycle, in female mice exposed to 50 lux (AB, AE, AH), 100 lux (AC, AF, AI), or 300 lux (AD, AG, AJ) daytime light under baseline conditions (blue circles) and after 2 weeks of DLaN exposure (black circles). Asterisks indicate significant differences assessed by using a two-tailed paired *t*-tests or a two-tailed Wilcoxon matched pairs signed-rank tests (**p* < 0.05, ***p* < 0.01). Detailed *P* values for each panel are provided in Supplementary Table 6. Sample sizes: 50 lux, *n* = 10 males and *n* = 9 females; 100 lux, *n* = 10 males and *n* = 8 females (DLaN: 7 females); 300 lux, *n* = 10 males and *n* = 10 females (DLaN: 9 males and 9 females). Data are shown as individual values.

phase but no delayed onset, revealing distinct strategies of circadian reorganization across sexes.

Both sexes displayed a loss of anticipatory wakefulness prior to light-on, a pattern reminiscent of human studies linking nighttime light exposure to delayed sleep timing, reduced morning alertness, and melatonin suppression²⁵. Importantly, these sex-specific effects were strongly modulated by daytime light intensity: brighter daytime light largely protected females from phase shifts and amplitude dampening, while males were less sensitive to daytime light, maintaining reduced amplitude but without phase alterations. Together, these findings indicate that female sleep-wake regulation is particularly sensitive to the combined effects of nighttime and daytime light, whereas males respond more uniformly with amplitude reduction. Acute DLaN rapidly perturbs sleep continuity in both sexes, while chronic exposure seems to engage circadian mechanisms that reshape sleep-wake organization in a sex- and light-intensity-dependent manner. This unified framework explains why sex differences in sleep-wake behavior are inconsistently observed across rodent studies and highlights daytime light as a critical contextual factor for circadian resilience and vulnerability.

Sleep pressure under acute and chronic exposure

Acute and chronic DLaN exert different effects on EEG SWA with pronounced sex differences. A single night of DLaN did not produce robust time-dependent changes in NREM sleep SWA in either sex, indicating that acute nighttime light exposure is insufficient to reorganize the temporal structure of SWA. Although modest main effects of DLaN on SWA were observed in both male and female mice, these effects were absent under brighter daytime light, suggesting that sufficient daytime illumination buffers against acute DLaN-induced increased sleep pressure. Overall, acute DLaN data indicates that SWA is relatively resilient to short-term nighttime light exposure, particularly under brighter daytime light. In contrast, chronic DLaN revealed pronounced sex differences in slow-wave sleep regulation. After two weeks of DLaN exposure, males exhibited limited alterations in SWA timing that were restricted to moderate daytime light, whereas females showed robust, time-dependent changes in SWA across all daytime light intensities, particularly during the light phase under dim daytime conditions. Because rapid changes in suprachiasmatic nucleus (SCN) neuronal firing are temporarily correlated with SWA²⁶, these findings suggest that chronic DLaN may alter circadian output signals that regulate sleep, particularly in females.

Human laboratory and real world studies provide important context. Acute exposure to dim light at night reduces slow-wave sleep and increases arousal, and lighter sleep stages in healthy volunteers²⁷. Similar disruptions occur in night-shift workers who frequently experience poor sleep quality and difficulty maintaining consolidated daytime sleep due to circadian misalignment²⁸. Nighttime light exposure during sleep and shift work are both associated with reductions in SWA, a hallmark of deep, restorative sleep, has been linked to impairments in learning and memory²⁹ and increased risk of metabolic disease³⁰. Together, our findings identify SWA as a sensitive marker of nighttime light exposure, particularly in females, and highlight daytime light intensity as a critical factor shaping vulnerability to sleep disruption in modern lighting environments.

Light intensity matters both day and night: brighter day and dimmer night

The question whether brighter daytime light can protect sleep under DLaN conditions is broadly relevant. The effects of daytime illumination on circadian organization and sleep have been examined across several species. In diurnal Nile grass rats (*Arvicanthis niloticus*), bright daytime light strengthened sleep-wake rhythms, increased c-Fos expression in the anterior paraventricular thalamus, a key relay between the SCN and arousal networks^{8,21}. One-hour bright light exposure at beginning of the day has also improve sleep-wake rhythms while enhancing central orexin signaling³¹. Similarly, in diurnal rodent *Rhabdomys pumilio*, brighter daytime light increased the amplitude of behavioral, physiological, and SCN electrophysiological rhythms, indicating that daytime irradiance can strengthen

diurnal oscillations¹¹. Comparable effects have been observed in non-human primates, where brighter daytime light increased the amplitude of the core body temperature rhythms in Japanese macaques³². Together, these findings support the concept that brighter daytime light strengthens circadian output and arousal-promoting pathways, potentially increasing the robustness of the circadian system and reducing its vulnerability to disruptive effects of nighttime light.

DLaN has been shown to induce maladaptive behavioral and physiological outcomes, including reduced sleep efficiency, depressive- and anxiety-like behaviors, metabolic dysfunction and cognitive impairment^{33–35}. Large-scale human studies further demonstrate that exposure to bright nights combined with insufficient daytime light is associated with circadian disruption and increased health risks, including elevated mortality^{10,14,36}. Conversely, greater contrast between bright days and dark nights is associated with earlier and more stable sleep timing¹⁸. Together, these observations suggest that sufficient daytime light exposure may buffer the negative consequences of nighttime light.

Our findings provide direct experimental support for this idea. Brighter daytime light attenuated the acute sleep-wake disruption induced by first night DLaN in both sexes, with particularly strong protection in females. Under chronic DLaN (10 lux) conditions, male mice exhibited increased total NREM sleep and reduced wakefulness across all daytime lighting conditions, a result that contrasts with previous studies reporting minimal effects of 5 lux DLaN in mice and rats^{4,17,24,37}. This discrepancy likely reflects the slightly higher nighttime light intensity used here. Consistent with this interpretation, our prior work showed that male and female wildtype mice exposed to 5 lux DLaN under 300 lux daytime lighting did not exhibit significant changes in sleep after six weeks of exposure⁴.

Together, these findings suggest a threshold-like relationship between nighttime light intensity and sleep-wake regulation: very low levels of nighttime illumination may be insufficient to disrupt overall sleep-wake balance, whereas modestly higher levels are sufficient to alter sleep architecture. More broadly, our results support the concept that brighter days combined with darker nights promote sleep and circadian health.

Mechanistic considerations

The mechanisms underlying this sex-specific circadian vulnerability to light at night are likely multifactorial and may arise at several levels of the circadian system. One possibility is differential sensitivity to photic input pathways, including sex-dependent modulation of retinal signaling, melanopsin-expressing intrinsically photosensitive retinal ganglion cells (ipRGCs), or downstream processing within the SCN. Consistent with this possibility, a recent bioRxiv preprint reported higher melanopsin expression per ipRGC across the day in female mice compared with male mice, suggesting that sex differences in melanopsin signaling may contribute to sex differences in light-driven circadian behavior³⁸. Alternatively, sex differences may emerge downstream of the core molecular clock, at the level of circadian output pathways that regulate sleep-wake timing, slow-wave activity, and arousal thresholds. Our observation that males primarily exhibit dampened daily amplitude whereas females show pronounced phase advances supports the idea that chronic DLaN engages distinct regulatory nodes in the two sexes. The marked reorganization of SWA timing in females further suggests altered coupling between circadian phase and sleep homeostasis, rather than a simple change in sleep pressure. Importantly, the protective effect of brighter daytime light suggests that enhanced daytime photic drive may stabilize circadian output signals, thereby buffering against nocturnal light-induced disruption, particularly in females. In contrast, the effects of acute DLaN appear to primarily reflect the direct effects of nighttime light on sleep regulation.

Limitations

Several limitations of this study should be considered. First, we focused on C57BL/6J mice under daytime light intensities commonly used in laboratory settings, circadian and sleep responses to light can vary across strains, and C57BL/6J mice do not synthesize melatonin. Given that light at night

can suppress melatonin secretion, future studies should extend these findings to additional strains and species, including melatonin-proficient rodents and diurnal species. This is particularly important for translation, as light at night suppresses melatonin onset in humans and has been associated with decreased slow-wave activity^{27,39}, whereas we observed increased slow-wave activity in nocturnal mice. Studies in diurnal, melatonin-proficient species will therefore be important for determining whether these effects generalize more directly to humans. Second, we did not explicitly control for the estrous cycle stage which could contribute to variability in female responses. Because females spend approximately 20–25% of time in proestrus, EEG recordings obtained without estrous staging are more likely to reflect non-proestrus phases. The robustness and consistency of the observed finding across individuals suggest that estrous-related variability alone is unlikely to account for the magnitude of the effects. Incorporating estrous cycle monitoring in future studies will be important for more accurately assessing sex-specific responses to daytime light intensity and DLan. Third, our study was conducted under stable light-dark conditions, and different results may emerge under conditions of circadian misalignment, jet lag, or constant light. Finally, while our data implicate circadian reorganization as a key mechanism underlying chronic DLan effects, direct measurements of SCN molecular rhythms and downstream neural circuits will be required to define the precise loci of sex-specific vulnerability.

Conclusions

Together, these findings identify daytime light intensity as a critical but underappreciated determinant of sex differences in sleep-wake regulation and vulnerability to light at night. By systematically varying daytime illumination within the range commonly used in laboratory environments, we demonstrate that sex differences in sleep-wake behavior are not fixed properties but emerge from interactions between biological sex, circadian regulation, and environmental light context. Brighter daytime light not only minimizes baseline sex differences but also protects against both acute and chronic DLan-induced sleep disruption, particularly in females. These results provide a unifying framework to reconcile inconsistencies in the rodent sleep literatures and highlight the importance of reporting and controlling daytime light intensity in circadian and sleep studies. More broadly our findings underscore the potential for daytime light exposure to shape resilience or vulnerability to nocturnal light pollution, with implications for understanding sex-specific sleep and circadian disturbances in modern light environments.

Methods

Ethical approval

All animal procedures were approved by the University of California, Los Angeles Animal Research Committee (ARC; Protocol No. ARC-2020-009) and were performed in accordance with institutional guidelines and the Guide for the Care and Use of Laboratory Animals. A completed ARRIVE Essential 10 checklist is provided in the supplementary file.

Animals and housing conditions

11 weeks old C57BL/6J wild-type mice (30 female, 30 male) and were used in this study. Sample size was determined a priori based on the effect sizes observed in our previous study investigating the effects of dim light at night on sleep-wake architecture⁴. C57BL/6J wild-type mice were obtained from the Jackson Laboratory (ME, USA). Mice were housed in light-tight ventilated cabinets in temperature- and humidity-controlled conditions, with free access to food and water.

Surgical procedure

At the age of 12 weeks, animals were anesthetized using ketamine/xylazine (100/8.8 mg/kg, intraperitoneal injection) and EEG/ electromyography (EMG) electrodes were implanted as described previously⁴. A prefabricated head mount (CAT:8201, Pinnacle Technologies, KS) was used to position four stainless-steel epidural screw electrodes. One front electrode was placed 1.5 mm anterior to bregma and 1.5 mm lateral to the central line, whereas

the second two electrodes (interparietal—located over the visual cortex and common reference) were placed 2.5 mm posterior to bregma and 1.5 mm on either side. A fourth screw served as a ground. Electrical continuity between the screw electrode and head mount was aided by silver epoxy. EMG activity was monitored using stainless-steel Teflon-coated wires that were inserted bilaterally into the nuchal muscle. The head mount (integrated 2 × 3 pin grid array) was secured to the skull with dental cement.

Experimental design and recording setup

60 mice were randomly divided into one of the three daytime lighting conditions (50, 100 and 300 lux), and 0 lx during the night (Supplementary Fig. 6A). Illuminance was measured at the base of the cage using a calibrated light meter with the sensor facing upward toward the ceiling in an open-top transparent EEG cage. The melanopic equivalent daylight illuminance (EDI) values corresponding to four lighting conditions was reported in Supplementary Fig. 6B. All mice were first entrained to a normal lighting cycle: 12 h light/12 h dark (LD) before the surgery for 1 week. After the surgery, these mice recovered under these 3 different daylight conditions for two weeks, 3 female mice died during the surgery, two female mice from 100lux group and 1 female mouse from 50lux group. Mice were connected to a lightweight tether attached to a low-resistance commutator mounted over the cage (Pinnacle Technologies, KS) as previously described. Mice were allowed a minimum of 2 additional days to acclimate to the tether and recording chamber. Subsequently, a baseline day was recorded, starting at lights on zeitgeber time 0. After the baseline recording, mice were exposure to a 10lux dim light at night for one day, was recorded as 1st night DLan. And these mice were continued with two weeks of DLan, the mice were attached to the commutator for the post 2 weeks DLan recording for 24 h (Supplementary Fig. 6C).

EEG data acquisition, sleep scoring and EEG power spectrum analysis

Data acquisition was done by Sirenia Acquisition software (Pinnacle Technologies, KS) as previously described⁴. EEG signals were low pass filtered with a 40 Hz cutoff and collected continuously at a sampling rate of 400 Hz. All data were recorded simultaneously in 1 s epoch. Wake, NREM, and REM sleep were determined visually. Wake was scored when there were a high-EMG and low-EEG amplitude and high theta activity (EEG power density in the theta band, 6.0–9.0 Hz), concomitant with irregular, high EMG values. NREM sleep was scored when there was a low-EMG and higher EEG amplitude compared with wake and high SWA. REM sleep was scored when the EMG and EEG amplitude was low, and high theta activity was visible in the EEG.

Movement artifacts were excluded for power spectral and slow-wave analysis. For the spectral analysis, complete and clean recordings are needed to enter the analysis. However, this was not possible in six animals, which were therefore excluded from the analysis of the absolute EEG power density spectra and SWA in NREM sleep. Spectral analysis was performed using fast Fourier transform (FFT; 0.1–40 Hz, 0.1 Hz resolution); the absolute power density spectra of NREM sleep in baseline and post DLan days were analyzed.

Three vigilance states (wake, NREM, and REM sleep) were scored offline in 10-s epochs by the Sirenia program. The manual scoring of vigilance states based on the EEG and EMG recordings was performed according to standardized criteria for mice. Two female mice and one male mouse in the DLan group were excluded from the EEG analysis due to insufficient recording quality. The average amount of the vigilance states (wake, NREM sleep, and REM sleep) and EEG spectral data and NREM sleep SWA were analyzed in 1 h, 12 h and 24 h intervals.

Bouts, episode duration and transitions analysis

Episode duration averages were compiled by the Sirenia Sleep Pro program, which uses a conservative algorithm for bout lengths requiring sustained changes in arousal state to switch state. A bout was defined as 3+ continuous epochs, and the occurrence of 3+ continuous epochs of the different stage

indicated the end of the current bout. Episode duration and bout number were analyzed in 12 h intervals. To evaluate state transitions, we used custom-written Pascal scripts to determine transitions between different vigilance states. A transition was included in the analysis when immediately before the transition at least 30 consecutive seconds (3 scored epochs) were of the corresponding first vigilance state, and immediately after the transition also at least 30 consecutive seconds of the second correspond vigilance state were found.

Spectral EEG analysis

To investigate the effect of DLaN on EEG power density of NREM sleep, we analyzed the relative EEG power density in the slow-wave range (SWA, 0.5–4.0 Hz) in NREM sleep as described previously⁴. Since for the slow wave activity analysis, it was necessary to re-calculate power density values relative to the control condition, complete and clean recordings were needed from all animals for both conditions to enter the analysis. Unfortunately, this was not possible in 3 male animals under DLaN, which were therefore excluded from the analysis of SWA.

Peak timing and amplitude analysis

Circadian peak timing and amplitude of vigilance states were analyzed using JTK_CYCLE, an efficient nonparametric algorithm for detecting rhythmic components in time-series datasets. This approach was applied to determine peak time and amplitude parameters for each vigilance state, allowing within-animal comparisons between baseline and DLaN conditions. Analyses were performed using Nitecap^{40,41}.

Statistical analysis

All statistical analyses were conducted using GraphPad Prism 10 (GraphPad Software). Hourly (1-h) data in Fig. 1 were analyzed using either mixed-effects models with restricted maximum likelihood (REML) or two-way repeated-measures ANOVA (repeated zeitgeber time). Hourly (1-h) data in Figs. 3–5 were analyzed using either mixed-effects models (REML) with the Geisser–Greenhouse correction or two-way repeated-measures ANOVA with the Geisser–Greenhouse correction (repeated in zeitgeber time and 1st night DLaN or DLaN) followed by Bonferroni multiple-comparisons test. All tests were two-sided with $\alpha = 0.05$. For 12-h and 24-h summary data, two-way ANOVA or mixed-effects models (REML) were used to assess the effects of sex, DLaN exposure, or daytime light intensity. Normality was assessed using the Anderson–Darling test, or the Shapiro–Wilk test when sample size was less than eight. When data were normally distributed, two-sided paired Student's *t* tests were used; otherwise, two-sided nonparametric Wilcoxon matched-pairs signed-rank test was applied to compare baseline and DLaN conditions for peak timing and amplitude. Detailed statistical results, including sample sizes and exact *p* value for each panel, are provided in Supplementary Tables 1–6.

Data availability

All data analyzed during this study are included in the manuscript and supporting files. Source data for Figs. 1–6 in the manuscript is in Supplementary Data 1.

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Author contributions

Conceptualization, Y.W., T.D. and C.S.C.; funding acquisition, K.N.P. and G.D.B.; experiment conduction, Y.W. and C.T.C.; data analyzing: Y.W. and T.D.; writing – original draft, Y.W., writing – review and editing, all authors.

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Competing interests

The authors declare no competing interests.

Additional information

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Correspondence and requests for materials should be addressed to Ketema N. Paul or Christopher S. Colwell.

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