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Day and night light exposure are associated with psychiatric disorders: an objective light study in >85,000 people

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Circadian rhythm disturbance is a common feature of many psychiatric disorders. Light is the primary input to the circadian clock, with daytime light strengthening rhythms and night-time light disrupting them. Therefore, habitual light exposure may represent an environmental risk factor for susceptibility to psychiatric disorders. We performed the largest to date cross-sectional analysis of light, sleep, physical activity, and mental health (n = 86,772 adults; aged 62.4 ± 7.4 years; 57% women). We examined the independent association of day and night-time light exposure with covariate-adjusted risk for psychiatric disorders and self-harm. Greater night-time light exposure was associated with increased risk for major depressive disorder, generalized anxiety disorder, PTSD, psychosis, bipolar disorder, and self-harm behavior. Independent of night-time light exposure, greater daytime light exposure was associated with reduced risk for major depressive disorder, PTSD, psychosis, and self-harm behavior. These findings were robust to adjustment for sociodemographics, photoperiod, physical activity, sleep quality, and cardiometabolic health. Avoiding light at night and seeking light during the day may be a simple and effective, non-pharmacological means of broadly improving mental health.

Healthy circadian rhythms are essential for mental health and well-being[1]. Many psychiatric disorders are characterized by disrupted circadian rhythms and sleep[2,3]. In humans, a central circadian (~24-hour) clock in the suprachiasmatic nuclei (SCN) of the hypothalamus regulates the timing of basic cellular functions[4], physiology, cognition, and behavior[5,6]. Rhythms within the SCN are regulated by daily light-exposure patterns. This biological system evolved under predictable conditions of bright light during the day and darkness at night to ensure stable, robust rhythms[7–9]. Humans in modern, industrialized societies challenge this biology, spending ~90% of the day indoors under electric lighting[10], which is dim during the day and bright at night compared with natural light/dark cycles[11]. Deviations from our natural

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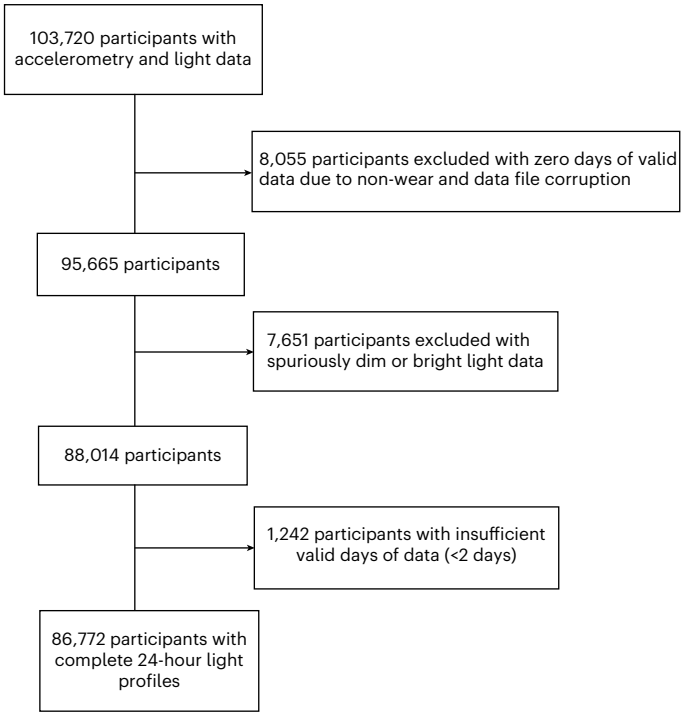

Fig. 1 | Flow diagram of the light-data study sample. Of the 103,720 participants that completed the seven-day actimetry and light monitoring assessment, 86,772 met the quality control criteria.

light/dark cycle lead to disrupted circadian rhythms and therefore could contribute to adverse psychiatric outcomes.

In this Article, we report the largest sample to date (n = 86,772) of objective recordings of individual, 24-hour light-exposure data obtained during seven days of actimetry and light monitoring. We investigated the association of day and night-time light with a constellation of psychiatric disorders that feature circadian rhythm disturbance, while controlling for confounding variables in multivariable models. We tested two primary hypotheses: (1) greater light exposure in the day is associated with lower risk for psychiatric disorders and better mood and (2) greater light exposure at night is associated with higher risk for psychiatric disorders and poorer mood. These hypotheses were motivated by the known effects of day and night-time light exposure on the human circadian system and the well-established links between circadian disruption and psychiatric disorders.

Results

Of the 103,720 UK Biobank participants that completed the seven-day actimetry and light monitoring assessment, we excluded those with poor-quality or unreliable accelerometry, sleep, and light data (detailed in Supplementary Methods and Fig. 1), leaving 86,772 participants. Of these, 86,631 had complete data for day and night-time light as well as the covariates in our fully adjusted Model 3. The mean and median daytime light exposures in the sample were 1,380.1 lux (standard deviation (s.d.) = 1,187.7) and 1,012.1 lux (interquartile range (IQR) = 1,578.1), respectively. The mean and median night-time light exposures in the sample were 24 lux (s.d. = 123.9) and 1.44 lux (IQR = 10), respectively. Mean and median lux values across quartiles of day and night-time light exposure are presented in Supplementary Table 1. The 24-hour light-exposure profile is presented in Supplementary Fig. 1. Characteristics of the sample and their missingness are presented in Table 1 for the lowest (Q1 and Q2) and highest (Q3 and Q4) quartiles of day and night-time light exposure. The light measurements showed very good reliability (intra-class correlation = 0.82 (95% confidence interval (CI): 0.81–0.83; Supplementary Methods) and a validation of the AX3 devices demonstrated they were able to accurately recover true lux after calibration for both low (r^2 = 0.82) and high (r^2 = 0.81) light levels (Supplementary Methods and Supplementary Fig. 2). The analytic sample comprised participants who completed both the mental health questionnaire (MHQ) and actigraphy assessment, and sample sizes varied depending on the outcome variable assessed, ranging from 26,956 to 61,442 for Model 1 and 26,824 to 61,147 in Model 3. Summary statistics for outcome variables derived from the MHQ are detailed in Supplementary Tables 2 and 3.

In fully adjusted regression models (Model 3, adjusted for age, sex, ethnicity, photoperiod, employment, and physical activity; Fig. 2 and Table 2), higher night-time light exposure was associated with higher odds of major depressive disorder (MDD) (χ^2 $P < 5 \times 10^{-16}$; fourth quartile odds ratio (OR_{Q4}) = 1.30, 95% CI = 1.23–1.38), self-harm (χ^2 $P = 1.52 \times 10^{-6}$; OR_{Q4} = 1.27, 95% CI = 1.14–1.42), generalized anxiety disorder (GAD) (χ^2 P = 0.0001; OR_{Q4} = 1.23, 95% CI = 1.11–1.36), post-traumatic stress disorder (PTSD) (χ^2 $P = 2.23 \times 10^{-10}$; OR_{Q4} = 1.34, 95% CI = 1.22–1.48), and psychosis (χ^2 P = 0.0009; OR_{Q4} = 1.21, 95% CI = 1.09–1.34). There was no overall association with bipolar disorder (χ^2 P = 0.18); however, those in the brightest night-time light quartile had 1.20 (95% CI = 1.02–1.42) times higher risk. Greater night-time light exposure was associated with higher scores on the Patient Health Questionnaire-9 (PHQ-9; χ^2 $P < 5 \times 10^{-16}$; standardized β_{Q4} = 0.13, 95% CI = 0.11–0.15), Generalized Anxiety Disorder-7 (GAD-7; χ^2 $P = 7.58 \times 10^{-10}$; β_{Q4} = 0.07, 95% CI = 0.05–0.09), and PTSD Checklist-6 (PCL-6; χ^2 $P = 2.84 \times 10^{-11}$; β_{Q4} = 0.11, 95% CI = 0.08–0.14) and lower wellbeing scores (χ^2 $P < 5 \times 10^{-16}$; β_{Q4} = −0.11, 95% CI = −0.13 to −0.08; Fig. 3 and Supplementary Table 4). The results for Model 1 (unadjusted) and Model 2 (adjusted for age, sex, ethnicity, and photoperiod) were similar in terms of direction, strength, and significance to Model 3.

Higher daytime light exposure (Model 3; Fig. 2 and Table 3) was associated with lower odds of MDD (χ^2 $P = 2.28 \times 10^{-8}$; OR_{Q4} = 0.81, 95% CI = 0.76–0.87), self-harm (χ^2 P = 0.0001; OR_{Q4} = 0.76, 95% CI = 0.67–0.87), PTSD (χ^2 P = 0.01; OR_{Q4} = 0.82, 95% CI = 0.73–0.92), and psychosis (χ^2 $P = 5.79 \times 10^{-9}$; OR_{Q4} = 0.69, 95% CI = 0.61–0.79). There was no association of daytime light exposure with GAD (χ^2 P = 0.61; OR_{Q4} = 0.96, 95% CI = 0.85–1.09) or lifetime bipolar disorder (χ^2 P = 0.22; OR_{Q4} = 0.86, 95% CI = 0.70–1.05). Figure 3 and Supplementary Table 5 show that greater daytime light exposure was associated with lower scores on the PHQ-9 (χ^2 $P = 1.61 \times 10^{-11}$; β_{Q4} = −0.09, 95% CI = −0.12 to −0.07), GAD-7 (χ^2 $P = 3.36 \times 10^{-5}$; β_{Q4} = -0.05, 95% CI = −0.08 to −0.03), PCL-6 (χ^2 P = 0.002; β_{Q4} = −0.08, 95% CI = −0.12 to −0.04), and higher wellbeing scores (χ^2 $P = 4.45 \times 10^{-8}$; β_{Q4} = 0.08, 95% CI = 0.05–0.11). The results for Model 1 (unadjusted) and Model 2 (adjusted for age, sex, ethnicity, and photoperiod) were similar in terms of direction and significance to Model 3. However, ORs and betas tended to be smaller in Model 3 after the addition of physical activity and employment covariates.

Increased night-time light exposure was also associated with a greater number of co-occurring psychiatric disorders (χ^2 $P = 1.50 \times 10^{-6}$; fourth quartile incidence rate ratio (IRR_{Q4}) = 1.15, 95% CI = 1.10–1.21), while increased daytime light exposure was associated with fewer co-occurring psychiatric disorders (χ^2 $P = 7.67 \times 10^{-8}$; IRR_{Q4} = 0.86, 95% CI = 0.81–0.91; Supplementary Table 6).

Day and night-time light exposure did not interact in their association with any psychiatric outcomes or symptom severity scales (Supplementary Tables 7 and 8); however, increased daytime light exposure attenuated the association of night-time light exposure with poorer wellbeing (interaction β = 0.01, 95% CI = 0.00–0.02, P = 0.01).

A series of sensitivity analyses were completed. First, we re-ran Model 3 regressions excluding participants who reported doing shift work (n = 6,840, 7.9%). The results for both psychiatric disorders and symptom severity scores were unchanged in this subsample (Supplementary Tables 9 and 10). In the subsample excluding shift workers, higher night-time light exposure was associated with higher

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Table 1 | Demographic characteristics of participants by low and high day and night-time light exposure

	Daytime light			Night-time light		
	Low Q1 and Q2; *N*=43,386	High Q3 and Q4; *N*=43,386	*P*	Low Q1 and Q2; *N*=43,387	High Q3 and Q4; *N*=43,385	*P*
Age						
Mean (s.d.)	62.0 (7.94)	62.7 (7.71)	$<5\times10^{-16}$*	62.6 (7.86)	62.1 (7.79)	$<5\times10^{-16}$*
Median [IQR]	62.9 [55.7, 68.4]	64.0 [57.0, 68.7]		63.8 [56.6, 68.8]	63.2 [56.1, 68.3]	
Sex						
Female	25,159 (58.0%)	24,321 (56.1%)	9.70×10^{-9}*	25,352 (58.4%)	24,128 (55.6%)	$<5\times10^{-16}$*
Male	18,227 (42.0%)	19,065 (43.9%)		18,035 (41.6%)	19,257 (44.4%)	
Body mass index						
Mean (s.d.)	26.7 (4.57)	26.7 (4.47)	0.21	26.3 (4.31)	27.1 (4.68)	$<5\times10^{-16}$*
Median [IQR]	26.0 [23.6, 29.1]	26.1 [23.6, 29.0]		25.7 [23.3, 28.6]	26.4 [23.9, 29.5]	
Missing	102 (0.2%)	83 (0.2%)		83 (0.2%)	102 (0.2%)	
White ethnicity	41,679 (96.1%)	42,226 (97.3%)	$<5\times10^{-16}$*	42,233 (97.3%)	41,672 (96.1%)	$<5\times10^{-16}$*
Missing	150 (0.3%)	141 (0.3%)		136 (0.3%)	155 (0.4%)	
Employment						
No	15,912 (36.7%)	17,412 (40.1%)	$<5\times10^{-16}$*	17,353 (40.0%)	15,971 (36.8%)	$<5\times10^{-16}$*
Yes	27,474 (63.3%)	25,974 (59.9%)		26,034 (60.0%)	27,414 (63.2%)	
Shift work						
No	39,780 (91.7%)	40,152 (92.5%)	2.97×10^{-6}*	40,545 (93.4%)	39,387 (90.8%)	$<5\times10^{-16}$*
Yes	3,606 (8.3%)	3,234 (7.5%)		2,842 (6.6%)	3,998 (9.2%)	
Physical activity						
Mean (s.d.)	27.3 (7.89)	29.0 (8.16)	$<5\times10^{-16}$*	28.1 (7.97)	28.3 (8.17)	0.0003*
Median [IQR]	26.4 [21.9, 31.7]	28.1 [23.4, 33.6]		27.1 [22.6, 32.5]	27.3 [22.6, 32.8]	
Missing	39 (0.1%)	94 (0.2%)		60 (0.1%)	73 (0.2%)	
Photoperiod						
Mean (s.d.)	10.9 (2.87)	14.1 (2.58)	$<5\times10^{-16}$*	12.4 (3.06)	12.6 (3.28)	$<5\times10^{-16}$*
Median [IQR]	10.2 [8.47, 12.8]	14.8 [12.3, 16.4]		12.4 [9.58, 15.2]	12.8 [9.61, 15.9]	
Sleep duration						
Mean (s.d.)	7.56 (1.06)	7.58 (1.02)	0.004*	7.79 (0.95)	7.34 (1.07)	$<5\times10^{-16}$*
Median [IQR]	7.60 [6.95, 8.21]	7.61 [6.98, 8.22]		7.81 [7.23, 8.38]	7.37 [6.72, 8.00]	
Sleep efficiency						
Mean (s.d.)	0.88 (0.06)	0.88 (0.06)	2.74×10^{-7}*	0.89 (0.06)	0.88 (0.07)	$<5\times10^{-16}$*
Median [IQR]	0.89 [0.85, 0.92]	0.90 [0.86, 0.92]		0.90 [0.86, 0.93]	0.89 [0.85, 0.92]	
Urban residence						
Yes	36,703 (84.6%)	35,533 (81.9%)	$<5\times10^{-16}$*	35,687 (82.3%)	36,549 (84.2%)	1.66×10^{-15}*
Missing	430 (1.0%)	404 (0.9%)		423 (1.0%)	411 (0.9%)	

P values correspond to *t* tests for continuous variables and χ^2 tests for categorical variables. *Significant difference at the .05 level.

odds of MDD (χ^2 $P < 5\times10^{-16}$; fourth quartile odds ratio (OR_{Q4}) = 1.30, 95% CI = 1.23–1.38), self-harm (χ^2 $P = 2.31\times10^{-5}$; OR_{Q4} = 1.28, 95% CI = 1.14–1.44), generalized anxiety disorder (χ^2 $P = 4.31\times10^{-5}$; OR_{Q4} = 1.27, 95% CI = 1.14–1.41), PTSD (χ^2 $P = 4.63\times10^{-8}$; OR_{Q4} = 1.31, 95% CI = 1.18–1.45), and psychosis (χ^2 P = 0.009; OR_{Q4} = 1.19, 95% CI = 1.06–1.32). As in the preceding analysis, there was no overall association with bipolar disorder in this subsample (χ^2 P = 0.25); however, those in the brightest night-time light quartile had 1.19 (95% CI = 1.06–1.32) times higher risk. Greater night-time light exposure was also associated with higher scores on the PHQ-9 (χ^2 $P < 5\times10^{-16}$; standardized β_{Q4} = 0.13, 95% CI = 0.11–0.15), GAD-7 (χ^2 $P = 1.88\times10^{-9}$; β_{Q4} = 0.07, 95% CI = 0.05–0.09), and PCL-6 (χ^2 $P = 1.63\times10^{-9}$; β_{Q4} = 0.10, 95% CI = 0.07–0.14) and lower wellbeing scores (χ^2 $P < 5\times10^{-16}$; β_{Q4} = −0.10, 95% CI = −0.12 to −0.08). In the subsample excluding shift workers, increased daytime light exposure was associated with lower odds of MDD (χ^2 $P = 4.56\times10^{-7}$; OR_{Q4} = 0.82, 95% CI = 0.76–0.88), self-harm (χ^2 $P = 9.43\times10^{-5}$; OR_{Q4} = 0.75, 95% CI = 0.65–0.86), PTSD (χ^2 P = 0.02; OR_{Q4} = 0.82, 95% CI = 0.73–0.93), and psychosis (χ^2 $P = 2.47\times10^{-7}$; OR_{Q4} = 0.71, 95% CI = 0.62–0.81). There was also no association of daytime light exposure with GAD (χ^2 P = 0.49; OR_{Q4} = 0.93, 95% CI = 0.82–1.06) or lifetime bipolar disorder (χ^2 P = 0.38; OR_{Q4} = 0.87, 95% CI = 0.70–1.07). Finally, in the subsample excluding shift workers, greater daytime light exposure was also associated with lower scores on the PHQ-9 (χ^2 $P = 4.73\times10^{-11}$; β_{Q4} = −0.10, 95% CI = −0.12 to −0.07), GAD-7 (χ^2 $P = 5.50\times10^{-6}$; β_{Q4} = −0.05, 95% CI = −0.08 to −0.02), and PCL-6 (χ^2 P = 0.002; β_{Q4} = −0.08, 95% CI = −0.12 to −0.04) and higher wellbeing scores (χ^2 $P = 9.89\times10^{-7}$; β_{Q4} = 0.07, 95% CI = 0.05–0.10).

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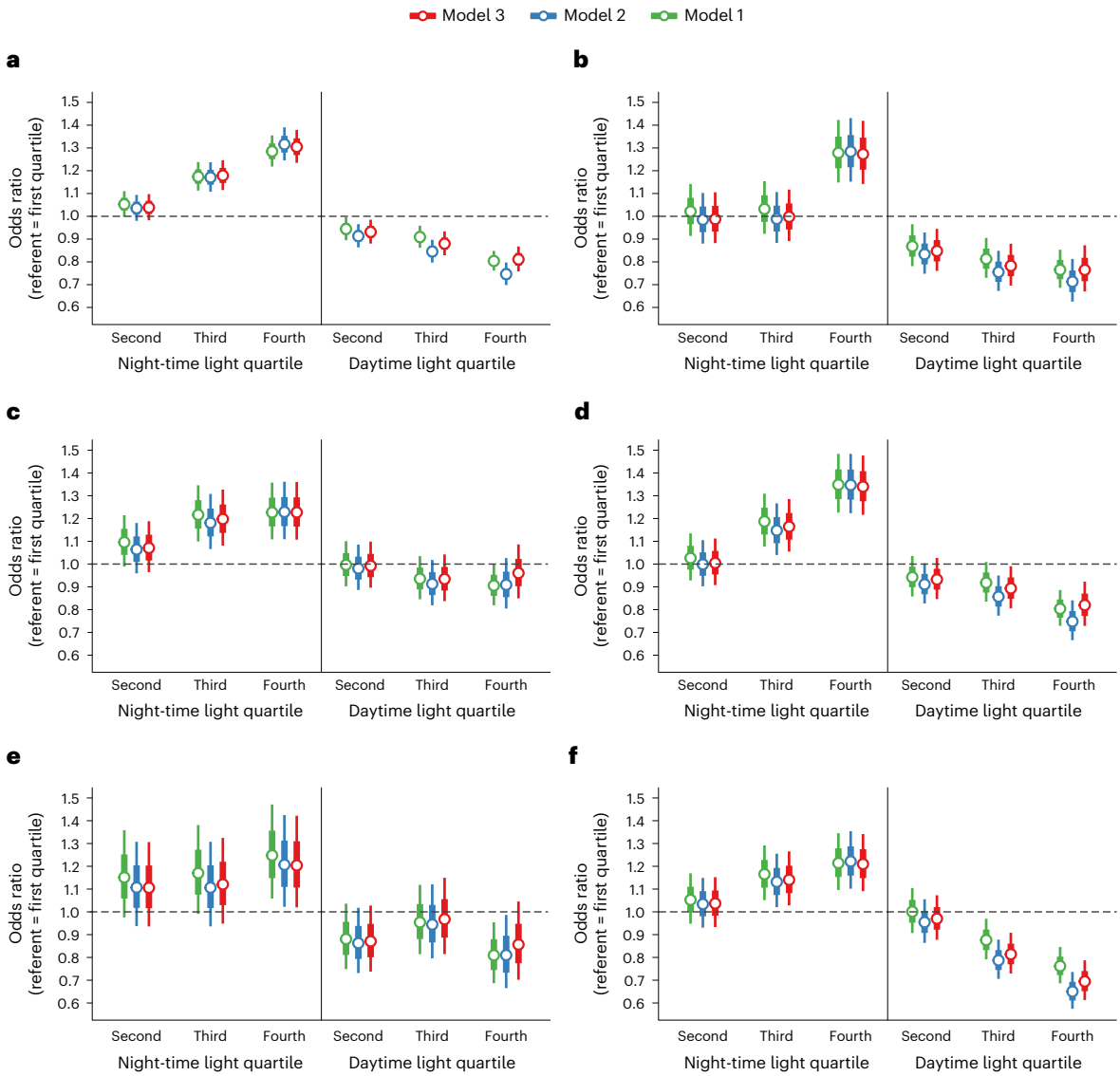

Fig. 2 | Associations of day and night-time light exposure with psychiatric disorders and self-harm. a–**f**, Coefficients plot for daytime and night-time light associations with **(a)** MDD (n_{cases} = 18,933; $n_{controls}$ = 25,509), **(b)** self-harm behavior (n_{cases} = 2,710; $n_{controls}$ = 58,670), **(c)** GAD (n_{cases} = 3,279; $n_{controls}$ = 46,554), **(d)** PTSD (n_{cases} = 3,535; $n_{controls}$ = 57,907), **(e)** bipolar disorder (hypomania/mania; n_{cases} = 1,192; $n_{controls}$ = 45,197), and **(f)** psychosis (n_{cases} = 3,110; $n_{controls}$ = 57,785). Coefficients represent the odds ratios ± the standard error of the mean (inner error bar) and 95% CI (outer error bar) for each quartile of day and night-time light exposure relative to the low light (Q1) referent. Three models are presented with increasing adjustment for confounders: Model 1 (green) is unadjusted, Model 2 (blue) adjusts for age, sex, ethnicity, and photoperiod, and Model 3 (red) additionally adjusts for employment and physical activity.

The second sensitivity analysis examined whether observed associations of day and night-time light with symptom severity scales and wellbeing were driven by the presence of clinical subgroups. We re-ran these analyses excluding participants with relevant disorders (Methods), adjusting for the same covariates in Model 3. The associations were unchanged excepting small variations in strength (Supplementary Table 11). The third sensitivity analysis examined whether the associations between light and psychiatric outcomes were independent of sleep characteristics. After adjustment for actigraphically derived sleep duration and sleep efficiency in addition to the Model 3 covariates (Model 4), the association of night-time light exposure with psychosis was no longer significant (χ^2 $P < 0.09$; OR_{Q4} = 1.12, 95% CI = 1.01–1.25). All other associations of day and night-time light with psychiatric outcomes and symptom severity scales were unchanged, although associations tended to be weaker (Supplementary Tables 12 and 13). In a fourth sensitivity analysis, we examined whether residential density as a measure of urbanicity could explain the light and mood relationship; we conducted an additional sensitivity analysis (Model 5) adjusting for residence type (urban versus rural) as well as the covariates in Model 3. Adjustment for urbanicity did not change any of the associations of day and night-time light with psychiatric outcomes and symptom severity scales in strength or significance (Supplementary Tables 14 and 15). The fifth sensitivity analysis examined whether factors relating to cardiometabolic health could explain the light and mood relationship. Adjusting for body mass index, systolic blood pressure, and diabetes status did not change the overall results; however, the association between daytime light and PTSD became weaker (Supplementary Tables 16 and 17).

Discussion

The present study observed that objectively measured light-exposure patterns under free-living conditions were associated with the risk for psychiatric disorders and the severity of mood symptoms. Brighter light at night was associated with a greater risk for MDD, self-harm

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Table 2 | Associations between night-time light exposure and psychiatric outcomes

Night light	Model 1 OR (95% CI)				Model 2 aOR (95% CI)				Model 3 aOR (95% CI)			
	Second quartile	Third quartile	Fourth quartile	χ^2 *P*	Second quartile	Third quartile	Fourth quartile	χ^2 *P*	Second quartile	Third quartile	Fourth quartile	χ^2 *P*
MDD	1.05 (1.00–1.11)	1.17 (1.11–1.24)*	1.28 (1.22–1.36)*	$<5\times10^{-16}$*	1.04 (0.98–1.09)	1.17 (1.11–1.24)*	1.32 (1.25–1.39)*	$<5\times10^{-16}$*	1.04 (0.98–1.10)	1.18 (1.12–1.25)*	1.30 (1.23–1.38)*	$<5\times10^{-16}$*
Self-harm	1.02 (0.91–1.14)	1.03 (0.92–1.15)	1.28 (1.15–1.42)*	6.77×10^{-6}*	0.99 (0.88–1.10)	0.99 (0.88–1.11)	1.28 (1.15–1.43)*	3.12×10^{-7}*	0.99 (0.88–1.11)	1.00 (0.89–1.12)	1.27 (1.14–1.42)*	1.52×10^{-6}*
GAD	1.10 (0.99–1.21)	1.22 (1.10–1.35)*	1.23 (1.11–1.36)*	9.98×10^{-5}*	1.06 (0.96–1.18)	1.18 (1.07–1.31)*	1.23 (1.11–1.36)*	0.0002*	1.07 (0.97–1.19)	1.20 (1.08–1.33)*	1.23 (1.11–1.36)*	0.0001*
PTSD	1.03 (0.93–1.13)	1.19 (1.08–1.31)*	1.35 (1.23–1.48)*	1.40×10^{-10}*	1.00 (0.90–1.11)	1.15 (1.04–1.27)*	1.35 (1.22–1.48)*	7.18×10^{-11}*	1.00 (0.91–1.11)	1.16 (1.05–1.29)*	1.34 (1.22–1.48)*	2.23×10^{-10}*
Bipolar disorder	1.15 (0.98–1.36)	1.17 (0.99–1.38)	1.25 (1.06–1.47)*	0.06	1.11 (0.94–1.31)	1.11 (0.94–1.31)	1.21 (1.02–1.43)*	0.18	1.11 (0.94–1.31)	1.12 (0.95–1.32)	1.20 (1.02–1.42)*	0.18
Psychosis	1.05 (0.95–1.17)	1.17 (1.05–1.29)*	1.21 (1.10–1.35)*	0.0005*	1.03 (0.93–1.15)	1.13 (1.02–1.26)*	1.22 (1.10–1.35)*	0.0004*	1.04 (0.93–1.15)	1.14 (1.03–1.27)*	1.21 (1.09–1.34)*	0.0009*

Logistic regression was used for all outcomes; ORs and adjusted ORs (aORs) are presented with their 95% confidence intervals for each ascending night-time light quartile relative to the low light (Q1) referent. Model 1 is unadjusted. Model 2 adjusted for age, sex, ethnicity, and photoperiod as a measure of seasonality. Model 3 is additionally adjusted for employment and physical activity. *Significance at the .05 level.

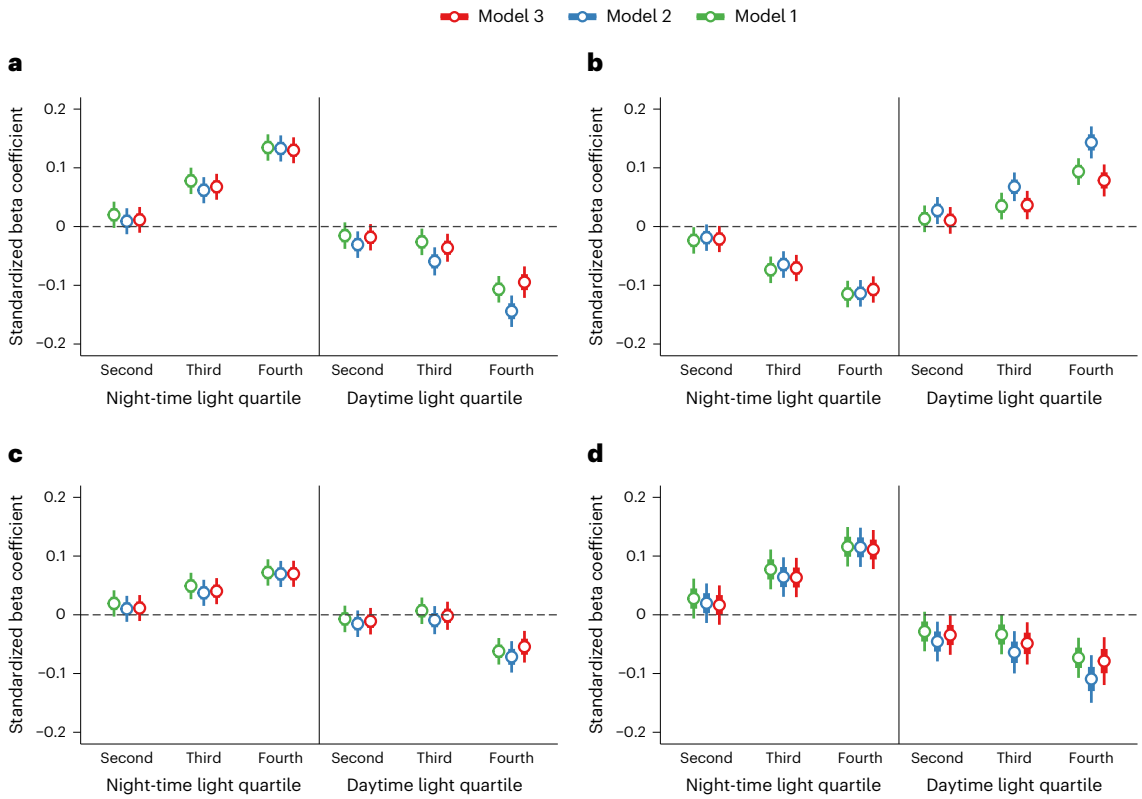

Fig. 3 | Associations of day and night-time light exposure with symptom severity scales and wellbeing. a–d, Coefficients plot for daytime and night-time light associations with **(a)** PHQ-9 score ($n = 60{,}556$), **(b)** wellbeing score ($n = 59{,}904$), **(c)** GAD-7 score ($n = 60{,}786$), and **(d)** PCL-6 score ($n = 26{,}956$). Coefficients represent the standardized betas ± the standard error of the mean (inner error bar) and 95% CI (outer error bar) for each quartile of day and night-time light exposure relative to the low light (Q1) referent. Three models are presented with increasing adjustment for confounders: Model 1 (green) is unadjusted, Model 2 (blue) adjusts for age, sex, ethnicity, and photoperiod, and Model 3 (red) additionally adjusts for employment and physical activity.

behavior, PTSD, psychosis, GAD, and bipolar disorder, as well as poorer self-reported mood and wellbeing. Conversely, brighter light in the day was associated with lower odds of MDD, self-harm behavior, PTSD, and psychosis, as well as better self-reported mood and wellbeing. Remarkably, these associations were independent and additive. For example, greater night-time light exposure was associated with increased odds of MDD even for those in the brightest daytime light quartile and, conversely, greater daytime light exposure was associated with reduced risk for MDD even amongst those in the brightest night-time light quartile. These associations were also independent of demographic, physical activity, photoperiod, and employment covariates. Sensitivity analyses showed these findings to be consistent when accounting for shift work, sleep quality, urbanicity, and cardiometabolic health.

For individuals in the brightest night-time light quartile, we observed ~30% higher risk of MDD and self-harm, while individuals in the brightest daytime light quartile had ~20% lower risk of MDD and self-harm. Night-time light exposure was also associated with poorer self-reported mood and wellbeing, while daytime light exposure was associated with better

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Table 3 | Associations between daytime light exposure and psychiatric outcomes

	Model 1 OR (95% CI)				Model 2 aOR (95% CI)				Model 3 aOR (95% CI)			
Daytime light	Second quartile	Third quartile	Fourth quartile	χ^2 P	Second quartile	Third quartile	Fourth quartile	χ^2 P	Second quartile	Third quartile	Fourth quartile	χ^2 P
MDD	0.94 (0.90–1.00)*	0.91 (0.86–0.96)*	0.80 (0.76–0.85)*	8.21×10^{-15}*	0.91 (0.86–0.97)*	0.85 (0.80–0.90)*	0.75 (0.70–0.80)*	$<5\times10^{-16}$*	0.93 (0.88–0.99)*	0.88 (0.83–0.93)*	0.81 (0.76–0.87)*	2.28×10^{-8}*
Self-harm	0.87 (0.78–0.97)*	0.81 (0.73–0.90)*	0.77 (0.69–0.85)*	9.73×10^{-6}*	0.83 (0.75–0.93)*	0.76 (0.67–0.85)*	0.71 (0.63–0.81)*	1.13×10^{-6}*	0.85 (0.76–0.94)*	0.78 (0.70–0.88)*	0.76 (0.67–0.87)*	0.0001*
GAD	1.00 (0.90–1.10)	0.94 (0.85–1.03)	0.91 (0.82–1.00)	0.14	0.98 (0.89–1.09)	0.91 (0.82–1.02)	0.91 (0.81–1.03)	0.28	0.99 (0.90–1.10)	0.93 (0.84–1.04)	0.96 (0.85–1.09)	0.61
PTSD	0.94 (0.86–1.04)	0.92 (0.83–1.01)	0.80 (0.73–0.89)*	0.0001*	0.91 (0.83–1.00)	0.86 (0.77–0.95)*	0.75 (0.67–0.84)*	2.26×10^{-5}*	0.93 (0.85–1.03)	0.89 (0.81–0.99)*	0.82 (0.73–0.92)*	0.01*
Bipolar disorder	0.88 (0.75–1.04)	0.95 (0.81–1.12)	0.81 (0.69–0.95)*	0.06	0.86 (0.73–1.02)	0.94 (0.80–1.12)	0.81 (0.67–0.99)*	0.10	0.87 (0.74–1.03)	0.97 (0.81–1.15)	0.86 (0.70–1.05)	0.22
Psychosis	1.00 (0.91–1.10)	0.88 (0.79–0.97)*	0.76 (0.69–0.85)*	7.69×10^{-8}*	0.95 (0.86–1.06)	0.79 (0.71–0.88)*	0.65 (0.57–0.74)*	2.82×10^{-12}*	0.97 (0.88–1.07)	0.81 (0.73–0.91)*	0.69 (0.61–0.79)*	5.79×10^{-9}*

Logistic regression was used for all outcomes, and ORs are presented with their 95% confidence intervals for each ascending daytime light quartile relative to the low light (Q1) referent. Model 1 is unadjusted. Model 2 adjusted for age, sex, ethnicity, and photoperiod as a measure of seasonality. Model 3 is additionally adjusted for employment and physical activity.
*Significance at the .05 level.

mood and wellbeing. Previous studies have reported an association of night-time light exposure with low mood[12], and one study has linked night-time light to MDD risk, although this study examined group-level outdoor light at night, which may not be a good proxy for individual ambient light exposure[13]. Other limitations of these studies were generally small sample sizes and poor control for confounders such as physical activity and sleep quality, and no studies considered the independent effects of both day and night-time light. Conversely, daytime light therapy has long been shown to be efficacious in treating depression[14] and has been shown to enhance treatment efficacy when combined with a selective serotonin reuptake inhibitor[15]. Fewer studies have linked free-living daytime light exposure to MDD risk, although one study linked self-reported time spent in outdoor light with lower risk[16]. Depression has long been associated with circadian disruption. Patients with depression have both delayed and low-amplitude circadian rhythms[17,18], which are reversed in recovery[19]. The severity of mood symptoms and the duration of depressive episodes are greater in those who experience circadian rhythm disturbance[17,20], and the presence of circadian rhythm disturbance in depression is predictive of recurrence[21]. Our findings are consistent with the known time-dependent effects of light on the properties of the circadian system, such that light at night tends to delay rhythms and reduce circadian amplitude, whereas early-morning and daytime light tends to advance rhythms and boost circadian amplitude[8,9,22,23]. Therefore, the euthymic effect of bright daytime light and dim night-time light exposure may occur by boosting the amplitude and advancing the timing of circadian rhythms, correcting the delayed and blunted rhythms seen in depression[17,18]. Seeking greater daytime light and minimizing night-time light exposure could be a simple means of improving depression trajectories by treating underlying circadian disturbance.

Greater light exposure at night was associated with higher risk for bipolar disorder. Bipolar disorder has long been associated with dampened amplitude of behavioral rhythms and more-variable circadian timing[24]. A recent study found that brighter night-time light exposure predicted manic/hypomanic episodes in patients with bipolar disorder[25]. and outdoor light at night has been associated with bipolar disorder[13]. Hypersensitivity of the circadian system to light at night has been proposed to be a trait marker of bipolar disorder[26]. Drugs used to treat bipolar disorder reduce the sensitivity of the circadian system to light[27], suggesting that reducing the effects of light at night on the circadian system may play a role in recovery. Consistent with this, night-time dark therapy and wearing blue-light-blocking glasses at night are effective at reducing mania in patients[28,29]. We did not see an association of bipolar disorder with daytime light exposure. This finding is new as the association of free-living daytime light exposure with manic symptoms in bipolar disorder and the efficacy of daytime light therapy on manic episodes in randomized controlled trial designs has not been examined[30]. This finding also contradicts previous case reports that suggested daytime light exposure was a risk factor for increased manic symptoms[31,32]. The avoidance of light at night specifically may be beneficial in mitigating risk for bipolar disorder.

We found both an adverse association of night-time light exposure and a beneficial association of daytime light exposure with PTSD risk and symptom severity. To our knowledge, no studies have examined the association of free-living light-exposure patterns in the day or night with PTSD risk. There is some evidence of disturbed circadian rhythms in PTSD. Delayed activity rhythms are associated with more-severe PTSD symptoms[33] while lower urinary melatonin rhythm amplitude after a trauma exposure predicts a higher risk for PTSD[34], a finding replicated in military personnel[35]. Our results suggest bright night-time and dim daytime light exposure may be antecedent factors leading to blunted and delayed rhythms. This is supported by evidence that daytime light therapy may be an effective treatment for PTSD symptoms[36]. Avoidance of light at night and seeking bright daytime light after trauma could reduce the risk of developing PTSD or the severity of symptoms in those with the disorder.

We observed an ~20% higher risk for GAD and increased GAD-7 scores among those in the highest quartile of night-time light exposure. While daytime light exposure did not associate with GAD, brighter daytime light exposure was associated with reduced GAD-7 scores. As yet, the literature on light exposure and anxiety in humans is limited and mixed, with some studies reporting a beneficial effect of daytime light exposure on anxiety and others reporting null effects[37,38]. Consistent with our findings, night-shift workers, who are chronically underexposed to daytime light and overexposed to night-time light, report elevated anxiety; however, this could also be driven by concomitant sleep disruption[39,40]. Together, these findings provide new evidence for an association of night-time light exposure with increased GAD risk and symptomology and some evidence for an effect of daytime light exposure on reducing GAD symptomology.

Finally, bright night-time light exposure was associated with ~20% increased risk for psychosis, while bright daytime light exposure was associated with ~30% reduced risk for psychosis. There are also little data linking free-living light exposure in the day or night to psychosis and psychotic disorders, despite sleep and circadian rhythm disruption being common features of patients on and off medication[1]. One small study found that patients with schizophrenia had lower daytime light exposure and in a natural experiment observed that boosting daytime light exposure could normalize the sleep and circadian disruption seen

in the disorder[41]. Studies of daytime light therapy and schizophrenia have reported mixed results, but these studies have been small and further, more rigorous, trials are needed[42,43]. In addition to daytime light, our findings point to night-time light as a new therapeutic target for psychosis.

Beyond effects on the circadian clock, non-visual photoreception is appreciated to have a direct effect on mood via projections to brain areas implicated in mood regulation. Light exposure acutely enhances both mood and alertness[44,45]. Intrinsically photosensitive retinal ganglion cells expressing the photopigment melanopsin are the primary input of light information to the circadian clock in the SCN[46]. These cells also project to the medial amygdala and lateral habenula, brain areas implicated in depression, and these projections mediate the acute euthymic effect of light exposure[47,48]. The direct effects of light may partially explain the association of daytime light with lower MDD and self-harm risk, although a mechanism for direct effects of daytime light on other disorders such as PTSD, psychosis, and bipolar disorder is unclear. As mood is generally poorer in the night/early-morning hours, people may seek out the acute euthymic effects of light at night. Although this may immediately improve mood, it would lead to circadian disruption in the long term and could perpetuate mental illness. This represents a potential challenge for promoting healthy light behaviors.

Taken together, our findings are consistent with bright daytime light and low night-time light strengthening circadian rhythms as an antecedent to more robust mental health. Patterns of bright daytime light and low night-time light serve to enhance the amplitude and stability of the circadian clock as well as align its timing appropriately with daily activities[7–9,22]. As modern humans spend ~90% of the day indoors[10], our light-exposure patterns are typically less bright in the day and more bright at night than naturalistic patterns across our evolutionary history[11]. Addressing this deviation from our natural light/dark cycles may improve the general mental health of people in industrialized societies.

This study has a number of important limitations. First, the findings we report are cross-sectional. While there are well-supported causal mechanisms linking bright night-time light and dim daytime light with circadian disruption, and linking circadian disruption with mental health, we acknowledge the possibility of reverse causation and that longitudinal studies will be needed to establish the temporality of the associations we observed. However, the robustness of our findings to adjustment for confounders, including physical activity and sleep, provides support for our interpretation. Second, light monitoring was performed using a wrist-worn device, which is not designed to measure light at the ocular level. The data therefore provide a coarse estimate of the actual effects of light on the circadian system. Third, the actigraphy data and outcome variables were not measured simultaneously, with the latter measured an average of 1.86 years later, and as such it is feasible that the habitual light-exposure patterns of participants could have changed in the interim. However, we note that we did observe good reliability of within-individual repeat light-exposure assessments completed over the course of a year, suggesting that light measurements at one timepoint are a good proxy for other timepoints. Fourth, we are unable to distinguish true darkness from device coverage, and this probably contributes to error variance in the dataset. Fifth, as the AX3 APDS9007 light sensor is calibrated to the human photopic spectral range, we cannot report melanopic equivalent daylight illuminance, which is optimal for the study of the non-visual effects of light. Finally, the lux values reported for the day and night-time light quartiles were derived from a validation of the AX3 devices against a lux photometer using a sample of the devices approximately eight years after the original study. However, we note that these devices were a very good predictor of true lux and that the calibrated lux values for day and night-time light exposure cohere with those reported in other studies[49–51].

This study reports an analysis of objectively measured light-exposure data from the UK Biobank and demonstrates its validity and reliability. This study is also the largest examination of objectively measured light exposure and mental health to date. Our findings demonstrate a consistent association of light-exposure patterns that are healthy for circadian rhythms with better psychiatric outcomes. These results suggest that light-exposure interventions may act in a transdiagnostic manner to improve mental health by strengthening circadian rhythms. Brighter days and darker nights may be a simple, freely available, non-pharmacological intervention to enhance mental health that is easily implementable in a community setting.

Methods

Study design and participants

In this cross-sectional study, we drew on the UK Biobank prospective general population cohort, which contains more than 502,000 UK residents recruited via National Health Service patient registers from 2006 to 2010. The study population is described in detail elsewhere[52,53]. Accelerometry and light data were measured in a subset of 103,720 participants in 2013–2015, and a separate subset of 157,366 participants completed an online MHQ in 2016–2017. Participants who accepted the invitation to join the UK Biobank cohort provided written, informed consent and were given reimbursement for travel expenses. The UK Biobank has generic ethical approval from the North West Multi-Center Research Ethics Committee (ref 11/NW/03820).

Measures

Light exposure. In 2013, 236,519 UK Biobank participants were invited to participate in a seven-day physical activity and light monitoring study. Of these participants, 103,720 (43.9%) accepted and returned the accelerometer to the UK Biobank. Participants who accepted the invitation received a wrist-worn AX3 triaxial accelerometer (Axivity) with in-built light sensor (APDS9007 silicon photodiode sensor; spectral sensitivity λ = 470–650 nm) and were asked to wear the device on their dominant wrist for seven days under free-living conditions.

Quality control and definition of light-exposure predictors was completed in R (version 4.1.0) as detailed in the Supplementary Methods. Briefly, the daily light profiles of participants meeting quality control criteria were entered into a factor analysis to identify independent patterns in light exposure in the sample. Factor analysis supported the extraction of day (7:30–20.30) and night (0:30–6:00) factors. Light exposure during the day had a small positive correlation with light exposure at night (r_s = 0.10, P < 0.0001). Due to large positive skew in both day and night-time light variables (skewness coefficient > 1 for both), both variables were converted into categorical predictors for analysis by dividing them into four quartiles of equal n in ascending brightness.

Psychiatric outcomes. A total of 339,092 UK Biobank participants were invited to complete the online MHQ in 2016, and 157,366 completed the questionnaire. Measurement of psychiatric outcomes as part of the MHQ took place an average of 1.86 years (s.d. = 0.66) after the actigraphy assessment. Of the 86,772 participants with complete light data, 61,466 (70.8%) completed the UK Biobank MHQ. Definitions of case/control psychiatric disorder outcomes from the MHQ are based on Composite International Diagnostic Interview and the *Diagnostic and Statistical Manual of Mental Disorders* 4th Edition[54] criteria and followed guidelines established by Davis et al.[55] Case/control outcomes were MDD, GAD, bipolar disorder (hypomania/mania), PTSD, psychosis, and self-harm. Detailed definitions are given in the Supplementary Methods. Continuous outcomes were symptom severity scales for depression (Patient Health Questionnaire, PHQ-9), anxiety (GAD-7), PTSD (PTSD Checklist-6, PCL-6), and an overall wellbeing score indexing euthymia and eudaemonia (Supplementary Methods).

Statistical analysis

The association between day and night-time light and case/control outcomes was examined with multiple logistic regression. Odds ratios and their 95% confidence intervals are reported. Multiple linear regression was used for the continuous symptom severity scales and wellbeing, with standardized beta values reported. These associations were tested hierarchically in three models with increasing adjustment for potential confounders. Each model included both day and night-time light categorical predictors to examine their independent effects. Likelihood-ratio χ^2 tests were used as an omnibus test of significance for the day and night-time light factors. Model 1 examined the unadjusted association between day and night-time light and psychiatric outcomes. Model 2 adjusted for age (at the time of actigraphy), sex, ethnicity (white versus non-white; data-field 21000), and photoperiod (as a measure of seasonality, defined as the duration between sunrise and sunset at the beginning of the actigraphy assessment). Finally, Model 3 additionally adjusted for employment (employed versus unemployed; data-field 6142) and physical activity (defined as overall acceleration average over the actigraphy period in milligravity units; data-field 90012). The physical activity variable was previously defined by the UK Biobank accelerometer expert working group[56]. An additional Poisson regression model adjusting for Model 3 covariates was run to examine the association of day and night-time light exposure with the number of co-occurring psychiatric disorders (range = 0 to 5), including MDD, PTSD, psychosis, bipolar disorder, and GAD. Incidence rate ratios and their 95% confidence intervals are reported for this model.

To assess the interaction between day and night-time light exposure on psychiatric outcomes and symptom severity scores, logistic and linear regressions using Model 3 covariates were run including the main effects and interaction term of $\log_{10}$-transformed day and night-time light exposure. Odds ratios per standard deviation of light exposure and standardized beta coefficients are reported with their 95% confidence intervals for the main effects and interaction terms of logistic and linear models, respectively.

A series of sensitivity analyses were completed to examine the robustness of the primary findings. The first sensitivity analysis examined whether the presence of shift workers in the sample (n = 6,840, 7.9%) was driving the observed associations. Model 3 regressions were re-run excluding those who reported doing shift work (work outside the "normal daytime working hours of 9am–5pm"; data-field 826) to any degree (sometimes, usually, or always). The second sensitivity analysis assessed whether observed associations of light with symptom severity scales and wellbeing were driven by clinical subgroups. We re-ran these analyses excluding participants with MDD for the PHQ-9 model; with GAD for the GAD-7 model; with PTSD for the PCL-6 model; and with MDD, GAD, PTSD, bipolar disorder, or psychosis for the wellbeing model. The third sensitivity analysis examined whether the relationship between light and psychiatric outcomes was independent of objectively measured sleep duration and efficiency (Model 4; Supplementary Methods). The fourth sensitivity analysis examined whether participants' residential population density as a measure of urbanicity could explain the light and mood relationship (Model 5). Urbanicity (data-field 20118; urban versus rural) was defined according to the UK Office for National Statistics population density classification of participant residential postcodes where urban postcodes have a population of 10,000 or more and rural postcodes have a population of less than 10,000. The fifth sensitivity analysis examined whether cardiometabolic health explained the light and mood relationship (Model 6). In addition to Model 3 covariates, systolic blood pressure (data-field 4080), body mass index (data-field 21001), and diabetes diagnosis (data-field 2443) were added as covariates. All statistical tests were two-sided, and inspection of residual plots confirmed the assumptions of linear models were met. Reporting of statistical analyses and results followed the STROBE guidelines.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The data used in this study are available in the UK Biobank resource subject to project approval by the UK Biobank Access Management Team. The authors of the present study are approved for access under application 6818.

Code availability

Data organization and statistical analysis were performed in R (version 4.1.0). Data analysis code is available at https://github.com/dpwindred/Axivity_AX3_Light.

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Author contributions

A.C.B., S.W.C., and A.J.K.P. conceptualized the research idea and acquired funding. All authors contributed to the methodology and development of the analysis plan. A.C.B., D.P.W., S.W.C., A.J.K.P., M.K.R., R.S., and J.M.L. acquired the data. A.C.B. completed the formal analysis, visualizations of the data, and wrote the original draft of the manuscript. All authors reviewed the manuscript and provided critical comments. All authors approved the final manuscript.

Competing interests

A.J.K.P. and S.W.C. have received research funding from Delos and Versalux, and they are co-founders and co-directors of Circadian Health Innovations PTY LTD. S.W.C. has also received research funding from Beacon Lighting and has consulted for Dyson. P.O. was a co-founder of Axivity Ltd and a director until 2015. C.V. is a board member of the Working Time Society and a research committee member for DiME. All other authors have no competing interests.

Additional information

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s44220-023-00135-8.

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Peer review information *Nature Mental Health* thanks Sarah Chellappa, Yuichi Esaki, Jamie M. Zeitzer, and the other, anonymous, reviewer(s) for their contribution to the peer review of this work.

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Reporting Summary

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Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

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☐	☒ A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
☐	☒ For null hypothesis testing, the test statistic (e.g. *F*, *t*, *r*) with confidence intervals, effect sizes, degrees of freedom and *P* value noted *Give P values as exact values whenever suitable.*
☒	☐ For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
☐	☒ For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
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Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection	Data was accessed via the UK Biobank Access Management System for which the authors are approved under project number 6818.
Data analysis	Data organization and analysis was completed in R-4.1.0. The 'GGIR' package was used to analyze activity data from the AX3 devices according to the pseudo-code described in the "Supplementary Methods: Accelerometry and sleep analysis". Light data was extracted from .cwa files and analyzed using in-built R functions according to the pseudo-code described in the "Supplementary Methods: Light Analysis" section and the code is available at https://github.com/dpwindred/Axivity_AX3_Light. The association of day and night light exposure with psychiatric outcomes and symptom severity scales was completed with in-built R functions ('lm'; 'glm'). Plotting of coefficients from resultant models was completed with the 'plot_summs' function from the 'jtools' package.

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Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
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The primary data set used in this study is available in the UK Biobank resource and access is subject to project approval by the UK Biobank Access Management Team.

Research involving human participants, their data, or biological material

Policy information about studies with human participants or human data. See also policy information about sex, gender (identity/presentation), and sexual orientation and race, ethnicity and racism.

Reporting on sex and gender	Sex was not considered an inferential variable in this study. We report descriptive statistics for sex in Table 1 and adjust for sex in statistical models.
Reporting on race, ethnicity, or other socially relevant groupings	Ethnicity was not considered an inferential variable in this study. We report descriptive statistics for ethnicity in Table 1 and adjust for ethnicity in statistical models.
Population characteristics	A total of 86,772 participants from the UK Biobank cohort study that completed a week of optional accelerometry (57% female; aged 62.4 ± 7.4 years).
Recruitment	Participants were recruited via the UK National Health Service (NHS) patient registers from 2006 to 2010. Questionnaire items may be subject to recall and response bias.
Ethics oversight	Participants provided written, informed consent and the UK Biobank has ethical approval from the North West Multi-Center Research Ethics Committee (ref 11/NW/03820).

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

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☐ Life sciences ☒ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

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Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	The present study is a quantitative cross-sectional study.
Research sample	A total of 86,772 UK adults (57% female; aged 62.4 ± 7.4 years) from the UK Biobank cohort who completed a 7-day accelerometry study. The UK Biobank is a non-representative sample.
Sampling strategy	Convenience sampling was used for both the accelerometry and light monitoring sub-study and the Mental Health Questionnaire (MHQ) sub-study. This sample is the largest to-date of objectively measured light, sleep, physical activity and mental health. Samples for analysis were determined by the maximum available n available for a given outcome and model covariates.
Data collection	Accelerometry and light monitoring sub-study: participants were invited via email to wear an accelerometer for seven days. Upon receiving the device, they were instructed to wear the device on their dominant wrist continuously and go about their normal activities. Mental Health Questionnaire (MHQ) sub-study: Participants were invited via email to complete the MHQ on the UK Biobank web-questionnaire online platform. Participants completed both the accelerometry and light monitoring sub-study and the MHQ sub-study under free-living conditions and without any experimenters present.
Timing	Accelerometry and light monitoring sub-study: 2013-2015; Mental Health Questionnaire (MHQ) sub-study: 2016-2017.
Data exclusions	Of the 103,720 participants with accelerometry and light data, 16,948 were excluded due to data file corruption, device non-wear and insufficient data for analysis.
Non-participation	Accelerometry and light study: 236,519 invited and 103,720 accepted. MHQ: 339,092 invited and 157,366 accepted.

Randomization	Participants were sorted into quartiles of day and night light exposure. Covaliates were age, sex, ethnicity, photoperiod, physical activity, sleep, cardiometabolic health and shift work.

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☒	☐ Dual use research of concern
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Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging

Clinical data

Policy information about clinical studies

All manuscripts should comply with the ICMJE guidelines for publication of clinical research and a completed CONSORT checklist must be included with all submissions.

Clinical trial registration	No clinical trial was performed.
Study protocol	The study protocol for obtaining the accelerometry, light and questionnaire data can be accessed in the open-source UK Biobank protocol documentation.
Data collection	Mental Health Questionnaire (MHQ; 2016) was completed via the UK Biobank study portal and accelerometry (2014) was completed under free-living conditions.
Outcomes	Psychiatric case/control outcomes and symptom severity scores were assessed via responses to Composite International Diagnostic Interview and DSM-IV items in the MHQ.