

# Social Rhythm Disruption is Associated with Greater Depressive Symptoms in People with Mood Disorders: Findings from a Multinational Online Survey During COVID-19

**La perturbation du rythme social est associée à des symptômes dépressifs marqués chez les personnes souffrant de troubles de l'humeur : résultats d'un sondage multinational en ligne durant la COVID-19**

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Piyumi Kahawage, BPPsych(Hons)<sup>1</sup> , Ben Bullock, PhD<sup>1</sup>,  
 Denny Meyer, DBL<sup>1</sup>, John Gottlieb, MD<sup>2</sup>, Marie Crowe, PhD<sup>3</sup>,  
 Holly A. Swartz, MD<sup>4</sup>, Lakshmi N. Yatham, MBBS, FRCPS<sup>5</sup>,  
 Maree Inder, PhD<sup>3</sup>, Richard J. Porter, PhD<sup>3</sup>, Andrew A. Nierenberg, MD<sup>6</sup>,  
 Ybe Meesters, PhD<sup>7</sup>, Marijke Gordijn, PhD<sup>8</sup>,  
 Bartholomeus C. M. Haarman, MD, PhD<sup>7</sup> , and Greg Murray, PhD<sup>1</sup> 

## Abstract

**Objectives:** Societal restrictions imposed to prevent transmission of COVID-19 may challenge circadian-driven lifestyle behaviours, particularly amongst those vulnerable to mood disorders. The overarching aim of the present study was to investigate the hypothesis that, in the routine-disrupted environment of the COVID-19, amongst a sample of people living with mood disorders, greater social rhythm disruption would be associated with more severe mood symptoms.

**Methods:** We conducted a two-wave, multinational survey of 997 participants ( $M_{Age} = 39.75 \pm 13.39$ , Female = 81.6%) who self-reported a mood disorder diagnosis (i.e., major depressive disorder or bipolar disorder). Respondents completed questionnaires assessing demographics, social rhythmicity (The Brief Social Rhythm Scale), depression symptoms (Patient Health Questionnaire-9), sleep quality and diurnal preference (The Sleep, Circadian Rhythms and Mood questionnaire) and stressful life events during the COVID-19 pandemic (The Social Readjustment Rating Scale).

<sup>1</sup> Centre for Mental Health, Swinburne University of Technology, Melbourne, Australia

<sup>2</sup> Department of Psychiatry and Behavioural Sciences, Northwestern University Feinberg School of Medicine, Chicago, IL, USA

<sup>3</sup> Department of Psychological Medicine, University of Otago, Christchurch, New Zealand

<sup>4</sup> Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

<sup>5</sup> Department of Psychiatry, University of British Columbia, Vancouver, Canada

<sup>6</sup> Dauten Family Center for Bipolar Treatment Innovation, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

<sup>7</sup> Department of Psychiatry Groningen, University of Groningen, University Medical Center Groningen, Groningen, the Netherlands

<sup>8</sup> Chrono@Work & Chronobiology Unit, Groningen Institute for Evolutionary Life Sciences, University of Groningen, Groningen, the Netherlands

## Corresponding Author:

Piyumi Kahawage, Department of Psychological Science, Centre for Mental Health, Hawthorn, Victoria 3122, Australia.  
 Email: [pkahawage@swin.edu.au](mailto:pkahawage@swin.edu.au)

**Results:** The majority of participants indicated COVID-19-related social disruption had affected the regularity of their daily routines to at least some extent ( $n=788$ , 79.1%). As hypothesised, lower social rhythmicity was associated with greater depressive symptoms when tested cross-sectionally (standardised  $\beta=-.25$ ,  $t=-7.94$ ,  $P=0.000$ ) and when tested using a 2-level hierarchical linear model across two time points ( $b=-0.14$ ,  $t=-3.46$ ,  $df=264$ ,  $P\leq 0.001$ ).

**Conclusions:** These results are consistent with the social zeitgeber hypothesis proposing that mood disorders are sensitive to life events that disrupt social rhythms.

## Abrégé

**Objectifs :** Les restrictions sociétales imposées pour prévenir la transmission de la COVID-19 peuvent perturber le style des comportements liés au rythme circadien, particulièrement chez ceux qui sont vulnérables aux troubles de l'humeur. Le but premier de la présente étude était d'investiguer l'hypothèse selon laquelle dans l'environnement de routine perturbée par la COVID-19, dans un échantillon de personnes vivant avec des troubles de l'humeur, une perturbation accrue du rythme social serait associée à des symptômes aggravés de l'humeur.

**Méthodes :** Nous avons mené un sondage multinational en deux cycles auprès de 997 participants ( $M_{age}=39.75 \pm 13.39$ , Femmes = 81.6 %) qui auto-déclaraient un diagnostic de trouble de l'humeur (c.-à-d. trouble dépressif majeur ou trouble bipolaire). Les répondants ont rempli des questionnaires évaluant les données démographiques, la rythmicité sociale (la brève échelle de rythme social), les symptômes de dépression (questionnaire-9 de la santé du patient), la qualité du sommeil et la préférence diurne (questionnaire Sommeil, rythmes circadiens et humeur) et les événements stressants de la vie durant la pandémie de la COVID-19 (l'échelle d'évaluation du réajustement social).

**Résultats :** En majorité, les participants ont indiqué que la perturbation sociale liée à la COVID-19 avait affecté la régularité de leurs habitudes quotidiennes du moins dans une certaine mesure ( $n=788$ , 79.1%). Comme nous l'avons postulé, la rythmicité sociale plus faible était associée à des symptômes dépressifs marqués quand elle était testée transversalement (normalisé  $\beta=-.25$ ,  $t=-7.94$ ,  $p=0.000$ ) et quand elle était testée à l'aide d'un modèle linéaire hiérarchique à deux niveaux et à 2 points dans le temps ( $b=-0.14$ ,  $t=-3.46$ ,  $df=264$ ,  $P\leq 0.001$ ).

**Conclusions :** Ces résultats concordent avec l'hypothèse du social zeitgeber qui propose que les troubles de l'humeur soient sensibles aux événements de la vie qui perturbent les rythmes sociaux.

## Keywords

COVID-19, depression, mood disorders, bipolar disorder, circadian rhythm, social rhythms

Disturbed circadian function has been hypothesised as a major contributor to the pathophysiology of mood disorders.<sup>1,2</sup> A wide range of human genetic, animal, physiological, and behavioural studies have reported circadian-related abnormalities including sleep/wake cycle disturbances, diurnal mood variations, and diminished amplitudes of body temperature, cortisol, thyroid-stimulating hormone, and melatonin rhythms in both depressive and bipolar spectrum disorders.<sup>3,4</sup> Social rhythm disruptions have also been implicated as determinants of symptoms in mood disorder populations, the focus of the present study. Social rhythms were markedly challenged for many individuals during the COVID-19 pandemic,<sup>5–7</sup> providing a novel context to explore the hypothesised link between disturbed social rhythms and mood.

## The Circadian Time-Keeping System

The human circadian system is a multi-level endogenous network of self-sustained oscillators (biological clocks), cycling for a period of approximately 24 h. The system is adapted to maintain the

optimal timing of a range of physiological, behavioural and cognitive processes such as sleep/wake, neurotransmitter, hormone secretion, alertness, and body temperature regulation.<sup>2</sup> The system's master oscillator is located in the suprachiasmatic nuclei (SCN) of the ventral anterior hypothalamus.<sup>8</sup> Critically for the present study, the circadian system is an open system: for these internal rhythms to synchronise appropriately to the external temporal environment, circadian clocks are entrained daily by environmental factors (zeitgebers).<sup>9</sup> In humans, light is the most powerful zeitgeber, however, 'social zeitgebers' such as exercise, mealtimes, social demands, and routine activities, also impact the circadian system.<sup>10</sup> A clinically important consequence of the system's open nature is that circadian function can be either disrupted or bolstered by the individual's behaviour in the environment.

## Social Rhythm Disruptions in Mood Disorders

The social zeitgeber hypothesis<sup>11</sup> posits that irregular social rhythms increase the likelihood of mood episodes in vulnerable individuals, with stressful life events (e.g., life transitions,

divorce, death of a loved one, loss of job) disrupting the timing at which key daily behaviours occur. It is proposed that the disruption of this zeitgeber input can lead to circadian dysregulation and consequently sleep/wake problems and relapse of mood episodes.<sup>10</sup> Indeed, this hypothesis has been influential in underpinning general recommendations to stabilise social rhythms in mood disorder treatment guidelines<sup>12</sup> as well as circadian-informed behavioural treatments such as Interpersonal and Social Rhythm Therapy (IPSRT).<sup>13–16</sup>

Although evidence for the social zeitgeber hypothesis is far from complete, a range of studies broadly supports the prediction that weakened social rhythmicity is a feature of people with mood disorders. As measured, for example, on the Social Rhythm Metric (SRM, a diary measure of the cross-day regularity of the timing of five key daily behaviours of getting out of bed, first social contact, commencing school/work, having dinner and going to bed<sup>17</sup>), social rhythms have been found to be less robust amongst individuals with unipolar depression,<sup>18</sup> on the bipolar spectrum<sup>19–21</sup> and those vulnerable to mood disorders.<sup>22–24</sup>

Reduced social rhythmicity has also been shown to predict the course of mood episodes. For example, Shen et al.<sup>22</sup> reported that lower SRM regularity scores at baseline predicted the onset of major depressive and hypomanic/manic episodes in 414 individuals diagnosed with either cyclothymia or bipolar II disorder after statistically controlling for baseline subsyndromal depressive, hypo/manic symptoms, and family history of bipolar disorder. Similarly, Sylvia et al.<sup>25</sup> found that individuals diagnosed with either bipolar II or cyclothymia experienced more social rhythm disrupting events in the 8 weeks before the onset of a depressive (but not hypomanic) episode than in a matched 8-week control period.

## The Present Study

Informed by circadian science, the social zeitgeber hypothesis makes testable predictions about the impact of social rhythms on mood, particularly amongst those vulnerable to mood disorders.<sup>26,27</sup> The current environment, in which public health measures have been implemented to curtail transmission of the COVID-19 virus, provides a unique opportunity to explore this prediction. Specifically, the social zeitgeber hypothesis would predict that social distancing, quarantine, and lockdown policies may challenge the regularity of daily social behaviours and sleep/wake schedules (social rhythms), weakening zeitgeber information to the circadian system in a large percentage of the population, generating mood problems proportionate to the individual's degree of social rhythm disruption. The overarching aim of the present study was to investigate the hypothesis that, in the routine-disrupted environment of the COVID-19 pandemic, amongst a sample of people living with mood disorders, greater social rhythm disruption would be associated with more severe mood symptoms.

## Methods

### Study Design

The Behaviour Emotion and Timing during COVID-19 [BEATCOVID] project is a population-based, two-wave, multinational survey delivered online via the Qualtrics survey platform. Data collection for both waves was open for 6 weeks (Wave 1: April–June 2020; Wave 2: August–September 2020). A design assumption was that COVID-related lockdowns would increase the risk of disrupted social rhythms in those with mood disorders. The pandemic, therefore, provided a novel context for testing the hypothesised relationship between social rhythmicity (predictor variable) and depressive symptoms (outcome variable) by looking at individual differences in both variables. The hypothesis was tested on the cross-sectional data of Wave 1, and prospectively in the two-wave data. Ethics approval for study procedures was provided by the Swinburne University Human Research Ethics Committee.

### Participants and Recruitment

Inclusion criteria for participation were: adults aged 18–65 years; self-reported diagnosis of either bipolar disorder or major depressive disorder (most circadian research has focussed on the former, but the hypothesis has also been applied to the latter<sup>13,28</sup>) and English proficiency sufficient to understand and respond to the survey (or Dutch proficiency for the subsample of participants recruited in the Netherlands). Study recruitment was via a digital advertisement containing a link to the survey. Participants were recruited via social media advertising (Facebook and Twitter), websites, news organisations, and mailing lists of relevant consumer, research, and community organisations in Australia, New Zealand, UK, USA, Canada, and the Netherlands. All participants received written information about the study and indicated consent before proceeding with the survey. Participation in Wave 1 was anonymous; however, participants were invited to provide an email address if they wished to be contacted about participating in Wave 2.

### Measures

A web-based survey was developed from commonly used validated questionnaires as well as individual items developed specifically for this study. Questions included a mix of multiple-choice, Likert scale, and free-text responses (the focus of a separate publication<sup>29</sup>).

**Demographics.** Participants provided information regarding their age, gender, country, state and city of residence, marital and employment status, and self-reported diagnosis of mood and other psychiatric disorders.

**Validated questionnaires.** *Mood Symptoms.* Although social rhythm disruption has been linked to both depressive and manic symptoms,<sup>12</sup> the former was chosen as the dependent variable here since depression is a relevant and prevalent outcome for both diagnoses of interest (bipolar and major depressive disorders). The severity and frequency of depressive symptoms were assessed by the Patient Health Questionnaire-9 (PHQ-9).<sup>30</sup> The nine items are scored on a 4-point Likert-type scale ranging from 0 (not at all) to 3 (nearly every day). The total score ranges from 0 to 27, with higher scores indicating greater severity of depressive symptoms. The PHQ-9 has sound psychometric properties, with good internal consistency (Cronbach's  $\alpha=.89$ ) and good test-retest reliability,  $r=.74$ .<sup>31</sup>

**Social Rhythmicity.** The regularity of daily activities was assessed using the 10-item Brief Social Rhythm Scale (BSRS).<sup>32</sup> The regularity of each activity is rated on a 6-point Likert-type scale ranging from 1 (very regular) to 6 (very irregular). Final item scores were reverse coded here for interpretability, with higher BSRS scores reflecting greater social rhythm regularity. The BSRS has been shown to have sound psychometric properties, including internal consistency ( $\alpha=.82$ ) and test-rest reliability ( $r=.70$ ).<sup>32</sup> The time frame of the scale was changed for the present project from the past 7–14 days to match the time frame of the PHQ-9.

**Sleep Quality, Mood, and Chronotype.** Sleep quality<sup>33</sup> and diurnal preference<sup>34</sup> are factors that are relevant to the proposed circadian aetiology of mood disorders and are therefore important potential covariates/confounds that need to be controlled in the present study analyses. The Sleep, Circadian Rhythms and Mood (SCRAM) questionnaire<sup>35</sup> contains three 5-item scales assessing each of these factors – sleep quality, circadian phase, and depressed mood. All items are measured on a 6-point Likert scale ranging from 1 (strongly disagree) to 6 (strongly agree). Each scale contains one reverse-scored item, and a scale score is the sum of ratings after reverse scoring. Higher scores indicate better sleep quality, morningness preference, and depressed mood, respectively. The SCRAM has been shown to have sound construct validity, adequate-good internal consistency (.81, .76, and .85, respectively), and strong test-retest reliability ( $r=.73$  to  $.83$ ).<sup>36</sup>

**Stressful events.** Factors other than social rhythms are also thought to impact mood symptoms, in particular, stressful events, and are also a potential confound that needs to be controlled for in the analyses. The Social Readjustment Rating Scale (SRRS)<sup>37</sup> is a checklist identifying 43 life events ranked in terms of proposed stressfulness. The total score is the sum of the pre-defined weights assigned for each event checked. Higher scores indicate greater chances of future health breakdowns due to stress. This inventory has shown adequate psychometric properties: Holmes and Rahe<sup>37</sup> found a positive correlation between life change scores and illness

scores (+.118), and Gerst et al.<sup>38</sup> found that rank-ordering remained consistent both for healthy adults ( $r=.96$  to  $.89$ ) and patients ( $r=.91$  to  $.70$ ). Participants in the present study were asked to consider stressful life events that they had experienced since the onset of COVID-19.

## Statistical Analyses

The study's single hypothesis was investigated firstly in the cross-sectional findings of the Wave 1 data alone, and secondly in within-subject analyses based on data from participants providing Wave 1 and Wave 2 data. Cross-sectional analyses involved a correlation analysis and a 3-stage hierarchical multiple regression for PHQ-9 scores, with individual differences in BSRS scores as the predictor of interest. Sensitivity analyses were conducted by controlling demographic variables of age, gender, country of residence, and diagnosis (bipolar disorder/depression) in Block 2 of the regression, and other clinical variables identified as bivariate correlates of PHQ-9 scores, which were subsequently controlled in Block 3. In the within-subject analyses using both waves of data, a 3-level hierarchical linear modelling (HLM<sup>39</sup>) was proposed to analyse a data structure where the IV (BSRS scores) and DV (PHQ-9 scores) (Level-1) were nested within participants, who differed in age, gender, and diagnosis (Level-2), who were nested within their country of residence (Level-3). The objective of this analysis was to study the relationship between the Level-1 variables – BSRS and PHQ-9, across time, while again controlling for the demographic variables, to provide additional confidence in the directionality of the findings.<sup>40</sup>

A Missing Values Analysis was conducted as part of the data preparation process to identify the rate and pattern of missing data. Both SPSS-26 and HLM-8 software packages were used for statistical analyses. Because of the large sample size,  $\alpha$  for significance was set at  $P<0.01$  (2-tailed).

## Results

### Descriptive Findings

A total of  $N=997$  individuals met eligibility criteria and consented to participate in the Wave 1 survey. A total of  $n=843$  (84.5%) responded to all items in the survey. The missing values analysis found that attrition increased across the survey. Accordingly, the pairwise deletion approach was used to handle missing data,<sup>41</sup> and the number of participants varied between  $n=863$  and  $n=963$  across analyses. Demographic and clinical characteristics of Wave 1 participants are presented in Table 1. Consistent with the design assumption, the majority of participants indicated that COVID-19-related social disruption had affected the regularity of their daily routines to at least some extent ( $n=788$ , 79.1%).

**Table I.** Demographic and Clinical Characteristics of Participants who Completed the First Wave of the BEATCOVID Survey.

	n	Proportion (%)	M(SD)
<b>Demographic Variables</b>			
Age	997		39.75 (13.39)
Gender			
Male	157	15.7	
Female	814	81.6	
Prefer not to say	9	0.9	
Other	17	1.7	
Country			
Australia	69	6.9	
New Zealand	521	52.3	
Canada	46	4.6	
USA	149	14.9	
England	40	4.0	
Scotland	61	6.1	
Netherlands	73	7.3	
Other	38	3.8	
Marital Status			
Single	402	40.3	
Married	305	30.6	
Divorced	68	6.8	
Separated	33	3.3	
De facto/common law/ domestic partner	177	17.8	
Widowed	12	1.2	
Current employment status			
Employed, full time	307	30.8	
Employed, part time	146	14.6	
Casual employment	46	4.6	
Not employed, looking for work	78	7.8	
Not employed, not looking for work	77	7.7	
Student, full time	116	11.6	
Student, part time	22	2.2	
Home duties	53	5.3	
Retired	36	3.6	
Disabled, not able to work	108	10.8	
Mood disorder diagnosis			
Bipolar Disorder	506	50.8	
Depressive Disorders	491	49.2	
Other mental health disorders			
Yes	552	55.4	
No	443	44.4	
Anxiety Disorder	414	41.5	
Substance Misuse Disorder	30	3.0	
Personality Disorder	79	7.9	
ADHD	84	8.4	
Psychotic Disorder	22	2.2	
PTSD	205	20.6	
Other	113	11.3	
Clinical Variables			
PHQ-9	963		12.80 (6.54)
Minimal Depression	117	12.1	
Mild Depression	196	20.4	
Moderate Depression	264	27.4	

(continued)

**Table I.** Continued.

	n	Proportion (%)	M(SD)
Moderately Severe Depression	207	21.5	
Severe Depression	179	18.6	
BSRS	863		34.17 (9.95)
SRRS	922		121.52 (87.39)
SCRAM Good sleep	871		14.80 (5.65)
SCRAM Morningness	871		16.70 (5.72)
SCRAM Depressed mood	871		17.68 (5.07)

Note. BSRS = Brief Social Rhythm Scale; PHQ-9 = Patient Health Questionnaire-9; SCRAM = The Sleep, Circadian Rhythms and Mood questionnaire; SRRS = The Social Readjustment Rating Scale.

### Hypothesis Testing

Bivariate analyses of the Wave 1 data demonstrated a negative relationship of moderate strength between the PHQ-9 and BSRS (i.e., lower social rhythmicity was associated with greater depressive symptoms;  $r = -.40$ ,  $P \leq 0.001$ ,  $n = 863$ ) with BSRS explaining 16% of the between-participant variation in PHQ-9. PHQ-9 scores were also associated with increased frequency of stressful events measured by the SRRS ( $r = .21$ ,  $P \leq 0.001$ ,  $n = 922$ ) and SCRAM-measured poorer sleep quality ( $r = -.49$ ,  $P \leq 0.001$ ,  $n = 871$ ), and decreased morningness chronotype ( $r = -.18$ ,  $P \leq 0.001$ ,  $n = 871$ ).

The regression assumptions of residual homoscedasticity and normality were supported. In addition, no outliers were detected using standardised residuals or Mahalanobis distances. As hypothesised, lower BSRS social rhythm scores were associated with higher levels of depression on the PHQ-9, when controlling for age, gender, country of residence, diagnosis, stressful life events, sleep quality, and morningness chronotype (standardized  $\beta = -.25$ ,  $t = -7.94$ ,  $P = 0.000$ ) (Table 2).

Two hundred and seventy participants consented to participate in Wave 2 and  $n = 265$  participants completed the survey ( $M_{Age} = 42.02 \pm 13.27$ , Female = 79.2%). T-tests and chi-square tests confirmed the two samples were similar in age, gender, diagnosis, and country of residence (Supplementary Table). HLM used an intention-to-treat analysis, which denoted that participants with missing data were included in the maximum likelihood estimation of parameters without the need for imputation of missing data. In the 3-level HLM model, the random intercept null model revealed that variation between countries had a non-significant relationship with depression ( $\chi^2 = 3.95$ ,  $df = 7$ ,  $P = 0.786$ ). Consequently, a 2-level HLM model was fitted instead, with the demographic variables of age, gender, and diagnosis at Level-2 of the model, and PHQ-9 (outcome variable) and BSRS (predictor variable) at Level-1. The intercept-only model revealed an ICC of .65. Thus, 65% of the variation in the PHQ-9 scores was attributed to between-participant variation. The relationship between BSRS and

**Table 2.** Summary of Hierarchical Regression Analysis Investigating Whether Greater Social Rhythm Disruptions are Associated with Greater Depressive Symptoms.

Variable	Model 1			Model 2			Model 3		
	B	SE	$\beta$	B	SE	$\beta$	B	SE	$\beta$
BSRS	-0.26	0.02	-0.40 <sup>b</sup>	-0.24	0.02	-0.37 <sup>b</sup>	-0.17	0.02	-0.25 <sup>b</sup>
Age				-0.08	0.02	-0.17 <sup>b</sup>	-0.07	0.02	-0.15 <sup>b</sup>
Gender									
Female vs Male				1.12	0.56	0.07	0.48	0.51	0.03
Prefer not to say vs Male				1.18	2.19	0.02	0.05	1.99	0.00
Other vs Male				1.64	1.63	0.03	0.86	1.48	0.02
Country									
NZ vs Australia				0.00	0.84	0.00	-0.67	0.76	-0.05
Canada vs Australia				0.10	1.23	0.00	-0.27	1.12	-0.01
USA vs Australia				-0.30	0.95	-0.02	-1.28	0.86	-0.07
England vs Australia				1.40	1.28	0.04	0.25	1.17	0.01
Scotland vs Australia				0.27	1.15	0.01	0.11	1.04	0.00
Netherlands vs Australia				0.55	1.11	0.02	0.88	1.01	0.04
Other countries vs Australia				0.03	1.30	0.00	-0.58	1.18	-0.02
Diagnosis									
Unipolar Depression vs Bipolar Disorder				-1.06	0.49	-0.08	-0.47	0.45	-0.04
SRRS							0.01	0.00	0.08 <sup>a</sup>
SCRAM Good sleep							-0.45	0.04	-0.39 <sup>b</sup>
SCRAM Morningness							0.02	0.04	0.02
R	0.40								0.59
R <sup>2</sup>	0.16								0.35
R <sup>2</sup> Change	0.16								0.14
F for change in R <sup>2</sup>	164.33 <sup>b</sup>								61.81 <sup>b</sup>

Note. <sup>a</sup> $p < .01$ ; <sup>b</sup> $p < .001$ ; BSRS = Brief Social Rhythm Scale; SCRAM = The Sleep, Circadian Rhythms and Mood questionnaire; SRRS = The Social Readjustment Rating Scale.

**Table 3.** Results of Hierarchical Regressions Examining the Temporal Relationship Between BSRS (Level-1 Predictor variable) and PHQ-9 (Level-1 Outcome variable) While Controlling for Demographic Variables.

Variable	B	SE	P
Gender	1.29	0.34	0.052
Age	-0.07	0.66	0.011
Diagnosis	-1.22	0.03	0.094
BSRS	-0.14	0.72	<0.001

Note. SE = standard error.

PHQ-9 across time remained negative and significant when controlling for the demographic variables of age, gender, and diagnosis ( $b = -0.14$ ,  $t = -3.46$ ,  $df = 264$ ,  $P \leq 0.001$ ) (Table 3). Interestingly, the variation in the slope between participants was not significant ( $\chi^2 = 246.90$ ,  $df = 236$ ,  $P = 0.30$ ) and only 4.9% of the within person PHQ-9 variation was explained by BSRS.

## Discussion

To our knowledge, this is the first study to explore associations between social rhythm disruptions and mood symptoms in a multinational sample of mood disorder participants during a

time of widespread lifestyle disturbance. Consistent with our hypothesis, we found that in a sample of individuals who are thought to be particularly vulnerable to rhythm disruptions, greater disturbances to social rhythms were associated with increased levels of depressive symptoms. This finding is congruent with the social zeitgeber hypothesis, highlighting the importance of loss of timed daily behaviours in the genesis of mood symptoms.

During the COVID-19 pandemic, various lockdown policies<sup>a,42</sup> and the resulting social changes may have decreased the regularity with which daily behaviours occurred. Indeed, participants endorsed that time cues such as sleeping, getting up, going to work, eating, exercising, and socialising were significantly altered due to the imposed restrictions; with the majority (79.1%) noting that this had an adverse effect on their social rhythmicity (see also qualitative findings in Kahawage et al.<sup>29</sup> that details participants' experiences of the social challenges associated with COVID-19 and challenges related to the disruption of rhythms). This finding is consistent with studies demonstrating that COVID-19 lockdown policies and the resulting social changes drastically impacted various lifestyle behaviours.<sup>7,43</sup> In the present study, this disruption may have weakened zeitgeber information to the circadian system, generating greater depressive symptoms proportionate to the individual's degree of social

rhythm disruption. Indeed, a strength of the current study was its design, where we were able to explore the nuances of the mechanistic underpinnings of the social zeitgeber theory by investigating individual differences in social rhythmicity (our predictor variable).

In Block 1 of the cross-sectional hierarchical multiple regression analysis, we found the expected negative relationship of moderate strength between BSRS and PHQ-9 scores. This relationship was found to be robust even after controlling for the demographic variables of age, gender, diagnosis, and country of residence in Block 2; and other ‘state-like’ clinical variables of diurnal preference, stressful life events, and sleep quality that are empirically thought to act as other sources of variance<sup>33,34</sup> in Block 3 of the model. These findings suggest that there is an independent relationship between an individual’s degree of social rhythm disruption and the severity of their mood disturbance.

A notable strength of our study was the investigation of the relationship between social rhythmicity and mood symptoms throughout a repeated assessment using HLM. A 3-level model was proposed *a priori*, with country of residence at Level 3. Here, the null model revealed that the variation between countries had a minimal effect on the outcome of interest (depressive symptoms). Findings from the 2-level HLM model revealed that when investigating change across 2 time points, the hypothesised relationship of interest persisted, even after controlling for demographic variables of age, gender, and diagnosis. However, it is worth noting that BSRS only accounted for 4.9% of the variance for within-participant PHQ-9 scores. Nevertheless, these results lend support for a temporal relationship between social rhythmicity and depressive symptoms. Taken together, our findings as a whole lend support for the social zeitgeber hypothesis and suggest that timed daily behaviours may be protective and an important aspect of mood stabilisation for those with mood disorders.

Our results broadly align with evidence that people with mood disorders are vulnerable to social rhythm disruption, with disruption impacting the course of the disorder.<sup>18,21,24</sup> Findings here also accord with general population investigations of the mental health and lifestyle impacts of COVID-19. For example, in a large ( $N=5854$ ) cross-sectional survey study of the Chinese general population, Yang et al.<sup>6</sup> measured social rhythmicity on a novel 17-item Chinese-language version of the BSRS, with the first nine items corresponding to the first nine items of the original 10-item BSRS used here. Consistent with the social zeitgeber hypothesis, Yang et al.<sup>6</sup> found the disruption of daily timing of sleep, getting up, and socialising were positively associated with self-reported symptoms of depression and anxiety (medium effect size, after controlling for demographic variables). Additional social rhythmicity items created by Yang et al.<sup>6</sup> for their study (regularity of eating, physical practice, and entertainment) did not show the expected relationship, highlighting the need for more rigorous attention to the measurement of social rhythmicity.

The present findings also suggest that the social rhythmicity of a significant proportion of the mood disorder population may be challenged by pandemic-related lifestyle disruption. Indeed, we have recently published a set of self-management strategies for increasing the regularity of timed daily behaviours that may be useful for those with mood disorders during periods of impaired lifestyle irregularity.<sup>44</sup> Several low-intensity, technology-aided, behavioural interventions based on the theoretical underpinnings of social rhythms may also be clinically useful at this time. These include *Mood Chart*,<sup>45</sup> *MoodRhythms*,<sup>46</sup> and more recently, *Rhythms And You (RAY)*,<sup>47</sup> which have all been piloted with promising results. Such interventions are cost-effective, feasible, and have not been associated with any serious adverse events.<sup>13,16</sup> The present findings suggest that further investigation of a readily disseminated intervention targeting social rhythmicity is an important research priority for potentially managing the impact of the pandemic, and future population-wide lifestyle disruptions.

Several limitations should be noted. First and foremost, the present findings cannot distinguish between cause and effect, and there is likely a bidirectional relationship between depressive symptoms and social rhythmicity.<sup>48</sup> Second, the inclusion criterion here of a mood disorder diagnosis was assessed only via self-report, hence, diagnoses must be considered provisional. Third, while the collection of data across two time points was a significant design strength, only a quarter of the Wave 1 sample provided data at Wave 2, potentially introducing a sampling bias (e.g., towards participants with particular challenges to the imposed restrictions/mental health). Similarly, the relatively small proportion of variance observed in the prospective analyses may be due to the relatively short time period between Wave 1 and Wave 2. Fourth, the generalisability of findings is limited by the availability sampling strategy, specifically, data collection via the internet, the disproportionate number of participants who were female and from New Zealand. Similarly, while the large sample size is a strength of the study, the relatively large proportion of participants with bipolar disorder (due to the researchers’ significant community networks in bipolar disorder) also challenges the generalisability of the present findings as applied to the larger population of all mood disorders. Lastly, as with all studies investigating associations between social rhythms and mood disturbances, social rhythmicity was measured using subjective self-report. To fully evaluate the merits of the social zeitgeber hypothesis, future research could usefully seek to replicate the present findings using more rigorous circadian assays such as dim light melatonin onset (DLMO), diary measures, or objective tools such as actigraphy.<sup>17,49,50</sup>

## Conclusion

Within its limitations, the present study concludes that for people with mood disorders, less regular social rhythms are

associated with more severe depressive symptoms under the lifestyle-disrupting sequelae of the COVID-19 pandemic. These results provide incremental support for the clinically influential, but still understudied social zeitgeber hypothesis which proposes that symptoms of mood disorders are sensitive to factors that disrupt social and biological rhythms. Future research is warranted into the development and refinement of interventions that target this vulnerability in mood disorder populations.

### Authors' Note

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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### ORCID iDs

Piyumi Kahawage  <https://orcid.org/0000-0001-8277-1754>  
 Bartholomeus C. M. Haarman  <https://orcid.org/0000-0002-9006-8863>  
 Greg Murray  <https://orcid.org/0000-0001-7208-5603>

### Note

- a. The extent of COVID-19-related restrictions varied by locality. Exploration of the relationship between stringency of lockdown (using the Oxford Stringency Index).<sup>42</sup> and social rhythmicity found no association [ $F(2, 860) = .99, P = 0.371$ ]. Further research

is required into the relationship between objectively measured public restrictions and subjectively experienced social rhythm disruption (the variable that was measured here), potentially mediated by objective/subjective changes in lifestyle at the individual level.

### Supplemental Material

Supplemental material for this article is available online.

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