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Social and circadian rhythm dysregulation and suicide: A systematic review and meta-analysis

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ABSTRACT

This systematic review of 52 studies provides a quantitative synthesis of the empirical literature on social and circadian rhythm correlates of suicidal thoughts and behaviors (STB). Small-to-medium pooled effect sizes were observed for associations between evening chronotype and STB and suicidal ideation (SI), although the pooled effect size diminished when accounting for publication bias. Three studies employed longitudinal designs and suggested eveningness was predictive of future STB, with a small-to-medium effect size. Social rhythm irregularity was also a significant correlate of STB with pooled effect sizes in the medium range. Overall circadian rhythm disruption was not associated with STB, although certain circadian rhythm metrics, including mean daytime activity, circadian rhythm sleep-wake disorder diagnosis, and actigraphy-assessed amplitude were associated with STB. Pooled effect sizes for these indices were in the medium to large range. There is a need for additional longitudinal research on actigraphy-based circadian parameters and objective markers of circadian phase (i.e., dim-light melatonin onset) to gain a clearer understanding of associations of endogenous circadian function and STB beyond that which can be captured via self-report.

1. Introduction

Suicide is a leading cause of death worldwide and, as such, a major public health issue. In the US alone, rates of suicide increased by 30% from 2000 to 2020, signifying a trend that warrants serious concern (Garnett et al., 2022). Indeed, after a 20-year increase, suicide rates declined in 2019 and 2020, but rose 4% in 2021 (Centers for Disease Control and Prevention, 2022). Further, over 12 million adults experience suicidal ideation annually, and over 20% of adolescents reported seriously considering suicide in the past year. In light of these alarming statistics, there is a need to better understand the etiology, phenomenology, and mechanisms of risk underlying suicidal thoughts and behaviors (STB) to ultimately reduce and prevent suicide. However, a 2017 meta-analysis of 365 studies found that across all risk factors examined in relation to STB, predictive validity only was slightly better than chance (Franklin et al., 2017). One limitation of traditional suicide prevention research is the reliance on at-risk individuals disclosing their thoughts, which they are often motivated to conceal (Blanchard and Farber, 2020). As such, there is increased interest in the identification and evaluation of potential risk factors whose measurement does not depend solely on the forthcomingness or insight of the at-risk individual.

Two distinct, yet related, potential risk candidates are sleep and circadian rhythm dysregulation. Circadian rhythms, relatively understudied in relation to STB, are biological processes that repeat roughly every 24-hours and help regulate a variety of mammalian functions, including the sleep-wake cycle (Ko and Takahashi, 2006; Reppert and Weaver, 2001). Regarded as an internal clock, circadian rhythms are governed by the suprachiasmatic nucleus (SCN), located in the hypothalamus, which serves as the endogenous pacemaker (Hastings et al., 2018). Circadian rhythms are entrained by external environmental cues (known as "zeitgebers," German for "time giver"), most notably light-dark cycles (Legates et al., 2014). In brief, the SCN receives photic input from the retina, and a multitude of afferent and efferent projections of the SCN synchronize rhythms in peripheral organs via an internal transcription-translation feedback loop. Several clock genes, such as Clock, Period (*Per1, Per2, Per3)*, and Bmal1 regulate the

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expression of proteins that synchronize physiological processes with the 24-hour cycle, thereby entraining an organism's innate sense of time with their external environment by modulating gastrointestinal processes (e.g., digestion and appetite), hormone secretion, body temperature, and rest-activity rhythms.

The circadian system also is entrained by nonphotic social rhythm cues. Social rhythmicity is the regularity of daily activities such as waketime, mealtime, exercise, and social activities (Monk et al., 1990, 1991). As such, social rhythm irregularity is characterized by a lack of consistency in daily routines and also can be operationalized as the frequency or quantity of events that acutely disrupt the timing of regular sleep-wake patterns (i.e., social rhythm disrupting events; Malkoff-Schwartz et al., 1998). Social rhythm regularity is hypothesized to help maintain the internal biological clock, and conversely, social rhythm irregularity and social rhythm disruption are thought to cause circadian instability and desynchrony (Ehlers et al., 1988; Grandin et al., 2006).

Once believed to be relevant primarily for regulating sleep-wake cycles, circadian dysfunction and dysregulation have been linked to multiple other negative physical and mental health outcomes (Abbott et al., 2020), including cardiovascular disease, diabetes, Parkinson's disease, affective disorders, and attention-deficit/hyperactivity disorder (Alloy et al., 2017; Baron and Reid, 2014; Crnko et al., 2019; Duncan et al., 2021; Leng et al., 2019; McMullan et al., 2013). Inasmuch as circadian science advances have found that nearly every organ contains biological clocks (Koronowski et al., 2019; Marcheva et al., 2010), it is unsurprising that circadian rhythms are associated with non-sleep related aspects of health and well-being. Indeed, the importance of social and circadian rhythms in affective disorders, particularly bipolar disorder, characterized by elevated rates of STB, is well documented. Evidence suggests that circadian disruption is associated with bipolar disorder and likely plays a causal role in its pathogenesis (Alloy et al., 2017; Murray and Harvey, 2010; Takaesu, 2018). The precise mechanistic underpinnings are unknown, but human genetic studies have found that among individuals with bipolar disorder, single nucleotide polymorphisms in the clock genes are associated with psychiatric outcomes, including a family history of suicide attempts (Pawlak et al., 2015). Further, when analyzed in concert, abnormalities in several of the main clock genes increased risk for bipolar disorder and major depression, providing additional evidence that molecular clock dysfunction may be involved in the development of mood disorders (McCarthy et al., 2012). Social rhythms also have been linked to mood disorder pathogenesis insofar as social rhythm irregularity has been shown to predict first onset of bipolar disorder among at-risk adolescents (Alloy et al., 2015). Taken together, it is plausible that social and circadian rhythm disturbance also may impact the onset and course of STB, a transdiagnostic phenomenon with increased prevalence among individuals with mood disorders.

However, for many years, the research on suicide and circadian rhythms focused on the epidemiologic distribution of suicides across a given time period, rather than considering the potential role of endogenous circadian dysfunction in the pathogenesis of STB. For example, there is ample literature showing that suicide follows seasonal and diurnal patterns in that suicide is more likely to occur during certain months and at certain times of the day (Christodoulou et al., 2012). Indeed, the seasonal effects in suicide have been highly replicated and population-based studies consistently have demonstrated that rates peak in the spring and decrease in the winter (Woo et al., 2012). Some hypothesize this pattern is driven by changes in meteorological factors, including sunlight exposure, which has direct relevance to the synchronization of circadian systems (Vyssoki et al., 2014).

From a research domain criteria (RDoC) perspective, difficulties with sleep, wakefulness, and circadian rhythms are classified as disruptions of the arousal and regulatory system, which aligns with the theoretical notion that suicide is more likely to occur during periods of heightened arousal (Glenn et al., 2017; Miller and Prinstein, 2019). These associations have been explored more thoroughly in the realm of sleep, with findings suggesting that sleep disruptions prospectively predict STB. There is a more limited, but growing body of literature examining associations between STB and social and circadian rhythm factors that occur at the individual level. Individual-level social and circadian factors are important to consider because, in contrast to circadian-relevant phenomena impacting entire populations (e.g., meteorological factors, geographic coordinates), they are most relevant for improving the identification and prediction of who is most at risk for STB. Core features of the circadian system (e.g., SCN activity) are difficult, if not impossible, to measure in living humans, which has led researchers to rely on the assessment of downstream constructs as indicators of circadian health (Murray et al., 2020).

Social and circadian factors that can be measured at the individual level include circadian rhythm variability, stability, circadian-rhythm-sleep wake disorders, chronotype, social jetlag, circadian amplitude, and social rhythm regularity. Chronotype is an individual's circadian preference, or their preferred timing of sleep and wake (Chauhan et al., 2023). Measures of chronotype tend to categorize people as evening types (e.g., "night owls"), morning types (e.g., "morning larks"), or intermediate types (neither). A negative relationship between self-reported eveningness and certain Cosinor parameters has been observed insofar as evening types tend to display lower amplitude in temperature rhythms and a lower rhythm-adjusted mean (mesor), providing evidence that eveningness is associated with less robust endogenous rhythmicity (Lipsanen et al., 2021; Martínez-Lozano et al., 2020). Actigraphy-based eveningness, as measured by sleep midpoint, also is inversely correlated with amplitude (Kuula et al., 2022). Social jetlag, a metric of misalignment, is the discrepancy between the innate biological clock and socially determined sleep timing, as measured by the difference in sleep duration on structured days (i.e., weekdays) and free days (i.e., weekend; Roenneberg, 2023). Circadian amplitude reflects the robustness of circadian rhythmicity and is quantified by the difference in the midpoint and high point of an oscillation.

An initial exploration of potential associations between individual-level circadian factors and STB took place in a 2018 systematic review of 13 empirical articles, which found greater eveningness and decreased rhythmicity were associated with STB (Rumble et al., 2018). It is interesting to note that this pattern of findings largely is parallel to those observed in bipolar disorder (enhanced eveningness and diminished rhythmicity), although the authors noted that none of the studies in their review of chronotype sampled individuals with bipolar disorder (Rumble et al., 2018). One limitation of this prior review is the absence of a formal quantitative assessment (i.e., meta-analysis), allowing for estimating the *strength* (i.e., effect sizes) of associations between circadian rhythms and STB, potentially due to the relatively low number of studies meeting inclusion criteria. The body of relevant literature has grown considerably over the past five years, offering a unique opportunity to provide a quantitative synthesis of the extant literature. Further, all but three studies in Rumble and colleagues' (2018) review relied entirely on self-report measures, which limit the study of circadian rhythms. As real-time monitoring tools such as actigraphy and other biomarkers of circadian rhythms become more commonly used in studies in this area, there are greater opportunities to move beyond subjective self-report measures of sleep behavior toward innovative objective indices that may provide unique insights.

The present study aimed to provide a systematic review and quantitative synthesis of associations between social and circadian rhythms and STB. Although there have been several recent meta-analytic reviews of aspects of sleep disturbance (e.g., insomnia, hypersomnia, nightmares), sleep characteristics (e.g., sleep onset latency, sleep efficiency) and STB (Harris et al., 2020; Liu et al., 2020), none have examined the circadian system, the primary focus of the current review. The goals of this study are as follows: i) to provide effect size estimates of the associations between indices of social and circadian rhythms, and where possible, specific forms of STB (e.g., suicidal ideation, suicide attempts),

ii) evaluate potential moderators of the strength of these associations, iii) integrate these findings with previous research and discuss implications, and iv) provide recommendations for future research.

2. Methods

Procedures for this meta-analysis were pre-registered on the International Prospective Register of Systematic Reviews (PROSPERO), registration number CRD42023400063.

2.1. Search strategy and eligibility criteria

A systematic search of the literature was conducted in MEDLINE and PsycINFO from inception to November 27, 2023 to identify studies relevant to the current review. The search terms for social and circadian rhythms used in this meta-analysis were based on those employed in a recent review of sleep and circadian rhythm disturbances in bipolar disorder (Scott et al., 2022), with the exception of search terms more germane to sleep disturbances and already covered in recent meta-analyses on sleep and STB (Harris et al., 2020; Liu et al., 2020). The following full text search string was applied: (“Circadian rhythm*” OR “circadian dysregulation” OR "social rhythm*" OR chronotype* OR morningness OR eveningness OR “circadian preference” OR “social jetlag” OR jetlag OR diurnal OR “biological rhythm” OR “biological clock” OR “rest-activity rhythm” OR "phase angle" OR “sleep wake disorder*” OR “sleep wake cycle*” OR “circadian rhythm sleep disorder*” OR “advanced sleep phase” OR “delayed sleep phase” OR “irregular sleep-wake” OR “non-24-hour sleep-wake” OR melatonin OR DLMO OR actigr* OR actom* OR “phase angle”) AND (suicid*). Search results were limited to: (i) English-language publications and (ii) peer-reviewed journals.

All unique reports were screened for inclusion by two independent coders as part of a two-step process. Agreement among independent coders was high ($\kappa = .85$). First, all unique reports were evaluated based on title and abstract. Second, the full text was reviewed in cases when eligibility could not be determined based on the title and abstract alone. Any discrepancies were discussed and resolved as a team. The study inclusion criteria were: (i) study assessed constructs specifically related to social or circadian rhythms (not sleep); (ii) study was conducted with human participants; (iii) suicidal thoughts and behaviors were analyzed distinctly from other constructs (e.g., excluded suicide risk scores and non-suicidal self-injury); (iv) circadian rhythms and STB were assessed systematically; (v) quantitative data were presented on the association between circadian rhythms and STB.

2.2. Data extraction

To conduct primary analyses, all data necessary for computing effect size were extracted. Whenever available, independent groups means and standard deviations were prioritized, but when these indices were unavailable, other statistics (e.g., correlation coefficients, unadjusted odds ratios or rates and group sample size) were used to compute effect sizes. When possible, the specific STB outcome (e.g., SI alone, SA alone) associated with the circadian correlate was extracted. In cases where the STB measure assessed both SI and SA together, the suicide correlate was coded as “STB.” If a study provided effects for specific types of SI (e.g., passive SI and active SI), both effect sizes were extracted and the mean was used to calculate pooled effect sizes. To conduct sub-analyses and to assess potential moderators in meta-analyses, data on 12 study characteristics were extracted. These included seven sample characteristics: (i) age group of the sample (youth [<18] or adult [≥ 18]); (ii) mean age of sample; (iii) sample type (community or clinical); (iv) percentage of female participants in the sample; (v) percentage of sample with depression; (vi) percentage of sample with bipolar spectrum disorders; and (vii) percentage of sample with a mood disorder. Data on five study design characteristics were extracted: (i) measure used to assess circadian rhythms; (ii) method used to assess circadian rhythms (self-report, actigraphy, biologic); (iii) method used to assess STB (self-report, interview); (iv) timeframe covered by STB measure (lifetime, current, etc.); and (v) whether the study analyses were cross-sectional or longitudinal. The full dataset used for analyses is available at https://osf.io/br5jz/.

2.3. Data Analysis

Comprehensive Meta-Analysis Version 3.3.070 (Biostat, 2014) was used for all analyses. Hedge’s g was used as the index of effect size for analyses examining associations between circadian factors and the STB outcome. Pooled effect sizes were calculated such that values more than 0 reflected positive associations between the circadian rhythm variable of interest and the STB outcome. Effect sizes of .10 are considered trivial, .20 small, .50 medium, and .80 large (Cohen, 1988; Kraemer et al., 2003).

A series of random-effects models were generated, as opposed to fixed-effects models, because random-effects models account for sampling and study-level error. Random-effects models were deemed more appropriate due to the anticipated heterogeneity across studies in terms of design, sampling methods, and assessment measures. The pooled effect size of random-effects models represents the weighted mean of a distribution of true effect sizes, whereas fixed-effects models rely on the assumption that one true effect size exists across all studies and all variance is due to sampling error.

The I^2 statistic was used to evaluate heterogeneity across the studies and corresponds to the percentage of variance in an effect estimate that is due to heterogeneity across studies rather than sampling error. Low, moderate, and substantial heterogeneity is indicated by I^2 values of 25%, 50%, and 75%, respectively (Higgins 2003). When significant heterogeneity was observed, moderator analyses were conducted to investigate potential sources of heterogeneity. Potential moderators were assessed individually such that effect sizes were estimated at each level of the moderator. When multiple moderators were significant, an unrestricted maximum likelihood multivariate meta-regression with a random-effects model was conducted simultaneously to evaluate the unique impact of all moderators found to be significant in univariate analyses on effect size estimates.

Publication bias was assessed only for the pooled effects for associations between chronotype and STB and chronotype and suicidal ideation (SI) due to the small number of unique effects available for other constructs. Duval and Tweedie’s trim-and-fill analysis (Duval and Tweedie, 2000) and Egger’s regression intercept (Egger et al., 1997) were used to assess the presence of publication bias. Publication bias is the tendency for studies with small or insignificant findings to often go unpublished, which may inflate the estimated overall effect size. Duval and Tweedie’s trim-and-fill provides an estimate of the number of potentially missing studies based on the degree of symmetry in the corresponding funnel plot as well as a bias-adjusted estimate of the overall effect size. Egger’s regression uses a linear regression approach to provide an estimate of publication bias by evaluating study effect sizes relative to their standard error.

3. Results

3.1. Included studies

A total of 2687 articles were identified using the above search parameters, of which 1797 were unique reports. A total of 1103 unique reports were excluded based on review of title and abstract alone. The remaining 694 underwent full-text review. If upon full-text review there was insufficient data for meta-analysis, study authors were contacted in order to obtain the relevant data. Additional data were obtained for three studies (Arrona-Palacios et al., 2021; Carbone and Casement, 2023; Tubbs et al., 2022). There were three instances of multiple studies

using overlapping samples. If the studies represented unique data (i.e., evaluated different aspects of circadian rhythms, or different STB outcomes), both studies were retained for inclusion in the relevant analyses. Otherwise, determination of which study to include was based on sample size, with larger sample sizes taking priority. If both studies had the same sample size, the study that assessed STB within a more recent timeframe was included. Three articles were excluded at this stage due to redundancy (Bradford et al., 2021; Palagini et al., 2019; Toh et al., 2023). A total of 642 studies were excluded during full-text review, leaving a final set of 52 articles included in the current review (see Fig. 1). Study characteristics for all eligible publications are presented in Table 1.

3.2. Chronotype

The most studied circadian feature was chronotype. As shown in Table 2, there was a total of 37 unique effects obtained for the association between eveningness and STB overall, and the pooled effect size was in the small-to-medium range (g=.31, 95% CI=.24–.38). Examining the association between eveningness and SI specifically, the pooled effect size was also small-to-medium (g=.28, 95% CI=.21–36). As for the relation between eveningness and suicide attempts (SA), a small effect size was obtained (g=.23, 95% CI=.06–.39).

Moderator analyses were conducted due to significant heterogeneity (STB: I^2=64.42%, $p < .001$; SI I^2 =59.60%, $p < .001$). In univariate moderator analyses, depicted in Table 3, sample type moderated the association between eveningness and STB. Specifically, there was a larger pooled effect for clinical samples (g=.48, 95% CI=.35–.61) than for community samples (g=.24, 95% CI=.18–.30). Chronotype measure also emerged as a moderator in analyses of STB. Pairwise comparisons revealed that the weighted effect size was significantly larger for studies using the Morningness Eveningness Questionnaire (MEQ; g=.63, 95% CI=.37–.90) compared to studies that used the Composite Score of Morningness (CSM) or the reduced Morningness Eveningness Questionnaire (rMEQ). None of the other potential moderators emerged as significant. In a meta-regression analysis accounting for 52% of the variance in the effect sizes, neither sample type nor chronotype measure remained a significant moderator of the strength of the association between eveningness and STB.

Moderation analyses for the association between eveningness and SI showed similar patterns. A larger pooled effect size was observed for clinical samples (g=.50, 95% CI=.32–.68) than community samples (g=.19, 95% CI=.14–.24) and studies that assessed chronotype using the MEQ also had larger effect sizes (g=.61, 95% CI=.34–.87) than the other measures (CSM and rMEQ). Additionally, age (as a continuous variable) moderated the association between eveningness and SI such that the strength of the association was stronger in older samples (B=.01, SE<.01, $p < .01$), although age as a categorical variable (youth versus adults) was not significant. Finally, diagnostic group also moderated the strength of the association between chronotype and SI; studies with a greater percentage of participants with a mood disorder had larger effect sizes (B=.004, SE<.01, $p < .01$). This finding appears to be driven by the percentage of sample with depression, as greater prevalence of depression (B=.004, SE<.01, $p = .02$), but not bipolar disorder (B=−.002, SE<.01, $p = .17$) was associated with larger pooled effect sizes. There were an insufficient number of studies relative to the number of

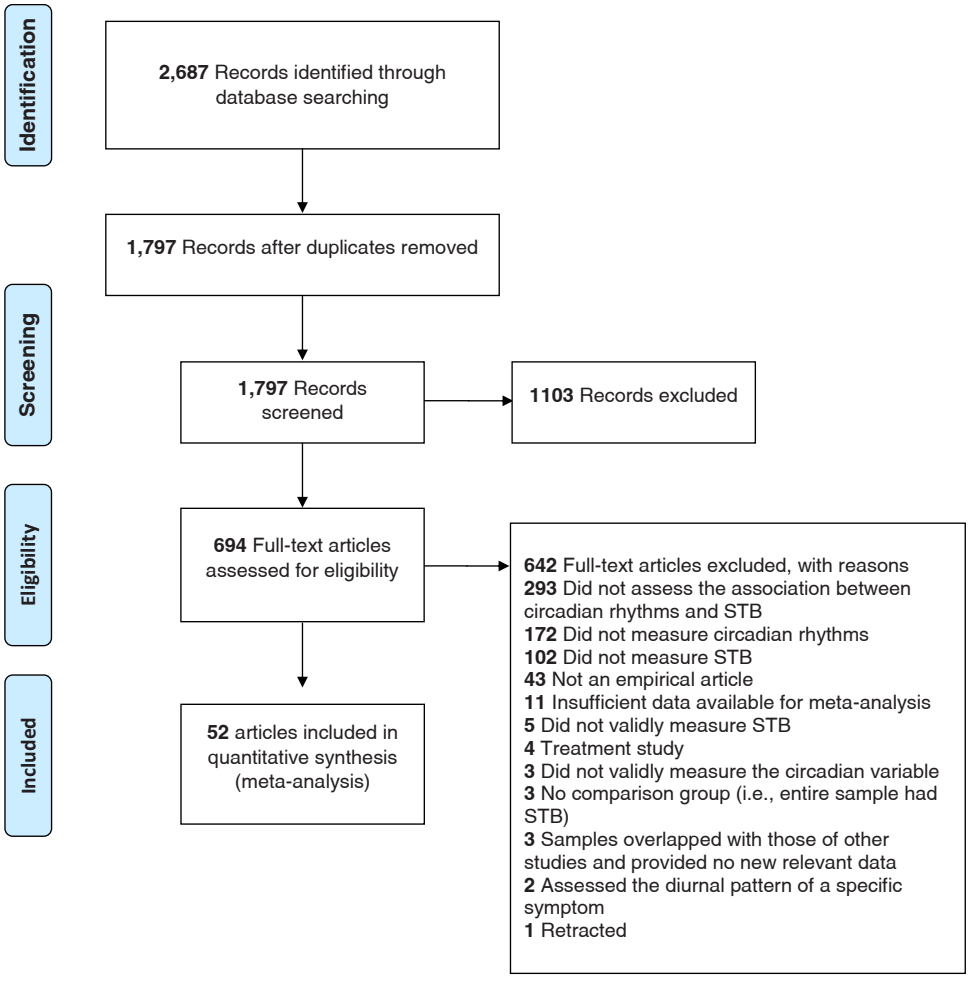

Fig. 1. PRISMA flow chart of literature search.

Table 1
Study characteristics.

Study Author (s) (year)	N[a]	% Female[a]	Mean Age[a]	Sample Type	Circadian Construct	Circadian Measure	Measure Type	Suicide Construct
Aronen et al. (2011)	44	36.36	10.70	Mixed	Rest-activity rhythms	Activity counts	Actigraphy	SI
Arrona-Palacio et al. (2021)	586	57.00	16.31	Community	Chronotype, Social jetlag	MESC, SSHS	Q	SI
Asarnow et al. (2020)	98	–	–	Clinical	Chronotype	CMEP	Q	SI
Bahk et al. (2014)	62	77.19	40.43	Clinical	Chronotype	MEQ	Q	SI
Beck Friis et al. (1985)	32	56.25	43.00	Clinical	Melatonin	Serum	Biological	SA
Benard et al. (2019)	236	60.17	43.44	Mixed	Amplitude, Chronotype, Inter-daily variability, Inter-daily instability, L5, M10, Rest-activity rhythms	Activity counts, CSM, CTI, L5, M10	Actigraphy, Q	SA
Benedetti et al. (2015)	87	65.52	46.16	Clinical	CLOCK gene	rs1801260 *C allele	Biologic	STB
Bertrand et al. (2020)	75	48.68	49.00	Clinical	Chronotype, Inter-daily variability	CenDI, SD of CenDI	Actigraphy	SI
Bradford et al. (2021)	259	65.64	21.15	Community	Chronotype	rMEQ	Q	STB
Carbone and Casement (2023)	65,230,478	56.59	–	Clinical	CRSD	ICD-10 code	Medical record	SI
Caruso et al. 2021[1]	162	59.30	46.90	Clinical	Social rhythm disruption	BRIAN	Q	SI
Chan et al. (2014)	253	82.61	50.80	Clinical	Chronotype	MEQ	Q	SI
Chan et al. (2020)	768	40.89	14.89	Community	Chronotype, Social jetlag	rMEQ, SSM	Q	SI
Chen et al. (2021)	414	55.8	13.8	Community	Chronotype	rMEQ	Q	SI
Chung et al. (2018)	301	77.41	49.65	Clinical	Chronotype	CSM	Q	SA
Fekih-Romdhane et al. (2019)	108	36.11	41.8	Clinical	Chronotype	rMEQ	Q	SA, STB
Gaspar-Barba et al. (2009)	100	79.00	33.90	Clinical	Chronotype	MEQ	Q	SI
Gau et al. 2007	1332	48.80	12.50	Community	Chronotype	CMES	Q	STB
Goldstein et al. (2008)	271	22.25	17.4	Mixed	DSWPD	K-SADS	I	Suicide Death
Indic et al. (2012)	36	75.00	46.10	Clinical	Amplitude	SSM	Actigraphy	SI
Karantonis et al. (2022)	67	55.28	24.40	Community, Clinical	Social rhythm disruption	SSM	Q	SI
Karaytug et al. (2022)	150	52.67	52.67	Clinical	Chronotype	MEQ	Q	SI
Khazaie et al. (2023)	2721	56.85	–	Community	DSWPD	ICSD	Q	SA
Kim et al. (2017)	2274	61.39	–	Community	Chronotype	CSM	Q	SA, SI
Kurtulus Dereeli et al. (2018)	42	26.19	32.55	Mixed	Melatonin	Serum, CSF	Biological	Suicide Death
Kuula et al. (2022)	307	40.06	17.40	Community	Chronotype	rMEQ	Q	STB
Lester (2015)	194	72.16	21.6	Community	Chronotype	MEQ	Q	SA, SI
Lyall et al. (2023)	19, 389	69.37	60.82	Community	Chronotype, L5, M10, Rest-activity rhythms	Activity counts, SSM	Actigraphy, Q	STB
Maruani 2023	244	39.75	52.51	Clinical	Chronotype	CSM	Q	SI, STB
McGlashan et al. (2018)	476	–	–	Clinical	Chronotype	rMEQ	Q	SI
Menculini et al. (2023)	178	53.37	44.76	Clinical	Chronotype	MEQ	Q	SA
Mokros et al. (2020)	289	74.05	21.28	Community	Chronotype	CSM	Q	STB
Nowakowska-Domagata et al. (2023)	600	68.17	21.94	Community	Amplitude, Chronotype	CQ-2	Q	SI
Nowakowska-Domagata et al. (2023b)	306	66.67	21.65	Community	Chronotype, Social jetlag	CSM, SSM	Q	STB
Palagini et al. (2022)[1]	197	43.00	46.40	Clinical	Chronotype, DSWPD, Social rhythm disruption	BRIAN	Q	SI, STB
Park et al. (2018)	5632	50.78	19.30	Community	Chronotype	CSM	Q	SI
Park (2020)	48	81.25	38.14	Clinical	Chronotype	CSM	Q	STB
Park et al. (2022)	79,009	43.02	40.15	Community	Shift work	–	–	SI
Rao et al. (1983)	12	50.00	–	Clinical	Melatonin	Urine	Biological	STB
Romo-Navo et al. (2020)	773	64.04	39.52	Clinical	Chronotype	SSM	Q	SA
Salvatore et al. (2023)	83	81.95	49.50	Clinical	Amplitude, Mesor, Rest-activity rhythms	Cosinor analysis	Actigraphy	SI
Sandyk & Awerbuch (1993)	28	85.71	40.20	Clinical	Melatonin	Blood	Biological	SA
Selvi et al. (2010)	80	55.00	30.60	Clinical, Community	Chronotype	MEQ	Q	SI
Silversten et al. (2010)	50,054	69.1	23.2	Community	Chronotype, DSWPD	ICSD	Q	SA, SI
Skene et al. (1990)	7	33.00	–	Clinical	Melatonin	Pineal gland	Biological	Suicide Death
Son & Lee (2021)	504	100	–	Community	Shift work	–	–	SI

(*continued on next page*)

Table 1 (*continued*)

Study Author (s) (year)	N[a]	% Female[a]	Mean Age[a]	Sample Type	Circadian Construct	Circadian Measure	Measure Type	Suicide Construct
Tubbs et al. (2021)	888	62.39	34.10	Community	Chronotype	SSM	Q	SI
Tubbs et al. (2023)	885	73.56	19.72	Community	Chronotype, Social jetlag	MCTQ	Q	SA, SI
Verkes et al. (1996)	59	64.41	37.50	Clinical	Amplitude, Rest activity rhythms	Activity counts, Period	Actigraphy	SI
Wong et al. (2022)	118	73.70	16.20	Clinical	Social rhythm disruption	DOTS-R	Q	SA
Xie et al. (2022)	7986	51.59	14.70	Community	Social rhythm disruption	SBRDA	Q	STB
Zhang et al. (2024)	39,731	13.49	45.62	Community	Social jetlag	SSM	Q	SI

Note. BRIAN = Biological Rhythms Interview of Assessment in Neuropsychiatry; CenDI = center of daily inactivity; CMEP = Children's Morningness-Eveningness Preferences Scale; CMES = Child Morningness/Eveningness Scale; CRSD = Circadian Rhythm Sleep Disorders; CSF = Cerebrospinal fluid; CSM = Composite Score of Morningness; CQ-2 = Chronotype Questionnaire; DOTS-R = Dimensions of Temperament Survey – Revised; DSWPD = Delayed Sleep-Wake Phase Disorder; ICD = International Classification of Diseases; ICSD = International Classification of Sleep Disorders; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; L5 = mean activity during an individual's least active 5 h; M10 = mean activity during an individual's most active 10 h; MEQ = Morningness Eveningness Questionnaire; MESC = Morningness-Eveningness Scale for Children; MCTQ = Munich Chronotype Questionnaire; rMEQ = reduced Morningness Eveningness Questionnaire; SD = standard deviation; SRBDA = Self-Rating Biological Rhythm Disorder; SSHS = School Sleep Habits Survey.
I = Interview; Q = Questionnaire; SSM = study-specific measure
SA = suicide attempt; SI = suicidal ideation; STB = suicidal thoughts and behaviors
[1] Studies with identical superscripts were drawn from same or overlapping samples but presented unique data included in this review.
[a]The sample size, mean age, and percentage female for participants included in relevant analyses, rather than of the entire study sample, are presented and were incorporated in moderator analyses whenever available. For ease of presentation, whenever the sample size, mean age, or percentage female varied across multiple relevant analyses within a study, data for the cumulative number of unique participants across these analyses are presented here, and the sample size used in each analysis was retained in the relevant meta-analysis for purposes of obtaining weighted effect sizes.

Table 2
Chronotype and Suicidal Thought and Behaviors.

	Effect Size Analyses				
	k	**N**	***g***	**95% CI**	***p***
Suicidal Thoughts and Behaviors					
Eveningness	37	88,768	.31	.24-.38	< .001
Eveningness (reference: Intermediate)	15	18,948	.32	.25-.38	< .001
Eveningness (reference: Morningness)	15	8702	.48	.30-.66	< .001
Morningness (reference: Intermediate)	14	20,910	-.09	-.24-.06	.24
Suicidal Ideation					
Eveningness	22	63,961	.28	.21-.36	< .001
Eveningness (reference: Intermediate)	9	4104	.33	.23-.44	< .001
Eveningness (reference: Morningness)	9	1483	.64	.33-.95	< .001
Morningness (reference: Intermediate)	8	3635	-.21	-.50-.08	.16
Suicide Attempts					
Eveningness	11	57,202	.23	.06-.39	< .01
Eveningness (reference: Intermediate)	3	2244	.18	-.09-.45	.18
Eveningness (reference: Morningness)	3	666	.36	-.02-.73	.06
Morningness (reference: Intermediate)	3	2240	-.18	-.51-.16	.30

Note: CI = confidence interval; k = number of unique effects; N = total number of participants included in pooled analyses.

significant moderators to conduct multivariate moderator analyses. Additionally, because there were relatively few unique effects ($k = 11$) for the association between eveningness and SA, moderation analyses were not conducted for this association.

To evaluate the specificity of chronotype – suicide association, additional analyses were conducted (see Table 2). Pooled effect sizes for associations between specific chronotypes were calculated (i.e., eveningness versus intermediate, eveningness versus morningness, and morningness versus intermediate). In analyses of STB, there were 15 unique effects examining eveningness relative to either intermediate types or morning types. A small-to-medium pooled effect was observed for eveningness relative to intermediate (g=.32, 95% CI=.25–.38), and a medium pooled effect size was obtained for eveningness relative to morningness (g=.48, 95% CI=.30–.66). The pooled effect size for the association between morningness (with intermediate as the reference group) and STB did not reach significance (g= −.09, 95% CI= −.24–.06).

A similar pattern emerged in analyses repeated with SI instead of STB as the outcome variable. The pooled effect for eveningness relative to intermediate was in the small-to-medium range (g=.33, 95% CI=.23–44), and in the medium range for eveningness relative to morningness (g=.64, 95% CI=.33–95). Based on 8 unique effects, morningness did not differ from intermediate in strength of association with SI (g= −.21, 95% CI= −.50–.08). Although the overall association between eveningness and SA was significant, none of the sub-analyses investigating specific chronotypes relative to one another and SA emerged as significant, although there were only 3 unique effects available for each of these sub-analyses.

Evidence of publication bias was found for both eveningness in association with STB and SI, respectively, based on Egger's regression test ($ps < .001$), Trim-and-fill analysis, and asymmetric funnel plots (see Figs. 2 and 3). Indeed, the adjusted effect sizes produced with the trim-and-fill method were smaller for both STB (adjusted g=.22, 95% CI=.15–.29) and SI (adjusted g=.19, 95% CI=.11–.27).

3.3. Other Social and Circadian Rhythm Indices

Table 4 presents analyses for all other social and circadian rhythm indices in relation to STB and SI; there was an insufficient number of studies evaluating other circadian factors and SA, so meta-analysis of SA was not conducted. Some of the largest pooled effect sizes were observed for circadian rhythm sleep-wake disorders (CRSWD) and STB (g=.63, 95% CI=.18–1.07) and SI (g=.63, 95% CI=.05–1.21). The majority of CRSWD studies assessed delayed sleep-wake phase disorder (DSWPD) specifically. A medium pooled effect size for DSWPD and STB was found (g=.47, 95% CI=.16–.78), although the pooled effect for DSWPD and SI did not reach significance (g=.42, 95% CI= −.01–84, p = .05). There were only two unique effect sizes for the association, however, suggesting the estimate is unstable. The associations between social rhythm disruption and STB (g=.52, 95% CI=.27–.78) and SI (g=.57, 95% CI=.33–.82) were also in the medium range. A medium pooled effect was observed for the association between mean daily activity (a facet of rest-activity rhythms), and STB (g=−.47, 95% CI=−.86 – −.08, p = .02), while associations with SI were in the large range (g= −.92,

Table 3
Moderator Analyses of the Associations Between Eveningness, Self-Injurious Thoughts and Behaviors, and Suicidal Ideation.

	Eveningness and Suicidal Thoughts and Behaviors										Eveningness and Suicidal Ideation					
	Univariate Moderator Analyses						Multivariate Meta-Regression Analysis				Univariate Moderator Analyses					
	k	***b***	**SE**	***g***	**95% CI**	***p***	***b***	**SE**	***p***	R^2	***k***	***b***	**SE**	***g***	**95% CI**	***p***
										.52						
Percentage Female	20	-.01	.01			.36					12	< .01	.01			.61
Age (Continuous)	18	< .01	< .01			.27					10	.01	< .01			< .01
Age (Categorical)	35					.67					21					.28
Youth	8			.29	.18-.39	< .001					5			.23	.10-.36	< .001
Adult	27			.32	.23-.40	< .001					16			.31	.22-.41	< .001
Sample Type	35					< .01					22					< .01
Clinical	16			.48	.35-.61	< .001	.18	.11	< .10		10			.50	.32-.68	< .001
Community	19			.24	.18-.30	< .001					12			.19	.14-.24	< .001
Percentage with Diagnosis																
% Mood Disorder	20	< .01	< .01			.24					12	< .01	< .01			< .01
% Depression	20	< .01	< .01			.24					12	< .01	< .01			.02
% Bipolar Spectrum Disorder	18	< .01	< .01			.60					9	< .01	< .01			.17
Chronotype Measure	23					.04					13					< .01
CSM[a]	9			.22	.04-.41	.02					3			.13	.06-.20	< .01
MEQ	8			.63	.37-.90	< .001	.20	.13	.13		7			.61	.34-.87	< .001
rMEQ	6			.30	.19-.42	< .001	-.01	.12	.93		3			.28	.14-.42	< .001
Suicide Measure Type	35					.47					22					.13
Questionnaire	22			.29	.22-.37	< .001					15			.25	.18-.32	< .001
Interview	13			.37	.18-.55	< .001					7			.46	.19-.74	< .01
Suicide Measure Timeframe	36					.39					22					.10
≤ 1 month	22			.34	.24-.44	< .001					17			.33	.22-.45	< .001
> 1 month	14			.28	.17-.38	< .001					5			.23	.17-.28	< .001

Note: CI = confidence interval; k = number of unique effects; N = total number of participants included in pooled analyses; CSM = Composite Score of Morningness; MEQ = Morningness Eveningness Questionnaire; rMEQ = reduced Morningness Eveningness Questionnaire.
[a]The category with the smallest effect size in univariate moderator analysis served as the reference group in the corresponding meta-regression analysis.

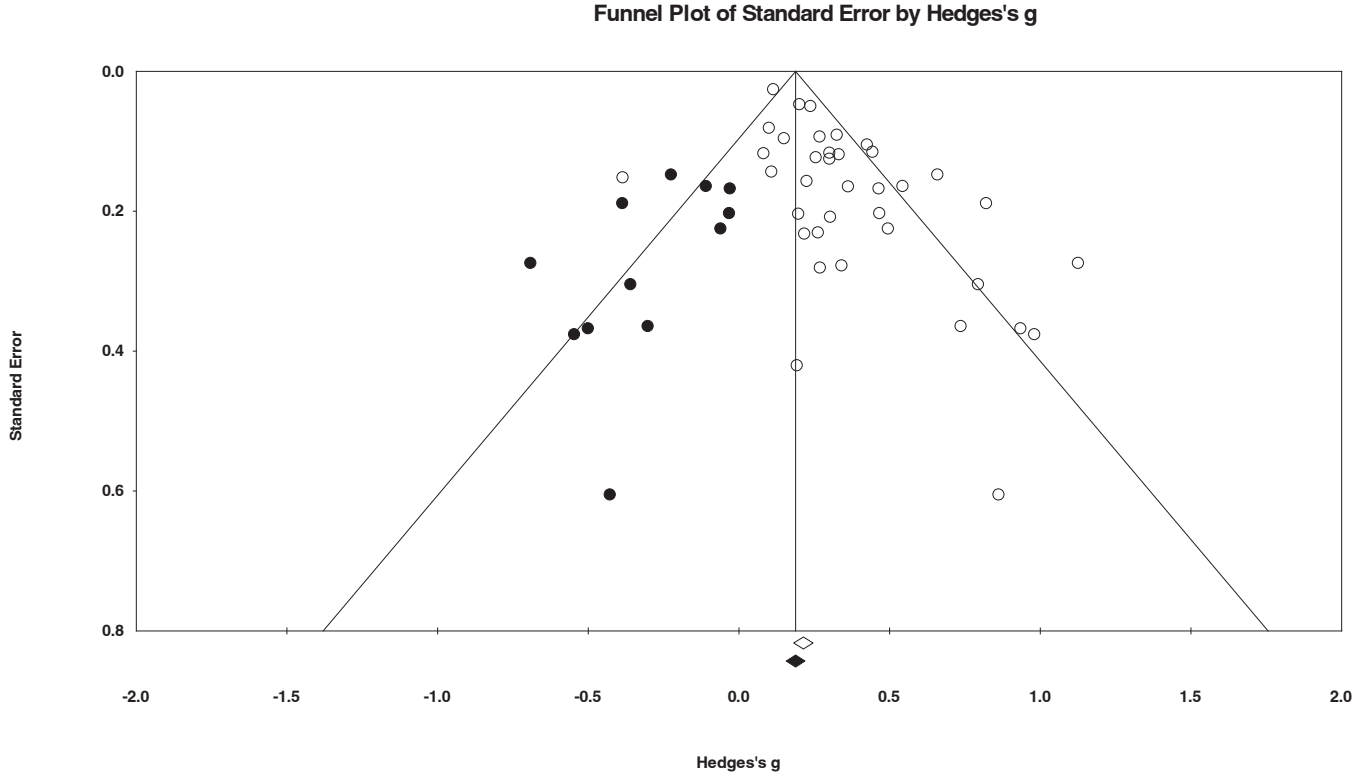

Fig. 2. Funnel plot for Eveningness and Suicidal Thoughts and Behaviors, The vertical line indicates the weighted mean effect. Open circles indicate observed effects for actual studies, and closed circles indicate imputed effects for studies believed to be missing due to publication bias. The clear diamond reflects the unadjusted weighted mean effect size, whereas the black diamond reflects the weighted mean effect size after adjusting for publication bias.

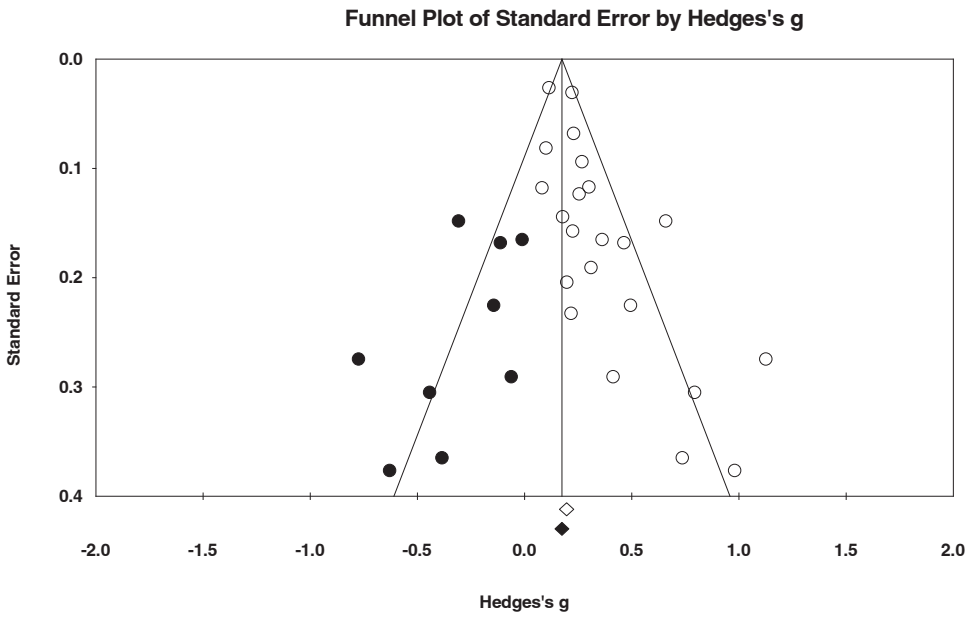

Fig. 3. Funnel Plot for Eveningness and Suicidal Ideation, The vertical line indicates the weighted mean effect. Open circles indicate observed effects for actual studies, and closed circles indicate imputed effects for studies believed to be missing due to publication bias. The clear diamond reflects the unadjusted weighted mean effect size, whereas the black diamond reflects the weighted mean effect size after adjusting for publication bias.

Table 4
Circadian Correlates of Suicidal Thoughts and Behaviors and Suicidal Ideation.

	Suicidal Thoughts and Behaviors					Suicidal Ideation				
Correlate	***k***	**N**	***g***	**95% CI**	***p***	***k***	**N**	***g***	**95% CI**	***p***
Social Jetlag	5	42,276	.14	-.07-.35	.19	4	41,970	.22	.05-.40	.01
Social Rhythm Disruption[1]	5	8368	.52	.27-.78	< .001	4	8215	.57	.33-.82	< .001
Shift Work[2]	-	–	–	–	–	2	79,513	.24	-.08-.56	.15
Circadian Rhythm Disruption	6	1126	.29	-.13-.72	.18	5	853	.41	-.16-.99	.16
Amplitude	5	1051	-.38	-.89-.13	.14	4	778	-.51	-1.27-.24	.18
Amplitude (actigraphy only)	3	215	-.77	-1.15–.38	< .001	3	178	-.80	-1.36- - .23	< .01
Circadian Instability/Variability	2	311	-.08	-.34-.18	.54					
L5	2	19,561	-.08	-.38-.21	.59					
M10	2	19,541	-.20	-.75-.34	.47					
Melatonin Concentration	5	121	-.40	-1.22-.43	.35					
Mean Daily Activity	5	19,811	-.47	-.86- - .08	.02	3	186	-.92	-1.70- -.14	.02
Circadian Rhythm Sleep-Wake Disorders	5	65,283,721	.63	.18-1.07	< .01	3	65,280,729	.63	.05-1.21	.03
Delayed Sleep-Wake Phase Disorder	4	53,243	.47	.16-.78	< .01	2	50,251	.42	-.01-.84	.05

Note: CI = confidence interval; *k* = number of unique effects; L5 = an individual's least active five hours; M10 = an individual's most active 10 h; N = total number of participants included in pooled analyses; effect sizes estimates where $k < 3$ are considered unstable and should be interpreted with caution; circadian rhythm disruption includes amplitude (reverse scored), mesor (reverse scored), and circadian instability/variability.
[1]Although shift work also could be classified as social rhythm disruption, the two studies on shift work and suicidal ideation were analyzed separately (i.e., were not included in the social rhythm disruption analyses) for greater granularity.
[2]The only two studies with data on shift work analyzed shift work in relation to suicidal ideation (not suicide attempts) and thus, the pooled effect size was not calculated for the association between shift work and suicidal thoughts and behaviors.

95% CI=−1.70- −.14). There were significant, yet small, associations between social jetlag and SI (g=.22, 95% CI=.05–.40), but not STB (g=.14, 95% CI= −.07–.35). None of the other social/circadian rhythm variables emerged as significant (overall circadian rhythm disruption, melatonin concentration, circadian amplitude, circadian instability/variability, L5, M10, or CLOCK gene variant). Sensitivity analyses for the associations between amplitude, STB, and SI were conducted using only studies where amplitude was assessed via actigraphy (i.e., excluding the two studies that measured amplitude via self-report). With regards to the association between actigraphy-derived amplitude and STB, a significant, large effect size was observed (g=−.77, 95% CI=−1.15 – −.38). This pattern of results also was seen regarding the association between actigraphy-derived amplitude and SI (g= −.80, 95% CI = −1.36– −.23).

3.4. Prospective Associations

Three prospective studies investigated whether chronotype was predictive of future SI. Two studies assessed SI at a two-week follow-up (Bertrand et al., 2020; Chen et al., 2021), and the third study had a one-year follow-up (Maruani et al., 2023). A small-to-medium pooled effect size (g=.35, 95% CI=.11–.59, p < .01) was observed.

4. Discussion

This systematic review provides the first quantitative synthesis of associations between circadian rhythms and STB. Findings generally suggest that evening chronotype is significantly, yet modestly, associated with STB, SI, and SA, although the strength of these associations is attenuated when accounting for publication bias. A medium effect size

was observed for the relation between social rhythm disruption and STB and SI, but not for overall circadian rhythm disruption and STB or SI. In analyses examining associations between specific indicators of circadian rhythmicity, overall circadian rhythm sleep-wake disorders, delayed sleep-wake phase disorder, mean daily activity, and actigraphy-assessed amplitude all emerged as significant, with effect sizes in the medium to large range. The other circadian rhythm indices either were not associated with STB or SI (e.g., melatonin concentration) or had too few unique effect sizes for meta-analysis (e.g., CLOCK gene variant, $k = 1$). These findings warrant elaboration.

4.1. Chronotype

The largest number of studies (k = 37) investigated associations between evening chronotype and suicide. Effect sizes for associations with STB and SI were in the small-to-medium range and in the small range for SA. However, both Egger's regression and trim-and-fill analysis found evidence of publication bias in analyses of STB and SI, indicating that the existing literature overestimates the strength of the associations, and the true effect size is likely in the small range. Suicide is a multifactorial phenomenon, more likely driven by multiple small risk factors than a handful of large risk factors, so despite the relatively small effect size, these findings should not be dismissed. To contextualize these findings, a more nuanced understanding of the chronotype construct is necessary.

Chronotype tends to be used synonymously with terms such as circadian preference or diurnal preference and traditionally is considered a proxy for circadian rhythms. Using this conceptualization, our findings suggest that individuals with disrupted circadian rhythms, as indicated by a delayed circadian phase, are more likely to experience STB. However, there is some debate as to whether chronotype reflects a psychological construct (leaning into the *preference* part of the definition), versus a biological construct (Roenneberg et al., 2019). Additionally, there is a question of whether chronotype reflects a stable trait-based feature versus a state-sensitive phenomenon (Roenneberg et al., 2019). A new multidimensional model of chronotype recently was proposed that places more emphasis on how social and environmental factors influence an individual's chronotype (Chauhan et al., 2023). This model grew from an observation that the current operationalization focuses primarily on the sleep dimension and thereby fails to adequately account for the effects of environmental or individual-level factors. Indeed, although chronotype tends to correlate with physiological circadian measures (e.g., dim light melatonin onset time), one limitation is that it is vulnerable to "masking," or the obscuring of the endogenous circadian rhythm by external factors, such as diet, movement, or temperature (Chauhan et al., 2023; Gall and Shuboni-Mulligan, 2022; Korczak et al., 2008). This malleability has generated debate as to what phenomenon chronotype actually captures (Chauhan et al., 2023).

Rather than trying to enhance the "purity" of the chronotype construct by reducing the influence of external factors, Chauhan and colleagues (2023) took a holistic approach and argued that we should not seek to disentangle social and environmental factors, but rather understand these elements as central features to the construct itself. In other words, chronotype cannot be studied in a vacuum, but needs to be considered comprehensively, taking into account the relative contributions of sleep timing and other lifestyle factors. Using this framework to interpret our findings, we found that a stronger evening preference (which is influenced in part by factors such as irregular eating habits, substance use, irregular lifestyle factors, and natural/artificial light exposure; Patterson et al., 2016; Teixeira et al., 2022; Vollmer et al., 2012) is associated with STB, particularly SI. Several of these factors also are associated with increased risk for STB, particularly substance use (Michael et al., 2020; Poorolajal et al., 2016). As such, it is possible that the association between evening chronotype and STB is not driven by endogenous circadian dysfunction, but rather is a consequence of STB's association with other risk factors that co-occur in individuals with evening preferences.

Lending weight to this possibility are the significant social rhythm disruption findings in the context of non-significant associations of STB with circadian rhythm disruption. Social rhythm disruption is correlated with circadian dysfunction and is hypothesized to play a causal role in circadian disruption (Ehlers et al., 1988; Grandin et al., 2006). Our findings regarding the SRD-STB relationship, but not circadian rhythm disruption-STB link, do not support the directionality of this theoretical pathway. Perhaps some of the non-sleep-related correlates of chronotype are being captured as social rhythm disruption. Social rhythm disruption often is operationalized as occurrences that alter patterns of behavior and cause irregularity in daily routines (Boland et al., 2012, 2016; Levenson et al., 2015), and likely can emerge from lifestyle factors in the absence of endogenous dysfunction. As such, eveningness, social rhythm disruption, and social rhythm irregularity may be standalone correlates of STB, or perhaps the directionality is reversed and social rhythm disruption functions as a mediator between endogenous circadian dysfunction and STB. Additional research is needed to directly test these possibilities.

4.2. Longitudinal Findings

The vast majority of studies that met inclusion criteria employed a cross-sectional study design, but three studies presented longitudinal data on prospective associations between chronotype and SI (Bertrand et al., 2020; Chen et al., 2021; Maruani et al., 2023). The pooled effect size for these studies was in the small-to-medium range (g=.35), similar to that of the unadjusted pooled effect size for all analyses of eveningness and SI. Two of the three longitudinal studies employed a two-week follow-up, while the third assessed STB at a one-year follow-up. It is well-established that the severity of SI fluctuates greatly within individuals even within a several-hour span (Kleiman et al., 2017), and as such, the dearth of studies with more temporally fine-grained evaluations of chronotype and SI limits the clinical utility of these findings, underscoring the importance of understanding the timescales with which these associations unfold. It seems unlikely that a rapid shift in chronotype is associated with STB at the daily level, but it is possible that evening types may be more vulnerable to the negative consequences of sleep disturbances. Additional prospective research, particularly using intensive longitudinal methods such as ambulatory assessment, is needed to further explore this possibility.

4.3. Delayed Sleep Wake Phase Disorder

Our findings also suggest that the eveningness – STB association may exhibit a dose-response relationship. There was a larger pooled effect size for the association between STB and delayed sleep-wake phase disorder, a disorder characterized by extreme eveningness, compared to the association between STB and eveningness. This may mean that a dimensional assessment of chronotype is more appropriate, and if chronotype is indeed a risk factor for future STB, there may be clinical utility in understanding the degree of eveningness to better characterize potential risk. Further, we conducted sub-analyses of evening, morning, and intermediate chronotypes in relation to one another whenever possible for a more fine-grained understanding. We consistently found stronger associations between eveningness and STB/SI when evening types were compared to morning types than when evening types were compared to intermediate types. A natural follow-up question is whether eveningness is driving the association with STB, or whether it merely reflects the absence of morningness (a potential protective factor). Prior work has demonstrated that adolescent morning types reported fewer sleep-related psychological problems than evening and intermediate types (Gelbmann et al., 2012). However, given that there were no significant differences in the pooled effect sizes for morning types compared to intermediate types, our findings are not consistent with the notion that morningness confers resilience. Taken together, our findings

suggest that eveningness is associated with STB, and the greater the evening preference, the stronger the association.

4.4. Potential Mechanisms

It is also possible that it is not eveningness itself driving associations with SI and other negative health outcomes, but rather the chronic misalignment evening types experience while living in a society designed for morning types. A standard US workday of 9 am – 5 pm and average school start time of 8:01 am (Paksarian et al., 2015) afford very little opportunity for evening types, who naturally go to sleep at 1 am or later, to get sufficient sleep (on average, adolescents need 8–10 h of sleep and adults need 7–9; American Academy of Sleep Medicine, 2023). The small yet significant pooled effect size for the association between SI and social jetlag, an indicator of misalignment, bolsters support for this mechanism. Further, executive functioning deficits (more common in evening types) have been linked to STB (Bredemeier and Miller, 2015). Indeed, in light of the chronic sleep deprivation experienced by evening types, it is unsurprising that morning types tend to have better academic performance, attention, and working memory (Facer-Childs et al., 2019; Heimola et al., 2021; Preckel et al., 2013). Eveningness may drive circadian misalignment, which leads to sleep deprivation, decreased cognitive control (Miranda et al., 2012), and increased impulsivity (Liu et al., 2017), which, in turn, contribute to an increase in STB. When considering this possibility, it is important to contextualize our findings alongside the previously observed associations between sleep disturbances and STB (Au and Reece, 2017; Liu et al., 2020).

Two recent meta-analyses found overall sleep disturbances and insomnia prospectively predict STB with small-to-medium effect sizes and identified sleep disturbance as a promising modifiable risk factor for reducing STB (Harris et al., 2020; Liu et al., 2020). Our results complement this work nicely and underscore the importance of identifying the mechanisms underlying sleep disturbances, a heterogeneous construct that could be due to a medical condition, psychological condition, environmental factor, or circadian dysregulation, in order to identify the appropriate treatment target (Thorpy, 2012). For example, Liu and colleagues (2020) found a small-to-medium (d=.45) pooled effect for insomnia predicting SI. However, it is unclear whether what is being captured as insomnia is primary insomnia (chronic physiological hyperarousal) or difficulty initiating or maintaining sleep because an individual is trying to sleep during times not aligned with their biological clock (Richardson et al., 2015; Roth, 2007). Prior work suggests that at least 10% of individuals diagnosed with chronic insomnia actually have delayed sleep-wake phase disorder (Magee et al., 2016). More longitudinal studies testing mechanistic pathways are needed for further elucidation.

One intriguing possibility lies in Tubbs and colleagues' Mind after Midnight hypothesis, which, in brief, purports that maladaptive behaviors including suicide, violence, and substance use, are more prevalent at night due to the downstream consequences of neurocognitive dysfunction that occurs during nocturnal wakefulness, when reason is "asleep" (Tubbs et al., 2022). We believe the Mind after Midnight hypothesis provides a helpful framework for future research. According to this theory, a multitude of factors (e.g., insomnia, life stress, limited social support, increased access to lethal means and substances) mediate the association between circadian disruption and risky behaviors, and the authors suggest interventions that help individuals obtain more overnight sleep may be helpful. Evening types, who are likely most at risk for the Mind after Midnight, may try to get more sleep by going to bed earlier, but find themselves unable to fall asleep due to their relatively delayed circadian rhythms. Insomnia is perpetuated by increased time awake in bed (Miller et al., 2014), and thus, an effort to get more overnight sleep actually may exacerbate sleep disturbances and the associated health risk behaviors. Considering our findings in the context of this theory, interventions that target circadian function, and not just sleep, may be important.

4.5. Moderator analyses and sub-analyses

Moderator analyses found that the associations between eveningness and STB/SI were significantly stronger in clinical samples relative to community samples. This was relatively unexpected because meta analyses tend to observe larger effect sizes in community, not clinical, samples (Liu et al., 2020; Nesi et al., 2021). Indeed, clinical samples tend to have greater comorbidity, and therefore, it would be expected that there would be higher rates of overall circadian rhythm disturbances in clinical samples (among individuals with and without STB), which would weaken the strength of the association between circadian disturbance and STB relative to that observed in community samples. However, in the multivariate moderator analysis, sample type was no longer significant, suggesting that sample type may not be a prominent clinical indicator of effect size.

Given that chronotype generally is thought to be more closely linked to affective disorders relative to other psychiatric conditions (Kivelä et al., 2018), we also examined whether associations were stronger in specific diagnostic groups. Diagnostic group did not moderate the chronotype-STB association, but the pooled effect size of the chronotype-SI association was incrementally larger in mood-disordered samples, specifically samples with a greater percentage of individuals with depression. This pattern of findings is potentially consistent with prior work showing that many correlates strongly associated with suicidal ideation (including depression in particular), do not differentiate ideators from attempters (May and Klonsky, 2016). In other words, the presence of depression does not confer additional risk for suicidal behaviors beyond its' association with SI (May and Klonsky, 2016), as observed in our findings. However, future studies of chronotype with suicidal behavior specifically are needed to more fully evaluate the possibility that chronotype is associated with SI more so than suicidal behavior.

The percentage of individuals with bipolar spectrum disorders was not a significant moderator. This is interesting given that rates of suicide are higher among individuals with bipolar disorder compared to other psychiatric illnesses, and individuals with bipolar spectrum disorders are more likely to be evening types than controls (Boudebesse et al., 2013; Gershon et al., 2018; Melo et al., 2017). Further, there is ample evidence supporting the role of circadian rhythm disruption in the pathogenesis of bipolar spectrum disorders. However, previous research on chronotype in bipolar spectrum disorders has found that eveningness is associated with depression, but not mania (Kivelä et al., 2018). One potential explanation for our findings is something akin to a ceiling effect in which circadian disruption is present to some degree among all individuals with bipolar spectrum disorders, and thus, eveningness is not an additive indicator of risk for SI.

4.6. Other Circadian Findings

There was a more limited number of studies with usable quantitative data evaluating other circadian factors in relation to STB. All findings are presented in Table 4, but please note pooled effect sizes with fewer than three unique effect sizes are considered unstable and should be interpreted with caution. We did not find support for associations between overall circadian rhythm disruption, melatonin concentration, or amplitude and STB. However, in sensitivity analyses restricted solely to actigraphy-assessed amplitude (i.e., excluding self-reported amplitude), a significant, large pooled effect was observed for both STB and SI such that low amplitude, indicating a less robust rhythm, was an indicator of risk for STB and SI. Below, we briefly review the findings of these studies and offer tentative explanations for this pattern of findings to stimulate future research in this area.

4.6.1. Amplitude

Five studies (three using actigraphy, two using self-report measures) examined constructs similar to amplitude, which is defined as the

distinctness, or differentiation, of rest-activity rhythms (Benard et al., 2019; Indic et al., 2012; Nowakowska-Domagała et al., 2023; Verkes et al., 1996). Four of the five studies (including all actigraphy studies) reported significant negative associations between amplitude and STB. Using actigraphy, Verkes and colleagues found that individuals with a history of recurrent suicide attempts were less likely to have distinct 24-hour activity peaks than healthy controls, and among suicide attempters, those lacking a distinct 24-hour peak had more suicidal ideation (Verkes et al., 1996). Another study of individuals with mood disorders used cosinor analysis and found a non-significant negative association between amplitude and passive SI (Salvatore et al., 2023). The authors of the other actigraphy study calculated a novel metric, the "vulnerability index," derived from multi-scale motility amplitude, and also found evidence that low amplitude is associated with greater suicidal ideation (Indic et al., 2012). Benard and colleagues employed the Circadian Type Inventory and demonstrated that adults with bipolar spectrum disorder and a history of SA have more languid (as opposed to vigorous) rhythms than adults without a history of SA and healthy controls (Benard et al., 2019). The only study to find a significant positive relationship assessed a slightly different construct, "subjective amplitude" which is multidimensional and conceptualized as the subjective reflection of the distinctness of diurnal rhythm fluctuations (Oginska et al., 2017). The developers of the Chronotype Questionnaire, which has a "distinctness" subscale used to assess subjective amplitude, state that this construct encapsulates feelings, behaviors, and preferences (Oginska et al., 2017). Given that this scale relies on subjective perceptions of behaviors, it may be inappropriate to consider subjective amplitude alongside circadian amplitude, which is derived solely from behaviors. This observation motivated the sensitivity analyses and the findings underscore the potential importance of using actigraphy, passive monitoring, and other objective measurement strategies to assess these circadian constructs.

If a less robust circadian amplitude is indeed associated with risk for STB, a natural follow-up question is whether amplitude is a reasonable target for intervention. This possibility has been explored in critical care research, wherein patients are confined to hospitals without social rhythm regularity or exposure to normal light/dark cues. This literature has found that circadian amplitude dampens with age and as organ function wanes (Schroeder and Colwell, 2013), but circadian amplitude enhancement is possible using strategies like timed exercise, timed fasting, light exposure, temperature control, melatonin administration, and cyclical feeding (Prin et al., 2023). It may be worthwhile to see whether circadian rhythm enhancement is effective as an adjunct treatment for individuals with psychiatric disorders and STB.

4.6.2. Daytime activity

Relatedly, there was a medium pooled effect for the association between lower daily activity and STB, and a large pooled effect for SI. It is interesting to note that this is the other circadian construct primarily measured via actigraphy, suggesting actigraphy may be important for detecting between-group differences in these circadian metrics. Diminished daytime physical activity has some conceptual similarities to dampened amplitude in that lower mean activity levels means there is less differentiation between day and night states. This may mean individuals with less daytime activity are less entrained to zeitgebers in their environment. These findings are consistent with previous research showing individuals with mood disorders are more sedentary than their same-aged peers (Vancampfort et al., 2016) and that low levels of physical activity are associated with mood symptoms (Walsh et al., 2023).

4.6.3. Melatonin

There were no studies examining associations between dim light melatonin onset (DLMO) time and STB. Inasmuch as DLMO is considered the gold standard for assessing circadian rhythms (Reid, 2019), this is a notable limitation of the current literature. The lack of DLMO studies also hindered examination of STB in relation to circadian misalignment, or the phase angle of entrainment (i.e., the interval between the endogenous circadian clock and the timing of an external cue), which can be calculated by computing the difference between DLMO time and bedtime onset (Emens et al., 2009). The paucity of studies on this topic represents a gap in the field, especially because it has been proposed that circadian misalignment is a component of most mood disorders (Lewy, 2009), and no studies have directly tested whether circadian alignment is associated with STB.

In the absence of DLMO studies, the pooled effect size from five studies that assessed mean melatonin concentrations among individuals with STB are reported in Table 4. There was no significant association, which is unsurprising because mean levels do not capture the cyclical nature of melatonin excretion and melatonin production can be impacted by posture, exercise, and diet, among other confounders (Kruk et al., 2021; Nathan et al., 1998).

4.7. Future directions

Our findings highlight several important areas for future research. First, there is a notable want of studies assessing circadian rhythms at the endogenous level. One study assessed a CLOCK gene polymorphism in relation to SI and SA (Benedetti et al., 2015), but there were no other genetic studies, nor were there any studies meeting inclusion criteria that measured HPA axis functioning, body temperature, or dim-light melatonin onset, the "gold standard" indicator of circadian phase. The assessment of core endogenous indicators of rhythms reduces the likelihood of "masking," or the obscuring of rhythms by environmental or behavioral factors and would provide greater insight into associations between the molecular clock and STB. Second future inquiry should utilize passive sensing techniques, such as actigraphy, to obtain more objective data about rhythmicity. Relatively few studies measured actigraphy-based rest-activity rhythm metrics (e.g., relative amplitude), and interestingly, a substantial portion of the circadian constructs that emerged as significant were based on primary studies that utilized actigraphy. The lack of ambulatory assessment and ecological momentary assessment studies also precludes a fine-grained investigation into the temporal associations, which would not only aid in identifying *who* is most at-risk for STB, but *when* they are at at-risk. Actigraphy also offers a reasonable intermediate between labor-intensive melatonin assays and self-report questionnaires, which are more vulnerable to confounding factors. Third, there is a need for more prospective studies to understand to what extent circadian factors have the potential to be modifiable risk factors. Nearly all studies meeting inclusion criteria reported cross-sectional findings, which offer important information about the strength of associations, but lack the temporal precedence that is a necessary condition for forming causal inference. The three longitudinal studies included in this meta-analysis provide preliminary evidence that eveningness may be a risk factor for subsequent STB, but more research is needed to fully explore this possibility. Fourth, given the majority of studies focused on chronotype, there is a need for enhanced specificity regarding the underlying construct. Moderator analyses revealed that effect sizes between chronotype and STB varied considerably depending on the assessment measure, which highlights the importance of heterogeneity in measurement in the field. A more consistent operationalization of chronotype would enhance the utility of relevant findings. Fifth, with the advent of nonparametric indices, future studies should incorporate variables such as intradaily variability, interdaily stability, L5, and M10. A small number of studies included in this review provided data about these constructs, but not enough to produce stable estimates.

More generally, future research is needed to improve conceptualization of circadian regulation. Specifically, studies that examine how activity/sleep rhythm parameters (e.g., amplitude, variability) relate to more direct assessments of clock function (e.g., core body temperature, dim light melatonin onset) in humans would advance understanding of circadian regulation. Likewise, studies that more directly test whether

social rhythm disruption or irregularity leads to circadian rhythm disruption or dysregulation also are needed.

4.8. Conclusions

Overall, this meta-analytic review of 52 studies found a small-to-medium pooled effect for the association between evening chronotype and STB/SI, although publication bias analyses suggest the true effect may be smaller than that observed. Effect sizes were largest in studies of clinical samples and in samples with higher percentages of mood disorders, specifically depression. There were a limited number of studies assessing endogenous or objective circadian constructs. Overall circadian rhythm disruption was not a significant correlate of STB, but two specific indices of circadian rhythmicity, actigraphy-based circadian amplitude and mean daytime activity, were both associated with STB and SI. Pooled effect sizes for these associations ranged from medium to large. Social rhythms also had significant associations with STB and SI with pooled effect sizes in the medium range. Future studies would benefit from utilizing actigraphy and other objective measures of circadian rhythmicity to evaluate the relative contribution of endogenous rhythm disruption and social or environmentally driven rhythm disruption.

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Data availability

The full dataset used for analyses is available at https://osf.io/br5jz/ .

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