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Review

Review

Chronotype, sleep timing, sleep regularity, and cancer risk: A systematic review

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Abstract

Sleep is a multidimensional modifiable lifestyle factor related to cancer risk. Prior research has primarily focused on sleep duration, despite the increasing importance of sleep timing and sleep regularity in the health research field. The objective of this systematic review was to synthesize the existing literature on the relationship of chronotype, sleep timing, and sleep regularity with cancer risk. We searched four databases (PubMed, CINAHL, PsychInfo, and Embase) in October 2024. The sleep exposures of interest included sleep timing, sleep regularity, sleep midpoint, social jetlag, chronotype, and weekend catch-up sleep, and the outcome of interest was cancer incidence (overall or site-specific). A total of 22 studies were included, of which 18 investigated chronotype, two investigated social jetlag, two investigated sleep midpoint, and one investigated weekend catch-up sleep as the sleep exposure. The majority of studies assessed sleep using self-reported questionnaires (95%) and investigated site-specific cancer incidence (91%). We found no consistent evidence linking late chronotype, later sleep midpoint, increased social jetlag, or weekend catch-up sleep to an elevated risk of cancer. This review highlights the heterogeneity in how sleep timing and sleep regularity are assessed. Future research should standardize measures on how to quantify sleep timing and sleep regularity and replication studies in diverse populations are needed. Current evidence linking sleep timing, sleep regularity, and chronotype with cancer risk remains inconclusive.

Introduction

Sleep is essential for favorable physical and mental health. Nonetheless, nearly one-third of US adults report insufficient sleep duration [1], trouble sleeping [2], and varied sleep timing throughout the week [2]. Insufficient sleep duration is associated with various adverse health outcomes including depression [3], adiposity [4], cardiovascular disease [5], and mortality [6]. Beyond sleep duration, the National Sleep Foundation (NSF) recently published a consensus statement on sleep timing and regularity stating that these sleep constructs are important for health and performance [7]. Findings from the NSF consensus statement reported that increased sleep timing variability and later sleep timing are associated with various adverse health outcomes (e.g., adiposity, hypertension, unfavorable markers of inflammation) [7]. These findings align with a 2020 systematic review of 41 articles that found later sleep timing and greater sleep variability were generally associated with 14 different health outcomes (e.g., mortality, cardiovascular disease, type II diabetes, adiposity) [8]. However, neither study investigated cancer incidence as an outcome of interest despite epidemiological and biological evidence linking sleep and cancer incidence.

Prior research on sleep and cancer risk has primarily focused on sleep duration [9, 10], despite sleep being multidimensional (e.g., duration, timing, regularity, quality) where behaviors intended to optimize a single domain may compromise others. Recently, there has been a greater focus on the relationship between sleep timing and regularity with cancer risk. Biological mechanisms linking sleep timing and sleep regularity with cancer risk include the many immune-, hormone-, and inflammatory-processes involved in circadian regulation [11]. Circadian rhythms dictate the timing of numerous physiologic processes and are critical to numerous aspects of health [11]. Variability in sleep timing and regularity introduces challenges to our circadian biology. Shift work is an example of severe disruption to circadian rhythms and has been deemed a Group 2A probable carcinogen to humans by the International Agency for Research on Cancer [12]. Irregular or sub-optimal sleep timing from sources other than shift work (i.e., social jetlag) presents a more subtle form of circadian

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misalignment. Numerous factors related to the misalignment or disruption of circadian rhythms may increase the risk of cancer independent of factors linking sleep duration and cancer risk; potential mechanisms may include exposure to artificial light at night (ALAN) [13], chronic suppression of the oncostatic hormone melatonin [14], abnormal cell proliferation [11], DNA damage [11], and other immune and endocrine alterations [11].

Prior reviews have summarized the evidence linking sleep duration [9, 10] and shift work [15] with cancer. To date, no study has gathered and reviewed the evidence on sleep timing and sleep regularity in relation to cancer risk. The objective of this systematic review was to synthesize the existing literature on the relationship between sleep timing and sleep regularity with cancer risk.

Methods

Study design

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines for this systematic review (Table S1). This systematic review of sleep timing and sleep regularity with cancer risk included prospective cohort, case–control, and nested case–control studies published through October 2024.

Definition of sleep parameters

Sleep regularity was captured through two measures: weekend catch-up sleep (WCS) and social jetlag. WCS is defined as the difference of weekend or non-workday sleep duration and weekday or workday sleep duration. Social jetlag quantifies the discrepancy between sleep midpoint on weekends or non-workdays versus on weekdays or workdays and represents the impact of the external environment on chronotype [16]. Sleep timing refers to the time of day that sleep occurs and can be measured using sleep midpoint, the median time between sleep onset and wake-up time, or chronotype. Related to sleep timing, chronotype is a biological construct describing the phase of entrainment as the alignment of the biological clock with the sun clock that is likely established by genetic, age, sex, and light-related exposures [17, 18]. In practice, chronotypes may range from early types (i.e., sleep bout from 08:00 pm to 04:00 am) to late types (i.e., sleep bout from 04:00 am to 12:00 pm), and chronotype may be influenced by a variety of social, behavioral, and environmental cues [18].

Inclusion/exclusion

Inclusion criteria were determined using the PECO (Population, Exposure, Comparison, Outcome) criteria. We included prospective cohort, case–control, and nested case–control studies with study populations of adults 18 years or older who were cancer-free at the time the sleep exposure was assessed or recounted. The sleep exposures of interest included sleep regularity, WCS, social jetlag, sleep timing, sleep midpoint, and chronotype. The outcome of interest was cancer incidence (overall or site-specific) presented as a hazard ratio, odds ratio, or risk ratio. Sleep parameters measured by accelerometry and self-reported questionnaire-based metrics were included. We excluded Mendelian randomization studies, randomized trials, review articles, and commentaries. We further excluded studies that reported sleep measures among cancer survivors, exclusively among individuals who engaged in shift work, and studies that assessed other sleep constructs as the exposure (e.g., shift work, sleep quality, sleep duration, insomnia symptoms, snoring).

Search strategy

We searched four electronic databases: PubMed, CINAHL, PsychInfo, and Embase. Peer-reviewed publications in the English language were included in the search and downloaded for review on October 16, 2024. The following search terms were used to combine (AND/OR) the essential phrase of "sleep and cancer risk": sleep midpoint, sleep timing, social jetlag, chronotype, weekend catch-up sleep, sleep regularity, cancer risk, cancer incidence. All sleep-related search terms were included in the search as keywords and/or Medical Subject Heading terms (tw, MeSH Terms, TX, MH). Cancer-related search terms were searched in the title and abstract (Title/Abstract, TI, AB) and as a proximity search within 2 words (~2, N2). The following search phrase was used for PubMed: ("sleep midpoint"[tw] OR "sleep timing"[tw] OR "social jetlag"[tw] OR "chronotype"[tw] OR "sleep regularity"[tw] OR "weekend catch-up sleep"[tw] OR "chronotype"[MeSH Terms]) AND ("cancer risk"[Title/Abstract:~2] OR "cancer incidence"[Title/Abstract:~2]). The following search phrase was used for CINAHL: (TX ("sleep midpoint" OR "sleep timing" OR "social jetlag" OR chronotype OR "sleep regularity" OR " weekend catch-up sleep") OR MH chronotype) AND (TI cancer N2 risk OR AB cancer N2 risk OR TI cancer N2 incidence OR AB cancer N2 incidence). The following search phrase was used for PsychInfo: (TX ("sleep midpoint" OR "sleep timing" OR "social jetlag" OR chronotype OR "sleep regularity" OR "weekend catch-up sleep") OR DE "chronotype") AND (TI cancer N2 risk OR AB cancer N2 risk OR TI cancer N2 incidence OR AB cancer N2 incidence). Lastly, the search phrase used for Embase is as follows: ("sleep midpoint" OR "sleep timing" OR "social jetlag" OR "social jetlag"/exp OR chronotype OR "chronotype"/exp OR "sleep regularity" OR "weekend catch-up sleep") AND ((cancer NEAR/2 risk) OR "cancer risk"/exp OR (cancer NEAR/2 incidence) OR 'cancer incidence'/exp). One author (SMD) searched the databases using the search terms above and downloaded search results. Two authors (SMD, BNB) independently completed title/abstract screening and conflicts were resolved by a third author (AIP). Eligible studies were included in full-text review and screened independently by two authors (SMD, BNB). Conflicts from the full-text screening were resolved by a third author (AIP). Eligible studies were assessed for quality by one author (SMD) and any queries were settled by another author (AIP). Studies were reviewed for quality with consideration of the guidelines outlined in the National Institutes of Health Study Quality Assessment Tool; however, no formal written or statistical assessment of study quality or risk of bias was conducted. Extraction of relevant information from the studies included in this review was completed by one author (SMD). One author (SMD) checked the references of all included studies for any additional relevant studies. The procedure of identification, screening, assessment of eligibility, and inclusion is shown in Figure 1.

Results

Figure 1 shows the results of the search and selection process. In total, 105 studies were eligible for title/abstract screening after removing duplicates. Seventy-seven studies were excluded through the title/abstract screening process and 28 studies were eligible for full-text review. Six studies were excluded during full-text screening [19–24], leaving 22 studies that met the requirements of the current systematic review.

Table 1 provides an overview of the studies included in this review. Of the 22 studies included, 18 investigated chronotype

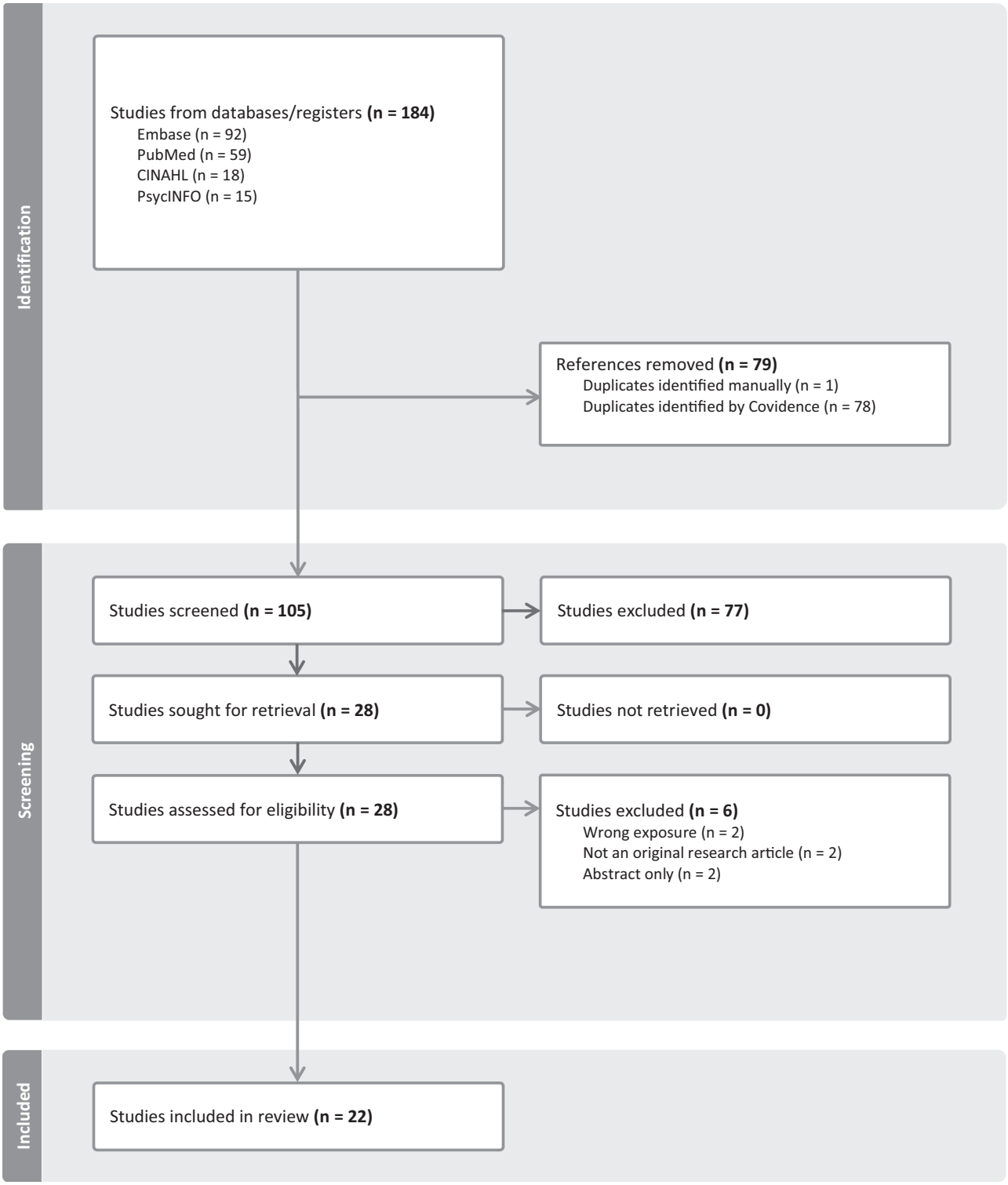

Figure 1. PRISMA flow chart of included studies.

[25–42], two investigated sleep midpoint [43, 44], two investigated social jetlag [44, 45], and one study investigated WCS [46] as the exposure of interest in a relationship with cancer incidence. Overall cancer incidence of any site was reported in three studies [25, 26, 43]. Site-specific cancers were more commonly reported and included breast [27–30, 43], colorectal [31, 32, 43], esophageal [35], endometrial [33, 34, 43], hematological [43], lung [36–38, 43], pancreatic [39], and prostate [29, 40–46], cancers. Approximately one-third of the studies utilized data collected in North America ($n = 8$) [25, 27, 28, 30, 33, 43, 45], and the publication year ranged from 2013 to 2024. Only one study utilized 7-day actigraphy to collect the sleep exposure of interest [44], while all other studies collected sleep constructs using questionnaires.

Table 1. Description of included studies

Study	Country of data collection	Sleep construct (reference group)	Data collection tool for sleep construct	Cancer type	Study design
Chronotype					
Kendzerska 2024 [25]	Canada	Chronotype (Intermediate type [categorical]; score of 63 [continuous])	MEQ	Overall	Retrospective cohort
Tian 2023 [26]	UK	Chronotype (definite morning)	UK Biobank questionnaire	Pancancer	Prospective cohort
Ramin 2013 [27]	US	Chronotype (definite morning type)	Nurses' Health Study II questionnaire	Breast	Nested case–control
Hurley 2019 [28]	US	Chronotype (definite morning)	MEQ	Breast (invasive only)	Nested case–control
Wu 2022 [29]	UK	Chronotype (extreme morning)	UK Biobank questionnaire	Breast	Prospective cohort
Von Behren 2024 [30]	US	Chronotype (morning type/ more morning than evening type)	MEQ	Postmenopausal breast (invasive only)	Prospective cohort
Chen 2022 [31]	UK	Chronotype (evening or more evening than morning)	UK Biobank questionnaire	Colorectal	Prospective cohort
Barber 2023 [32]	US	Chronotype (morning)	Black Women's Health Study questionnaire	Colorectal	Prospective cohort
Von Behren 2021 [33]	US	Chronotype (morning type)	MEQ	Endometrial (invasive only)	Nested case–control
Costas 2022 [34]	Spain	Chronotype (morning type [MCTQ], clearly morning [self-report])	MCTQ and single-item self-reported mid-sleep time question	Endometrial	Case–control
Wang 2023 [35]	UK	Chronotype (morning)	UK Biobank questionnaire	Esophageal adenocarcinoma (EAC) and esophageal squamous cell carcinoma (ESCC)	Prospective cohort
Xie 2021 [36]	UK	Chronotype (morning preference)	UK Biobank questionnaire	Lung	Prospective cohort
Peeri 2022 [37]	UK	Chronotype (definite morning)	UK Biobank questionnaire	Lung	Prospective cohort
Cordina-Duverger 2022 [38]	France	Chronotype (neutral type)	Single-item interview question	Lung	Case–control
Freeman 2024 [39]	UK	Chronotype (definitely morning)	UK Biobank questionnaire	Pancreatic	Prospective cohort
Dickerman 2016 [40]	Finland	Chronotype (definite morning type)	Older Finnish Twin Cohort questionnaire	Prostate	Prospective cohort
Lozano-Lorca 2020 [41]	Spain	Chronotype (morning type)	MCTQ	Prostate	Case–control
Lv 2022 [42]	UK	Chronotype (definitely morning)	UK Biobank questionnaire	Prostate	Prospective cohort
Wu 2022 [29]	UK	Chronotype (extreme morning)	UK Biobank questionnaire	Prostate	Prospective cohort
Sleep midpoint					
McNeil 2019 [43]	Canada	Sleep timing midpoint (intermediate sleep timing midpoint = 03:47–04:08 am)	Albert Tomorrow Project questionnaire	Breast, colon, prostate, lung, endometrial, non-Hodgkin lymphoma, hematological, and combined	Prospective cohort
Freeman 2024 [44]	UK	Sleep midpoint (intermediate sleep midpoint timing = 04:00–04:59 am)	7-day Actigraphy	Prostate	Prospective cohort
Social jetlag					
Hu 2020 [45]	Canada	Social jetlag (0 to <1 h)	Alberta Tomorrow Project questionnaire	Prostate	Prospective cohort

Table 1. Continued

Study	Country of data collection	Sleep construct (reference group)	Data collection tool for sleep construct	Cancer type	Study design
Freeman 2024 [44]	UK	Social jetlag (<1 h)	7-day Actigraphy	Prostate	Prospective cohort
Weekend catch-up sleep					
Corinda-Duverger 2022 [46]	France	WCS (0 h)	EPICAP face-to-face standardized computerized questionnaire	Prostate	Case–control

Abbreviations: US, United States; UK, United Kingdom; MEQ, Morningness-Eveningness Questionnaire; MCTQ, Munich Chronotype Questionnaire; WCS, weekend catch-up sleep.

Evidence synthesis of chronotype and cancer incidence

A total of 18 studies investigated the association of chronotype and cancer incidence [25–42]. Four studies [25, 28, 30, 33] collected chronotype using the Horne-Ostberg Morningness-Eveningness questionnaire (MEQ); one study [41] collected chronotype using the Munich Chronotype Questionnaire (MCTQ) [17]; 12 studies [26, 27, 29, 31, 32, 35–40, 42] collected chronotype using a self-reported single-item question; and one study [34] collected chronotype using the MCTQ and a self-reported single-item question. Three studies were case–control studies [34, 38, 41], three studies were nested case–control studies [27, 28, 33], one study was a retrospective cohort [25], and the remaining studies were prospective cohort studies [26, 29–32, 35–37, 39, 40, 42].

Six studies reported elevated site-specific cancer risk among individuals with an evening chronotype as compared to those with morning chronotypes; adjusted hazard ratios (HR) and odd ratios (OR) ranged from 1.13 to 1.30 and 1.24 to 1.44, respectively [28–30, 33, 36, 40] (Table 2). The associations with evening chronotype were found with respect to risk of breast (invasive [28], all cases [29], and postmenopausal invasive [30]), endometrial (invasive) [33], lung [36], and prostate [40] cancers.

The remaining studies of chronotype and cancer risk noted mostly null associations [26, 29, 31, 32, 35, 37–39, 41, 42]. Costas et al. measured chronotype using the MCTQ and a single-item self-reported question [34]. When chronotype was measured by the MCTQ, intermediate (i.e., "neither") chronotype showed an inverse association (adjusted OR = 0.41; 95% CI = 0.24, 0.68) with endometrial cancer compared to morning chronotype, while evening chronotype showed no association (adjusted OR = 1.00; 95% CI = 0.45 to 2.25) [34]. Yet, when utilizing a single-item question to measure chronotype, no association was found between chronotype and endometrial cancer risk [34]. Contrary to Costas et al., Ramin et al. reported an elevated risk of breast cancer among women with neither morning nor evening chronotype compared to definite morning chronotype (adjusted OR = 1.27; 95% CI = 1.04, 1.56) [27]. Kendzerska et al. measured chronotype using the MEQ, and when MEQ scores were categorized into chronotypes using predefined cut-points [25, 47], no association was found between chronotype and overall cancer incidence [25]. Kendzerska et al. also utilized MEQ score as a continuous measure ranging from 16 to 86 where lower scores represent evening-leaning chronotypes and higher scores represent morning-leaning chronotypes [47]. In models of continuous MEQ scores, Kendzerska et al. found higher MEQ scores (morning-leaning chronotypes) to be positively associated with overall cancer incidence (MEQ score 76–80 compared to MEQ score 63 adjusted HR range: 1.83–2.01) when compared to the median MEQ score ("neither" chronotype) [25]. However, 95% confidence intervals for the point estimates were relatively wide and MEQ scores are typically examined as a categorical measure rather than a continuous measure.

Evidence synthesis of sleep midpoint and cancer incidence

Two studies [43, 44] investigated the relationship between sleep midpoint and cancer incidence. McNeil et al. collected sleep midpoint by asking participants "On average, over the past 7 days, at what time did you normally go to sleep?" and "On average, over the past 7 days, at what time did you normally wake up?" and sleep timing midpoint was calculated as wake-time – ½ of total sleep duration [43]. Sleep midpoint was categorized as early sleep timing midpoint (<03:47 am), intermediate sleep timing midpoint (03:47–04:08 am), and late sleep timing midpoint (>04:08 am). Compared to the intermediate sleep timing midpoint, no association was found between early nor late sleep timing midpoint with colorectal, lung, prostate, or hematological cancer risk. Authors reported a positive association between late timing midpoint compared to intermediate timing midpoint and breast cancer risk (adjusted HR = 1.49; 95% CI = 1.09, 2.03) and overall cancer risk (adjusted HR = 1.20; 95% CI = 1.04, 1.37) [43]. Freeman et al. utilized 7-day actigraphy data to calculate the average sleep midpoint among participants of the UK Biobank and found no association with early (<04:00 am) or late (≥05:00 am) sleep midpoint compared to intermediate (04:00–04:59 am) sleep midpoint with prostate cancer risk (early midpoint adjusted HR = 1.00; 95% CI = 0.87, 1.16; late midpoint adjusted HR = 0.79; 95% CI = 0.57, 1.10) [44] (Table 2).

Evidence synthesis of social jetlag and cancer incidence

Two studies [44, 45] investigated the relationship between social jetlag and prostate cancer risk and the results varied. Hu et al. collected social jetlag by asking "On average, over the past 7 days, at what time did you normally go to sleep?" and "On average, over the past 7 days, at what time did you normally wake up?" [45]. This information was collected for weekdays and weekends; social jetlag was defined as the absolute difference in wake times between weekend days and weekdays. Authors reported a positive association between social jetlag and prostate cancer risk compared to < 1 h of social jetlag (1 to <2 h social jetlag adjusted HR = 1.45; 95% CI = 1.05, 2.01; ≥2 h social jetlag adjusted HR = 1.54, 95% CI = 1.04, 2.27) [45]. Conversely, Freeman et al. measured social

Table 2. Summary of results from included studies

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Chronotype							
Kendzerska 2024 [25]	1,781	Median 6.9 (IQR: 5.3–8.4)	Median 54.0 (IQR: 40.0–64.0)	53	Apnea-hypopnea index, mean oxygen saturation, total sleep time, sleep onset latency, age, sex, body mass index, alcohol use disorder, and Charlson comorbidity index	Morning HR = 1.36 (95% CI = 0.90, 2.06); Evening HR = 1.45 (95% CI = 0.56, 3.74); MEQ continuous total score of 76–80 vs. 63 HR range = 1.06–2.01	No association between categorical MEQ scores and cancer risk. Compared to the median MEQ score, higher MEQ scores were positively associated with overall cancer risk when MEQ was modeled continuously.
Tian 2023 [26]	326,417	Mean 11.28 years (SD: 2.64)	Mean 55.86 (SD: 8.12)	51	Age, sex, assessment center, top 10 genetic principal components, genotyping array, body mass index, employment status, Townsend deprivation index, smoking status, drinking status and mental health issues, vegetables and fruit intake, sedentary behavior, comorbidity, total physical activity, education, ethnicity, family history and other sleep traits	More morning HR = 0.99 (95% CI = 0.96, 1.01); More evening HR = 0.99 (95% CI = 0.96, 1.02); Definite evening HR = 1.03 (95% CI = 0.99, 1.07)	No association between chronotype and pancancer risk
Ramin 2013 [27]	72,517	N/A	Mean by chronotype categories: Definitely morning type = 53.2; More of a morning than an evening type = 52.8; More of an evening than a morning type = 52.7; Definitely evening type = 52.9; Neither morning nor evening type = 53.1	100	Age, family history of breast cancer, age at menarche, history of rotating night-shift work in years, smoking status, body mass index, alcohol intake in g/day, physical activity, history of benign breast disease, oral contraceptive use, menopausal status, age at menopause in years, parity and age at first birth, and postmenopausal hormone use	More of a morning than an evening type OR = 0.99 (95% CI = 0.87, 1.12); More of an evening than a morning type OR = 0.96 (95% CI = 0.84, 1.09); Definitely evening type OR = 1.15 (95% CI = 0.98, 1.34); Neither morning nor evening type OR = 1.27 (95% CI = 1.04, 1.56)	Neither morning nor evening types was associated with higher rates of breast cancer compared to definite morning types
Hurley 2019 [28]	39,686	N/A	N (%) 40–49 = 719 92), 50–59 = 6,706 (17), 60–69 = 16,531 (42), 70–79 = 10,568 (27), 80–89 = 5,162 (13)	100	Age, race, family history of breast cancer, age at menarche, smoking pack-years, body mass index, alcohol consumption, physical activity, age at first full-term pregnancy, breast feeding history, age at menopause, ever use of hormone therapy	More morning than evening OR = 1.09 (95% CI = 0.98, 1.22); Neither morning/evening OR = 1.11 (95% CI = 0.98, 1.23); More evening than morning OR = 1.11 (95% CI = 0.99, 1.26); Definite evening OR = 1.20 (95% CI = .06, 1.35)	Evening types was associated with higher rates of breast cancer compared to definite morning types

Table 2. Continued

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Wu 2022 [29]	110,070	Median 8.0	Mean 56.3 (SD: 8.0)	100	The first 10 columns of the genetic principal components of ancestry for genotype, the region of UK Biobank assessment center, Townsend deprivation index, age at recruitment, body mass index, smoking status, alcohol intake frequency, red meat intake, physical activity, type 2 diabetes, family history of breast cancer, menopausal status, usage of hormone replacement therapy, and usage of oral contraceptive pills	Breast moderate morning HR = 1.02 (95% CI = 0.92, 1.13); Breast moderate evening HR = 1.13 (95% CI = 1.01, 1.26); Breast extreme evening HR = 1.14 (95% CI = 0.97, 1.33)	Moderate evening chronotype was associated with a higher risk of breast cancer compared to extreme morning chronotype
Von Behren 2024 [30]	39,555	Mean 6.5	Mean 68	100	Age, race/ethnicity, body mass index, and family history of breast cancer	Neither morning/evening type HR = 0.94 (95% CI = 0.78, 1.14); Evening type HR = 1.19 (95% CI = 1.04, 1.36)	Higher postmenopausal breast cancer risk seen among evening chronotypes compared to morning chronotypes
Chen 2022 [31]	392,353	Median 8.51	% ≤ 55 = 43; % 55–65 = 39, % ≥ 65 = 18	55	Age, sex, education, household income, family history of colorectal cancer, and healthy lifestyle score	Early chronotype HR = 0.99 (95% CI = 0.95, 1.02)	No association between chronotype and colorectal cancer risk
Barber 2023 [32]	33,698	N/A	Mean 39.0 (SD: 10.8)	100	Age, time period, family history of colorectal cancer, body mass index, smoking status, alcohol consumption, vigorous physical activity, and processed meat consumption	Evening HR = 0.96 (95% CI = 0.73, 1.27); Neither morning nor evening HR = 0.77 (95% CI = 0.52, 1.14)	No association between chronotype and colorectal cancer risk
Von Behren 2021 [33]	27,190	N/A	N (%) < 40 = 3,361 (12), 40–49 = 11,138 (41), 50–59 = 8,476 (31), 60–69 = 3,717 (14), ≥ 70 = 498 (2)	100	Age at baseline, race/ethnicity, BMI at baseline, height, family history of endometrial cancer, family history of breast cancer, history of oral contraceptive use, history of live births combined with breast feeding, history of NSAID use (from questionnaire 5)	More morning than evening OR = 1.06 (95% CI = 0.81, 1.39); Neither OR = 1.00 (95% CI = 0.74, 1.37); More evening than morning OR = 1.14 (95% CI = 0.85, 1.52); Evening type OR = 1.44 (95% CI = 1.09, 1.91)	Women who were definite evening types had an elevated odds ratio of incident endometrial cancer compared to women who were morning types

Table 2. Continued

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Costas 2022 [34]	398	N/A	N (%) cancer cases < 60 = 51 (28.3), 60–69 = 60 (33.3), 70+ = 69 (38.3); noncancer controls < 60 = 69 (31.7), 60–69 = 68 (31.2), 70+ = 81 (37.2)	100	Age, education, body mass index, menstrual status, use of hormonal contraceptives, diabetes, hypertension, smoking status, and history of other cancers	Neither type OR = 0.41 (95% CI = 0.24, 0.68); Evening type OR = 1.00 (95% CI = 0.45, 2.25)	Neither type collected from the MCTQ was inversely associated with endometrial cancer compared to morning type. No association was found between self-reported chronotype and endometrial cancer
Wang 2023 [35]	393,114	N/A	Mean (SD) by composite sleep score: good 55.7 (8.1); intermediate 58.1 (7.9); poor 58.3 (7.9)	55	Age at baseline, sex, race, education, the Townsend Deprivation Index, history of GERD, body mass index, smoking status and intensity, alcohol consumption status and intensity (ESCC models only), physical activity, and shift work status	EAC More morning than evening HR = 0.99 (95% CI = 0.74, 1.33); EAC More evening than morning HR = 0.96 (95% CI = 0.70, 1.31); EAC Evening HR = 1.18 (95% CI = 0.78, 1.78); ESCC More morning than evening HR = 1.02 (95% CI = 0.59, 1.76); ESCC More evening than morning HR = 1.15 (95% CI = 0.70, 2.01); ESCC Evening HR = 1.86 (95% CI = 0.96, 3.60)	No association between chronotype and EAC or ESCC risk
Xie 2021 [36]	416,044	Median 7.13 (IQR: 6.43–7.74)	Mean (SD) cancer cases = 61.86 (5.78), noncancer controls = 56.29 (8.11)	54	Ethnic background, age, sex, smoking status, pack-years, alcohol intake, education, physical activity, body mass index, sleep medication, history of consulting for mental health, respiratory comorbidities, and sleep apnea	Intermediate preference HR = 1.03 (95% CI = 0.92, 1.14); Evening preference HR = 1.25 (95% CI = 1.07, 1.46)	Evening preference was associated with a higher risk of lung cancer compared to morning preference
Peeri 2022 [37]	382,966	Median 6.4 (IQR: 3.50–9.03)	Median 58 (IQR: 50–63)	53	Race, sex, body mass index, coffee and tea consumption, ambient nitrogen dioxide levels at baseline place of residence, pack-years smoked, smoking status, alcohol use, age, solid fuel cooking/heating, respiratory comorbidities, education, and genetic kinship	Slight morning HR = 1.01 (95% CI = 0.92, 1.10); Slight evening HR = 1.05 (95% CI = 0.96, 1.15); Definite evening HR = 1.10 (95% CI = 0.98, 1.24)	No association between chronotype and lung cancer risk

Table 2. Continued

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Cordina-Duverger 2022 [38]	1,474	N/A	N (%) by age category: < 50 = 181 (12); 50–59 = 371 (25); 60–69 = 662 (45); ≥ 70 = 240 (16)	100	Age, area of residence, marital status, socio-professional category, comprehensive smoking index, and body mass index 2 years before the interview	Morning OR = 1.38 (95% CI = 0.96, 1.99); Evening OR = 1.19 (95% CI = 0.82, 1.73)	No association between chronotype and lung cancer risk
Freeman 2024 [39]	420,302	Mean (SD) by sleep duration category: >7 h = 10.5 (2.2), 7 to <9 h = 10.5 (2.2), ≥9 h = 10.3 (2.5)	Mean (SD) by sleep duration category: >7 h = 56.7 (7.9), 7 to <9 h = 56.7 (8.2), ≥9 h = 58.8 (8.1)	60	Age, sex, body mass index, alcohol intake, smoking status, duration, and frequency, race, diabetes status, and employment/shift work	More morning than evening HR = 1.08 (95% CI = 0.92, 1.26); More evening than morning HR = 1.08 (95% CI = 0.91, 1.28); Definitely evening HR = 0.99 (95% CI = 0.77, 1.29)	No association between chronotype and pancreatic cancer risk
Dickerman 2016 [40]	11,370	Median 30	Mean 40.0 (SD: 12.1)	0	Age, education, body mass index, physical activity, social class, smoking status, alcohol use, snoring, zygosity, and shift work	Somewhat morning HR = 1.0 (95% CI = 0.8, 1.2); Somewhat evening HR = 1.3 (95% CI = 1.1, 1.6); Definite evening HR = 0.9 (95% CI = 0.6, 1.2)	Somewhat evening types had higher risk of prostate cancer compared to definite morning types
Lozano-Lorca 2020 [41]	875	N/A	Mean (SD) cancer cases = 67.7 (7.5), noncancer controls = 65.6 (7.9)	0	Age, education, first-degree family history of prostate cancer, physical activity, and smoking status	Neither OR = 0.94 (95% CI = 0.69, 1.28); Evening OR = 1.18 (95% CI = 0.72, 1.93)	No association between chronotype and prostate cancer risk
Lv 2022 [42]	213,999	Mean 10.45	Mean 56.42 (SD: 8.20)	0	Age, body mass index, ethnicity, qualification, smoking status, alcohol intake, coffee intake, vegetable intake, fruit intake, processed meat intake, red meat intake, PSA test, diabetes, and family history of prostate cancer	More morning HR = 0.99 (95% CI = 0.93, 1.05); More evening HR = 1.01 (95% CI = 0.95, 1.08); Definitely evening HR = 1.04 (95% CI = 0.94, 1.14)	No association between chronotype and prostate cancer risk
Wu 2022 [29]	106,632	Median 8.0	Mean 56.6 (SD: 8.1)	0	The first 10 columns of the genetic principal components of ancestry for genotype, the region of UK Biobank assessment center, Townsend deprivation index, age at recruitment, body mass index, smoking status, alcohol intake frequency, red meat intake, physical activity, type 2 diabetes, and family history of prostate cancer	Prostate moderate morning HR = 1.07 (95% CI = 0.98, 1.18); Prostate moderate evening HR = 1.01 (95% CI = 0.91, 1.11); Prostate extreme evening HR = 1.03 (95% CI = 0.89, 1.20)	No association between chronotype and prostate cancer risk

Table 2. Continued

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Sleep midpoint							
McNeil 2019 [43]	19,822	Mean (SD) cancer cases = 4.48 (2.46), noncancer controls = 8.70 (0.22)	Mean (SD) cancer cases = 60.7 (8.5); noncancer controls = 55.0 (9.0)	Cancer cases = 56, noncancer controls = 63	Age, sex (nonsex-specific cancers), total household income, employment status, marital status, education, ethnicity, smoking status, body mass index, presence of at least one medical conditions/comorbidity, presence of depression, family history of cancer, sleep duration, and gravidity (breast cancer only)	Combined site early sleep midpoint HR = 1.01 (95% CI = 0.88, 1.17); Combined site late sleep midpoint HR = 1.20 (95% CI = 1.04, 1.37); breast cancer early sleep midpoint HR = 0.99 (95% CI = 0.70, 1.40); breast cancer late sleep midpoint HR = 1.49 (95% CI = 1.09, 2.03)	Compared to intermediate sleep timing midpoint, late sleep timing midpoint was associated with an increased risk of combined cancer and breast cancer incidence
Freeman 2024 [44]	34,260	Mean 7.6	Mean (SD) by sleep duration category: ≤5 h = 64.0 (7.4), >5–6 h = 62.4 (8.0), >6–7 h = 62.2 (8.0), >7–8 h = 63.4 (7.7), >8 h = 64.4 (7.4)	0	Age, body mass index, overall health rating, smoking status, alcohol intake, education, income, moderate to vigorous physical activity, employment and shift work, race and ethnicity, Townsend Deprivation Index, history of prostate-specific antigen testing, family history of prostate cancer, diabetes status, coffee intake, and tea intake	<04:00 am HR = 1.00 (95% CI = 0.87, 1.16); ≥05:00 am HR = 0.79 (95% CI = 0.57, 1.10)	No association between sleep midpoint and prostate cancer risk
Social jetlag							
Hu 2020 [45]	7,455	Median 9.57	Mean (SD) by social jetlag category: 0–1 h = 58.77 (9.26), 1–2 h = 53.44 (8.14), ≥2 h = 51.86 (7.21)	0	Age, marital status, highest level of completed education, total household income, employment status, smoking status, frequency of alcohol consumption, recreational physical activity, total sitting time, pre-existence of medical conditions, chronotype, sleep duration, body mass index, daily caloric consumption, family history of cancer, and PSA screening	Social jetlag 1 to <2 h HR = 1.45 (95% CI = 1.05, 2.01); Social jetlag ≥ 2 h HR = 1.54 (95% CI = 1.04, 2.27)	Social jetlag longer than 1 h was associated with higher risks of prostate cancer compared to social jetlag less than 1 h

Table 2. Continued

Study	Total sample size	Follow-up years	Age (years) of included participants	% Female	Covariates included in adjusted models	Multivariable adjusted magnitude of associations	Result summary
Freeman 2024 [44]	25,826	Mean 7.6	Mean (SD) by sleep duration category: ≤5 h = 64.0 (7.4), >5–6 h = 62.4 (8.0), >6–7 h = 62.2 (8.0), >7–8 h = 63.4 (7.7), >8 h = 64.4 (7.4)	0	Age, body mass index, overall health rating, smoking status, alcohol intake, education, income, moderate to vigorous physical activity, employment and shift work, race and ethnicity, Townsend Deprivation Index, history of prostate-specific antigen testing, family history of prostate cancer, diabetes status, coffee intake, and tea intake	Social jetlag 1 to <2 h HR = 1.06 (95% CI = 0.89, 1.25); Social jetlag ≥ 2 h HR = 0.90 (95% CI = 0.65, 1.26); Per hour increase HR = 0.98 (95% CI = 0.89, 1.09)	No association between social jetlag and prostate cancer risk
Weekend catch-up sleep							
Corinda-Duverger 2022 [46]	1,698	N/A	N (%) by age categories: < 55 = 107 (6.3); 55–59 = 198 (11.7); 60–64 = 418 (24.6); 65–69 = 559 (32.9); ≥ 70 = 416 (24.5)	0	Age, family history of prostate cancer, ethnicity, body mass index, educational level, nightwork, and chronotype	WCS 1 to <2 h OR = 0.78 (95% CI = 0.57, 1.06); WCS ≥ 2 h OR = 1.35 (95% CI = 0.75, 2.45)	No association between WCS and prostate cancer risk

Abbreviations: IQR, inter-quartile range; SD, standard deviation; MET, metabolic equivalent; HR, hazard ratio; OR, odds ratio; CI, confidence interval; MEQ, Morningness-Eveningness Questionnaire; WCS, weekend catch-up sleep.

jetlag using data collected from 7-day actigraphy and found no association between social jetlag and prostate cancer risk (1 to <2 h social jetlag adjusted HR = 1.06; 95% CI = 0.89, 1.25; ≥2 h social jetlag adjusted HR = 0.90, 95% CI = 0.65, 1.26) [44] (Table 2).

Evidence synthesis of weekend catch-up sleep and cancer incidence

One study investigated the relationship between WCS and prostate cancer risk [46]. Corinda-Duverger et al. [46] retrospectively collected average sleep duration for weekdays and weekends separately during four different age ranges (i.e., before age 30 years, age 30–50 years, age 65–60 years, after age 60 years). WCS was calculated as the difference between weekend and weekday sleep duration for each age range. The four WCS measures were then averaged to create a single WCS measure across a lifespan. Authors describe WCS as "sleep deprivation," despite the derivation of "sleep deprivation" being identical to the definition of weekend catch-up sleep. WCS was categorized as no deprivation or <1 h (referent), 1–2 h, ≥2 h. No association between WCS and prostate cancer incidence was found (WCS 1 to <2 h adjusted OR = 0.78, 95% CI = 0.57, 1.06; WCS ≥ 2 h adjusted OR = 1.35, 95% CI = 0.75, 2.45) [46] (Table 2).

Discussion

This is the first systematic review of the associations of sleep timing and sleep regularity with cancer risk. We searched four databases and identified 22 studies that satisfied the inclusion criteria. Measures of sleep timing and sleep regularity included chronotype, sleep midpoint, social jetlag, and weekend catch-up sleep. The main findings of this systematic review suggest no consistent evidence linking late chronotype, later sleep midpoint, increased social jetlag, or WCS to elevated risk of cancer. However, the comparability between studies is limited due to the variety of measurement tools used to collect sleep parameters, inconsistent terminology, and heterogeneity of cancer site(s) evaluated.

Six of the 18 included studies investigating the relationship of chronotype and cancer risk reported higher cancer incidence among individuals who reported later chronotypes [28–30, 33, 36, 40], while ten studies reported no association between chronotype and cancer risk [26, 29, 31, 32, 35, 37–39, 41, 42]. Two studies [27, 34] reported contrasting results for "neither" chronotype in relation to cancer risk, and another study [25] reported morning chronotype to be associated with elevated cancer incidence. Discordance in results may be due to variations in how chronotype was assessed across studies. For example, the MEQ asks participants to report their preference in sleep behaviors while the MCTQ asks participants to report actual sleep behaviors and calculates chronotype using sleep midpoint on workdays and freedays [47, 48]. All of the included studies that captured chronotype from a single-item self-reported question [26, 27, 29, 31, 32, 35–40, 42] reported using a question similar to the last question of the MEQ which collects preferred sleep behaviors by asking participants to report if they consider themselves a morning or evening type. Given that preferred sleep timing and actual sleep timing often differ [18], it is likely that the MEQ and single-item questions versus the MCTQ are capturing different aspects of chronotype making it difficult to compare results across studies. It remains unclear on how best to measure self-reported chronotype to accurately capture circadian timing due to the dynamic nature

of entrainment of the biological clock from internal and external signals [18, 49]. Future studies should consider the addition of objective measures of circadian timing (e.g., urinary, salivary, or serum melatonin) to better capture the timing of the circadian phase in relation to sleep timing.

Chronotype was once thought of as a fixed trait but is now recognized as a state that is dependent on genetics, zeitgeber signals, and age [18]. The age-dependent nature of chronotype can be seen from inter-individual changes across the lifespan. Chronotype progressively delays throughout adolescence and then advances until the end of life [50, 51]. The interaction of age and natural light exposure may also contribute to the dynamic nature of chronotype [18]. Studies in the current review generally included middle- to older-aged adults. Yet, it is unknown when to measure chronotype in relation to cancer risk. Von Behren et al. assessed the relationship of chronotype during teens/college years, age 30–40 years, and postmenopause with postmenopausal breast cancer risk and found that the magnitude of association was similar when chronotype was assessed at 30–40 years of age and during postmenopause [33]. However, findings from Von Behren et al. [33] may have been impacted by recall and/or survivor bias due to chronotype being assessed after diagnosis of cancer. Five studies [25, 27, 28, 32, 33] assessed chronotype after the diagnosis of cancer and assumed that chronotype was relatively stable across adulthood. Repeat assessment of sleep patterns is necessary to further understand when sleep may be most impactful on subsequent cancer incidence and leveraging of consumer wearable devices may present the opportunity to more accurately assess sleep as a dynamic factor.

Generally, we found null findings for the association between sleep midpoint and cancer incidence for several cancer sites [43, 44]. McNeil et al. reported a positive association between late sleep midpoint and breast cancer and combined cancer risk [43]. Although, the association reported for combined cancer was likely driven by breast cancer due to breast cancer contributing to approximately 20% of all cancer cases in that study [43]. Sleep midpoint has been reported to be a valid proxy for chronotype [17]. Interestingly, the three studies that investigated the association of chronotype and breast cancer risk reported higher risk of breast cancer among women with late chronotypes [28–30]. However, two of the three studies used data from the California Teachers Study and participants of this cohort may have engaged in early work schedules due to early school start times regardless of their desired sleep schedule [28, 30]. Results highlight potential evidence of the relationship between late sleep timing and breast cancer risk, yet replication studies are needed to better understand how the relationship of sleep midpoint and breast cancer risk may differ from other site-specific cancers.

Social jetlag describes shifting the timing of sleep on free-days compared to days with work, school, or other obligations [16]. We identified two studies with contrasting findings investigating the relationship of social jetlag with prostate cancer risk. While Freeman et al. found no associations [39], Hu et al. reported social jetlag and prostate cancer risk to be positively associated [45]. Prior reports have shown social jetlag to be associated with metabolic changes, obesity, and inflammation [52], all of which are risk factors for cancer [53]. However, evidence of social jetlag and cancer risk remains inconclusive and more epidemiologic and mechanistic studies are needed to further investigate the potential relationship between social jetlag and overall and site-specific cancer risk.

Evidence on the mechanisms linking sleep with cancer is scarce. While the exact mechanisms linking sleep timing and sleep regularity with cancer remain unknown, several possible mechanisms involving the impact of circadian disruption have been explored [54]. Circadian disruption is the misalignment of biological and social clocks often due to sleep/wake and light/dark factors [55]. Social jetlag is a chronic, yet subtle, form of circadian misalignment [56], where social timing does not match biological time organization [16]. A pathway that has received significant attention is melatonin. Melatonin is a hormone essential to circadian rhythm and the duration of melatonin production defines the biological timing of night [57]. The presence of nightly melatonin has demonstrated anticancer effects by inhibiting cell proliferation, reducing oxidative stress, and promoting cellular differentiation and apoptosis [58–61]. For women, melatonin is a key suppressor of ovarian estrogen synthesis [62]. Studies have shown that high levels of endogenous estrogen can increase risk of breast cancer [63], suggesting the impact of altered melatonin production may be greater for breast cancer compared to other cancer sites. In this review, we identified four papers that found late chronotype [28–30] and later sleep midpoint [43] to be associated with higher risk of breast cancer incidence, which is more evidence than we found for any other cancer site. Future research should focus on the relationship of sleep patterns and breast cancer risk given that the current epidemiologic and mechanistic evidence appears stronger for breast cancer incidence than any other cancer site.

Natural melatonin onset and sleep midpoint are highly correlated [64]. However, it remains unclear if later sleep midpoint timing independently alters melatonin production and/or rhythm, especially if late sleep midpoint timing is in agreement with chronotype. Exposure to light during the biological night suppresses the release of melatonin and delays circadian phase [65]. Late sleep midpoint may alter melatonin production and/or rhythm leading to circadian disruption due to increased exposure to ALAN [66]. Research using rat models has demonstrated that exposure to ALAN suppresses melatonin and increases tumor growth rates [67, 68]. Epidemiologic studies of night-shift workers have shown that ALAN increases risk of cancer [15], and the International Agency for Research on Cancer Working Group deemed shift work as a probable carcinogen to humans due to "light during daily dark period" [12]. It is plausible that more ALAN exposure due to later sleep midpoint may increase cancer risk through melatonin suppression. Although the exact mechanisms of the relationship between sleep timing and sleep regularity with cancer remain elusive, mechanisms likely vary due to site-specific heterogeneity in cancer pathogenesis.

The field of sleep timing and sleep regularity with cancer risk is growing. There have been several systematic reviews and meta-analyses summarizing the evidence linking sleep duration [9, 10] and shift work [15] with cancer risk, which is why we decided a priori to focus on other, more under-studied aspects of sleep. Measures of sleep regularity beyond social jetlag and WCS are limited in the context of cancer. We found only one study that investigated WCS with cancer risk and only two studies that investigated social jetlag and cancer risk, highlighting a gap in existing research. Many legacy prospective cohorts only captured "usual" sleep patterns and do not collect day-level or workdays and non-workday sleep separately, limiting the ability to investigate daily and/or weekly changes in sleep patterns. Additionally, clear and consistent terminology is needed when describing sleep patterns for increased comparability. With the increasing use of

wearable devices in research, future studies should investigate day- and week-level sleep regularity as more granular sleep data become available.

Strengths of this review include the broad range of sleep measures and site-specific cancers included. This synthesis included information from 22 studies, incorporating different study designs, data collection tools, and data from over 600,000 individuals. There were several limitations to this review. Many studies utilized different measures of chronotype, sleep midpoint, and social jetlag making it difficult to aggregate the findings. Terminology of related sleep constructs varied, which may have limited our ability to accurately identify all relevant articles. Additionally, all but one study [33] measured sleep at one time point which may not reflect the chronic and dynamic nature of sleep patterns across the life course. The latency/induction period of sleep with cancer is unknown, further complicating when it is best to measure sleep to capture its potential relationship on cancer pathogenesis. Studies varied in confounders included in multivariable models, which may influence adjusted results between studies. Although some studies included occupational status in adjusted models, further research is needed to stratify by occupation type to understand how different types of occupation (e.g., outdoor laborers, office jobs, first responders, remote work) may impact the relationship between sleep timing and regularity with cancer risk. Although we excluded studies investigating only shift workers, heterogeneity within occupational status should be explored. Similarly, studies of more diverse populations are needed to increase generalizability. All studies included in this review had majority White participant populations and many studies utilized similar datasets (e.g., nine studies utilized data from the UK Biobank [26, 29, 31, 35–37, 39, 42, 44], three studies utilized data from the California Teachers Study [28, 30, 33], and two studies utilized data from the Alberta's Tomorrow Project [43, 45]). Results highlight the need for robust sleep measures to be incorporated in diverse study cohorts to further disentangle the relationship between sleep timing and sleep regularity among non-White participants of different geographical regions. Lastly, the relatively small sample size of studies investigating sleep midpoint, social jetlag, and WCS with cancer risk limited the generalizability of these results.

Sleep timing and sleep regularity are of increasing importance to the sleep health research field. We synthesized 22 articles that investigated sleep timing and/or sleep regularity with cancer risk and the general findings remain inconclusive. This review highlights the heterogeneity in how sleep timing and sleep regularity are assessed. For example, different methods were used to measure chronotype, social jetlag, and sleep midpoint across studies and reference groups varied across all measures. Future research should standardize measures on how to quantify sleep timing and sleep regularity given the pending consensus on this matter [7]. Efforts that comprehensively assess sleep timing and sleep regularity, as well as other sleep parameters (i.e., sleep quality) individually and together, are needed to characterize the relationship between multiple dimensions of sleep and cancer risk. Replication studies in diverse populations are needed. The incorporation of repeated sleep measures may more accurately demonstrate the potential relationship of sleep and cancer. In conclusion, although some studies have found later sleep timing and sleep timing variability to be associated with increased cancer risk, current evidence linking these potential relationships remains inconclusive.

Supplementary material

Supplementary material is available at *SLEEP* online.

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Data Availability

Data available on request from the authors.

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