

# Risk for Suicide and Homicide Peaks at Night:

# Findings From the National Violent Death Reporting System, 35 States, 2003–2017

Andrew S. Tubbs, MD, PhD; Fabian-Xosé Fernandez, PhD; Elizabeth B. Klerman, MD, PhD; Jordan F. Karp, MD; Mathias Basner, MD, PhD; Subhajit Chakravorty, MD; Ellen Watkins, BS; Michael L. Perlis, PhD; and Michael A. Grandner, PhD, MTR

#### Abstract

**Objective:** The Mind after Midnight hypothesis proposes that nocturnal wakefulness increases the risk for dysregulated behaviors. Prior studies highlight a greater risk for suicide at night after adjusting for population wakefulness. How this risk varies hour to hour, differs across subgroups, or applies to other behaviors is unknown.

**Methods:** Data on 78,647 suicides and 50,526 homicides from the National Violent Death Reporting System were combined with population wakefulness data for 2003–2017 from the American

Time Use Survey. Hourly incident risk ratios (IRRs) were estimated after adjusting for population wakefulness. Two-way analysis of variances identified significant time-by-subgroup interactions that were quantified in post hoc analyses.

**Results:** Suicide counts peaked at 12:00 PM, while homicide counts peaked at 10:00– 11:00 PM. Adjusting for demographics and population wakefulness revealed a 5-fold greater risk for suicide at 3:00 AM (aIRR: 5.20 [4.74–5.70]) and an 8-fold greater risk for homicide at 2:00 AM (aIRR: 8.04 [6.35–10.2]). Hourly risk for suicide varied by age, ethnicity, blood alcohol level, and current partner conflict. Hourly risk for homicide varied by sex and blood alcohol level.

**Conclusions:** Risk for suicide and homicide is greater at night than expected based on the number of people awake at that time. Nighttime risk was greater among young adults and those intoxicated with alcohol, but not among those with a history of suicidal ideation or attempts. Further research should evaluate mechanisms of risk and confirm these findings at an individual level.

J Clin Psychiatry 2024;85(2):23m15207

Author affiliations are listed at the end of this article.

D isrupted sleep may increase the risk for suicide<sup>1,2</sup> through chronically insufficient or poor-quality sleep. The Mind after Midnight hypothesis<sup>3,4</sup> proposes that disrupted sleep also creates acute risk for dysregulated behaviors through nocturnal wakefulness (ie, prolonged wakefulness during the habitual sleep period). Regardless of the cause, nocturnal wakefulness drives risk by combining circadian processes that promote sleep at night with sleep deprivation–induced cognitive deficits. Nocturnal wakefulness also occurs at times of psychosocial isolation—friends and family are asleep, and community services are shuttered. These factors may enable vulnerable individuals to progress from maladaptive thoughts to impulsive, dysregulated behaviors.

Evidence for the Mind after Midnight hypothesis partially derives from 24-hour patterns of suicide timing and population wakefulness. Although raw hourly suicide counts peak around noon (see Perlis et al<sup>4</sup> for a comprehensive review), adjusting raw counts for the number of people awake (and thus capable of attempting suicide) revealed a 3-fold peak in incident suicide risk between midnight and 6:00 AM. Subsequent work has replicated this nighttime suicide risk in Australia,<sup>5</sup> identified greater nighttime risk for military service members,<sup>6</sup> and measured increased nocturnal wakefulness among those with suicidal ideation.<sup>7-9</sup>

Nocturnal wakefulness may affect several cognitive domains. Positive mood is minimal and negative mood is maximal during this time,<sup>10–13</sup> risk/reward processing is altered due to changes in subcortical reward circuits,<sup>14–17</sup> and executive functions are compromised owing to circadian changes in cortical connectivity and

## Scan Now

Cite and Share this article at Psychiatrist.com

#### **Editor's Note**

We encourage authors to submit papers for consideration as a part of our Focus on Suicide section. Please contact Philippe Courtet, MD, PhD, at pcourtet@psychiatrist.com.

### **Clinical Points**

- Risk for death by suicide and homicide is elevated at night, particularly among younger age groups and individuals intoxicated with alcohol.
- Wakefulness at night may predispose individuals to dysregulated behaviors, and interventions to reduce or mitigate disrupted sleep may reduce risk.

mounting sleep pressure in the frontal cortex.<sup>18-24</sup> Such deficits are correlated with suicide risk: individuals with multiple suicide attempts (or at risk for suicide) have impaired executive function,<sup>25-28</sup> and the suicide crisis syndrome may emerge from disrupted neurocognition.<sup>29,30</sup> Individuals who cannot regulate their emotions, manage risk, or inhibit their impulses may also resort to violence<sup>31,32</sup> or inadvertently place themselves in danger. Few studies have examined timeof-day trends in violent crime. Although some analyses highlight a peak in homicides between 6:00 PM and 6:00 AM<sup>33-35</sup> and approximately 55% of all violent crimes occur between 7:00 PM and 7:00 AM,<sup>36</sup> hour-byhour incidence data remain limited. If homicide and suicide share a nighttime peak in incidence, then these behaviors may share nocturnal wakefulness as a common risk factor.

Subgroup variations in overnight suicide risk may suggest potential mechanisms. The prefrontal cortex does not fully mature until age 25,<sup>37</sup> so nocturnal wakefulness likely strains underdeveloped executive functions in adolescents and young adults. Circadian misalignment in youth also appears to dysregulate risk/reward processing, particularly as it relates to substance use.14,38,39 Alcohol impairs executive functions40 and may increase nighttime risk for suicide<sup>41</sup> or homicide. The interpersonal theory of suicide<sup>42</sup> and the suicide crisis syndrome<sup>30</sup> emphasize disrupted interpersonal relations as potential precipitants of suicidal behavior, so individuals experiencing interpersonal conflict may be at similar risk for dysregulated behaviors. Finally, greater negative mood during nocturnal wakefulness may place a particular burden on individuals with preexisting mood disorders.

To gain further insights into subgroup variations in overnight risk for suicide and homicide, the present study leveraged 15 years of US national archival data to (1) examine the population wakefulness–adjusted risk for suicide and homicide on an hour-by-hour basis and (2) explore these patterns in risk among multiple demographic and clinical subgroups. Based on prior publications and the above reasoning, it was hypothesized that hour-by-hour risk for suicide and homicide would peak during the night (11:00 PM to 5:00 AM), with risk further increased in individuals with underdeveloped or compromised executive function (eg, young adults and intoxicated adults), current depressed mood, or experiencing current partner conflict.

#### **METHODS**

#### Datasets

Case data were acquired from the National Violent Death Reporting System (NVDRS) on 78,647 suicides and 50,526 homicides between 2003 and 2017. Data are derived from law enforcement reports, death certificates, and medical examiner reports that are maintained by the Centers for Disease Control and Prevention. The NVDRS began with data from 6 states and by 2017 included 35 states plus the District of Columbia and Puerto Rico. Data can be accessed by application to the NVDRS Restricted Access Database (www.cdc.gov/ violenceprevention/datasources/nvdrs/dataaccess.html). Suicides from 2003 to 2010 were previously analyzed,<sup>43,44</sup> but those reports did not specifically address hour-by-hour incident risks. These data were combined with new data to improve statistical power for detecting between-group differences.

Population estimates of sleep/wake timing were derived from the American Time Use Survey (ATUS, https://www.bls.gov/tus) for the same years. The ATUS is a weighted, nationally representative telephone-based survey conducted by the US Bureau of Labor and Statistics among 10,000 US adults annually. Respondents recorded their activities, including sleeping, in 30-minute epochs for 24 hours. Population estimates of epoch-by-epoch wakefulness were then calculated by age, sex, race, and ethnicity for 2003–2017 and averaged to obtain hour-by-hour estimates of population wakefulness (ie, minutes awake per hour).

The University of Arizona Institutional Review Board determined that this project involved de-identified human subject data and was thus exempt from further review.

#### Variables

NVDRS case variables included time and date of fatal injury, age, sex, race, ethnicity, military service, autopsy testing for alcohol and cannabis, current depressed mood, current partner conflict, and prior suicidal ideation or attempts. Not all data were available for all cases. Military service was defined as current or past military service and categorized as no or yes. Autopsy data on blood alcohol levels (BALs) were categorized as none, <80 mg/dL, or ≥80 mg/dL. Cannabis was categorized as absent or present. Current depressed mood was defined as "victim was perceived by self or others to be depressed at the time of injury" ("no or unknown" vs "yes"), and current partner conflict was defined as "problems with a current or former intimate partner appear to have contributed to the [suicide/homicide]" ("no or unknown" vs "yes"). Suicides were characterized by a history of suicidal ideation ("no or unknown" vs "yes") or a suicide attempt ("no or unknown" vs "yes").

#### Statistical Analyses

All analyses were conducted in R (version 4.2.1, R Foundation for Statistical Computing, Vienna, Austria); the code is available at https://github.com/atubbs-sleep. For descriptive analyses, the time was divided into 4 categories consistent with prior reports<sup>8,9,43</sup>: morning: 0500–1059, afternoon: 1100–1659, evening: 1700–2259, and night: 2300–0459. Inferential analyses were conducted in 2 steps for suicides and homicides: (1) hour-by-hour incident risk for the whole sample and (2) significant time-by-subgroup variations. Across analyses, effect coding was used so that clock hour estimates were compared to the grand mean across clock hours rather than a reference clock hour.

Analysis 1: hour-by-hour incident risk for the whole sample. Individual NVDRS cases were tabulated into hourly case counts by hour of fatal injury, age, sex, race, and ethnicity. Counts were then matched with estimated population wakefulness by age, sex, and race/ethnicity from the ATUS. Robust Poisson multivariable models estimated the incident risk ratio (IRR) with clock hour as the predictor/independent variable, case count as the outcome/dependent variable, and population wakefulness as an exposure/offset term. Model 1 was unadjusted, and Model 2 was adjusted for age, sex, race, ethnicity, military service, current depressed mood, current partner conflict, prior suicidal ideation, and prior suicide attempt. IRRs are reported as point estimates and 95% confidence intervals (eg, IRR [95% CI]).

**Analysis 2: significant time-by-subgroup interactions.** Two-way analysis of variances examined whether any covariates had significant interactions with time of day in predicting risk. All tests were adjusted for age, sex, race, and ethnicity, where appropriate, and *P* values were adjusted for multiple comparisons according to the familywise error rate. Marginal time-by-subgroup effects were examined for significant interactions.

#### **RESULTS**

The characteristics of individuals who died by suicide or homicide are presented in Tables 1 and 2, respectively. High missingness was noted for BAL (suicide: 50.5% and homicide: 37.5%) and cannabis (suicide: 68.5% and homicide: 62.4%). Suicide case counts were highest during the afternoon (N = 24,573, 1100–1659) and lowest at night (N = 14,833, 2300–0459). For homicide, case counts were highest at night (N = 17,997, 2300–0459) and lowest in the morning (N = 6,434, 0500–1059). IRRs were elevated between 12:00 AM and 5:00 AM for both suicide (Figure 1A) and homicide (Figure 1B) compared to the 24-hour average risk. The peak unadjusted suicide risk (Model 1) was 526% greater at 2:00 AM (IRR: 5.26 [3.26–8.47]), and the peak adjusted suicide risk (Model 2) was 520% greater at 3:00 AM (aIRR: 5.20 [4.74–5.70]). For death by homicide, the peak unadjusted homicide risk was 980% greater at 2:00 AM (IRR: 9.80 [5.69–16.9]), and the peak adjusted homicide risk was 804% greater at 2:00 AM (aIRR: 8.04 [6.35–10.2]).

Time-of-day-by-subgroup interactions in suicide risk were observed for age (P < .001), ethnicity (P = .014), BAL (P < .001), and current partner conflict (P < .001)(Figure 2). Compared to individuals aged 45-54 years (the largest age group), risk peaked around 4:00 AM for individuals aged 15-24 (aIRR: 3.11 [2.39-4.05]) and 25-34 (aIRR: 2.15 [1.63-2.86]) years. Individuals aged 35-44 years showed modest peaks in risk at 2:00 AM (aIRR: 1.40 [1.03-1.91]) and 3:00 AM (aIRR: 1.49 [1.12–1.99]), but they, like those aged 55–64 years, were overall not substantially different from individuals aged 45-54 years. Finally, there was a peak in risk at 6:00 AM for individuals aged 65–74 (aIRR: 1.78 [1.40-2.25]) and 75+ (aIRR: 2.25 [1.69-3.00]) years. Hispanic individuals had elevated risk between 10:00 PM and 3:00 AM with a maximal 232% increase at 2:00 AM (aIRR: 2.32 [1.67-3.23]). Individuals with a positive BAL showed increased risk between 8:00 PM and 4:00 AM; those with a BAL less than 80 mg/dL had a peak risk of 168% (aIRR: 1.68 [1.40-2.01] at 12:00 AM), and those with a BAL of 80 mg/dL had a peak risk of 233% (aIRR: 2.33 [1.88–2.89] at 1:00 AM). Additionally, individuals with a positive BAL had reduced daytime risk (8:00 AM-4:00 PM). Individuals who had a current partner conflict had an elevated risk from 8:00 pm to 11:00 pm and from 1:00 AM to 3:00 AM with a peak risk at 11:00 PM (aIRR: 1.53 [1.24-1.89]).

Time-of-day-by-subgroup interactions in risk for death by homicide were observed for sex (P < .001) and BAL (P < .001) (Figure 3). Female homicide victims were more likely to die between 5:00 AM and 9:00 AM (peak risk at 7:00 AM, IRR: 1.90 [1.42–2.53]) and less likely to die between 7:00 PM and 11:00 PM. Individuals with a positive BAL showed increased risk between 11:00 PM and 5:00 AM with a peak risk at 2:00 AM (<80 mg/dL, IRR: 2.17 [1.46–3.23];  $\geq$  80 mg/dL, IRR: 2.96 [2.12–4.14]). No other subgroups showed significant differences in time-of-day risk.

#### **DISCUSSION**

Adjusting for population wakefulness yielded a 5-fold greater risk for suicide and an 8-fold greater risk for homicide from 2:00 AM to 3:00 AM when compared to the

#### Table 1.

# Demographic and Clinical Characteristics of Suicide Victims in the NVDRS by Time of Day

Characteristic	Total	Morning <sup>a</sup>	<b>Afternoon</b> <sup>a</sup>	<b>Evening</b> <sup>a</sup>	Night <sup>a</sup>
N	78,647	18,062	24,573	21,179	14,833
Age, y					
15–24	11,114 (14.3%)	1,918 (10.7%)	3,080 (12.7%)	3,260 (15.7%)	2,856 (19.4%)
25–34	13,311 (17.1%)	2,628 (14.6%)	3,589 (14.8%)	3,743 (18.0%)	3,351 (22.8%)
35–44	13,725 (17.6%)	2,864 (16.0%)	4,118 (16.9%)	4,002 (19.2%)	2,741 (18.6%)
45–54	15,297 (19.7%)	3,514 (19.6%)	5,047 (20.8%)	4,250 (20.4%)	2,486 (16.9%)
55–64	11,253 (14.5%)	2,867 (16.0%)	3,824 (15.7%)	2,871 (13.8%)	1,691 (11.5%)
65–74	6,622 (8.5%)	1,940 (10.8%)	2,373 (9.8%)	1,509 (7.2%)	800 (5.4%)
75+	6,462 (8.3%)	2,210 (12.3%)	2,265 (9.3%)	1,191 (5.7%)	796 (5.4%)
Unknown	863	121	277	353	112
Sex					
Male	63,979 (81.4%)	14,871 (82.3%)	19,767 (80.4%)	17,146 (81.0%)	12,195 (82.2%)
Female	14,664 (18.6%)	3,190 (17.7%)	4,806 (19.6%)	4,030 (19.0%)	2,638 (17.8%)
Unknown	4	1	0	3	0
Race					
White	68,528 (87.3%)	15,823 (87.7%)	21,656 (88.3%)	18,452 (87.3%)	12,597 (85.3%)
Black	5,838 (7.4%)	1,342 (7.4%)	1,697 (6.9%)	1,582 (7.5%)	1,217 (8.2%)
Asian	1,133 (1.4%)	237 (1.3%)	356 (1.5%)	304 (1.4%)	236 (1.6%)
Other race	2,991 (3.8%)	637 (3.5%)	824 (3.4%)	807 (3.8%)	723 (4.9%)
Unknown	157	23	40	34	60
Ethnicity					
Non-Hispanic	74,028 (94.9%)	17,044 (95.2%)	23,291 (95.5%)	19,915 (94.8%)	13,778 (93.7%)
Hispanic	3,981 (5.1%)	861 (4.8%)	1,090 (4.5%)	1,100 (5.2%)	930 (6.3%)
Unknown	638	157	192	164	125
Military service					
No	59,188 (79.6%)	13,134 (76.8%)	18,306 (78.4%)	16,433 (81.6%)	11,315 (82.2%)
Yes	15,194 (20.4%)	3,971 (23.2%)	5,051 (21.6%)	3,716 (18.4%)	2,456 (17.8%)
Unknown	4,265	957	1,216	1,030	1,062
Blood alcohol level					
No alcohol	23,787 (61.1%)	5,943 (71.7%)	8,562 (71.7%)	5,997 (53.5%)	3,285 (43.7%)
< 80 mg/dL	3,560 (9.1%)	704 (8.5%)	994 (8.3%)	1,059 (9.5%)	803 (10.7%)
≥ 80 mg/dL	11,609 (29.8%)	1,639 (19.8%)	2,389 (20.0%)	4,144 (37.0%)	3,437 (45.7%)
Unknown	39,691	9,776	12,628	9,979	7,308
Cannabis					
Not present	20,835 (84.1%)	4,754 (85.3%)	6,562 (85.4%)	5,743 (84.6%)	3,776 (79.8%)
Present	3,942 (15.9%)	817 (14.7%)	1,126 (14.6%)	1,043 (15.4%)	956 (20.2%)
Unknown	53,870	12,491	16,885	14,393	10,101
Current depressed mood	26,403 (33.6%)	6,147 (34.0%)	8,670 (35.3%)	7,041 (33.2%)	4,545 (30.6%)
Current partner conflict	22,658 (28.8%)	4,347 (24.1%)	6,164 (25.1%)	6,936 (32.7%)	5,211 (35.1%)
Prior suicidal ideation	11,375 (14.5%)	2,457 (13.6%)	3,237 (13.2%)	3,256 (15.4%)	2,425 (16.3%)
Prior suicide attempt	12,125 (15.4%)	2,507 (13.9%)	3,860 (15.7%)	3,418 (16.1%)	2,340 (15.8%)

<sup>a</sup>Morning: 0500–1059; afternoon: 1100–1659; evening: 1700–2259; night: 2300–0459. Abbreviation: NVDRS = National Violent Death Reporting System.

24-hour average risk. Nighttime risk was greater among adolescents/young adults, those intoxicated with alcohol, and those experiencing current partner conflict, but not among those who used cannabis or were currently depressed. This study supports the Mind after Midnight hypothesis in 3 ways. First, it augments prior work by estimating the hour-by-hour change in overnight suicide risk. Second, it expands the scope of risk to include death by homicide, a distinct behavior from suicide. Third, the subgroup analyses yield circumstantial evidence for potential neurobiological mechanisms of risk. Suicide and homicide share little in common, but their highly concordant overnight risk patterns suggest a common feature: nocturnal wakefulness. Insomnia, nightmares, substance use, shift work, and medical disease may all cause nocturnal wakefulness, but being awake when reason sleeps<sup>4,44</sup> likely carries a distinct risk for dysregulated behaviors. The Mind after Midnight<sup>3</sup> hypothesis highlights executive dysfunction, reward processing, and mood dysregulation as possible neurocognitive mechanisms of risk, and subgroup findings offer some insights into which mechanisms may be at work.

#### Table 2.

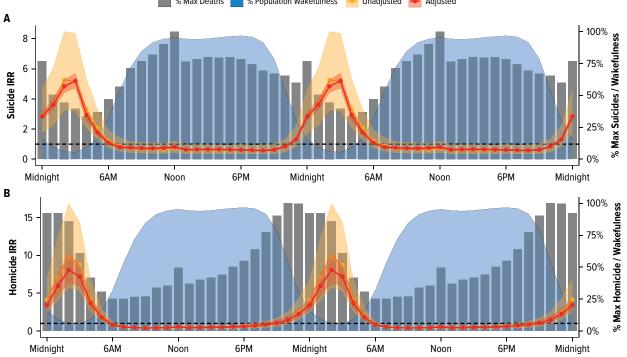
# Demographic and Clinical Characteristics of Homicide Victims in the NVDRS by Time of Day

Characteristic	Total	Morning <sup>a</sup>	<b>Afternoon</b> <sup>a</sup>	<b>Evening</b> <sup>a</sup>	Night <sup>a</sup>
N	50,526	6,434	9,465	16,630	17,997
Age, y					
15–24	15,250 (31.5%)	1,385 (23.0%)	2,714 (30.5%)	5,297 (33.0%)	5,854 (33.4%)
25–34	14,355 (29.6%)	1,659 (27.5%)	2,388 (26.8%)	4,550 (28.4%)	5,758 (32.9%)
35–44	8,514 (17.6%)	1,198 (19.9%)	1,483 (16.7%)	2,819 (17.6%)	3,014 (17.2%)
45–54	5,591 (11.5%)	863 (14.3%)	1,117 (12.6%)	1,899 (11.8%)	1,712 (9.8%)
55-64	2,831 (5.8%)	491 (8.1%)	667 (7.5%)	917 (5.7%)	756 (4.3%)
65–74	1,190 (2.5%)	240 (4.0%)	316 (3.6%)	347 (2.2%)	287 (1.6%)
75+	755 (1.6%)	198 (3.3%)	213 (2.4%)	209 (1.3%)	135 (0.8%)
Unknown	2,040	400	567	592	481
Sex					
Male	41,597 (82.3%)	4,741 (73.7%)	7,507 (79.3%)	14,036 (84.4%)	15,313 (85.1%)
Female	8,928 (17.7%)	1,693 (26.3%)	1,958 (20.7%)	2,594 (15.6%)	2,683 (14.9%)
Unknown	1	0	0	0	1
Race					
White	18,079 (36.0%)	2,829 (44.2%)	3,630 (38.5%)	5,768 (34.8%)	5,852 (32.7%)
Black	28,779 (57.2%)	3,114 (48.6%)	5,197 (55.1%)	9,667 (58.4%)	10,801 (60.4%)
Asian	586 (1.2%)	99 (1.5%)	102 (1.1%)	207 (1.3%)	178 (1.0%)
Other race	2,830 (5.6%)	362 (5.7%)	496 (5.3%)	912 (5.5%)	1,060 (5.9%)
Unknown	252	30	40	76	106
Ethnicity					
Non-Hispanic	43,882 (87.9%)	5,550 (87.5%)	8,298 (88.9%)	14,468 (88.1%)	15,566 (87.4%)
Hispanic	6,020 (12.1%)	790 (12.5%)	1,033 (11.1%)	1,961 (11.9%)	2,236 (12.6%)
Unknown	624	94	134	201	195
Military service					
No	44,519 (93.8%)	5,562 (92.2%)	8,361 (93.6%)	14,768 (94.0%)	15,828 (94.3%)
Yes	2,947 (6.2%)	470 (7.8%)	572 (6.4%)	949 (6.0%)	956 (5.7%)
Unknown	3,060	402	532	913	1,213
Blood alcohol level					
No alcohol	19,518 (61.8%)	2,588 (71.3%)	4,346 (77.8%)	7,018 (66.7%)	5,566 (46.9%)
< 80 mg/dL	4,046 (12.8%)	363 (10.0%)	506 (9.1%)	1,250 (11.9%)	1,927 (16.2%)
≥ 80 mg/dL	8,040 (25.4%)	681 (18.8%)	734 (13.1%)	2,251 (21.4%)	4,374 (36.9%)
Unknown	18,922	2,802	3,879	6,111	6,130
Cannabis					
Not present	11,287 (59.4%)	1,534 (64.2%)	2,153 (58.6%)	3,603 (57.9%)	3,997 (59.5%)
Present	7,718 (40.6%)	857 (35.8%)	1,523 (41.4%)	2,620 (42.1%)	2,718 (40.5%)
Unknown	31,521	4,043	5,789	10,407	11,282
Current depressed mood	151 (0.3%)	34 (0.5%)	39 (0.4%)	45 (0.3%)	33 (0.2%)
Current partner conflict	869 (1.7%)	157 (2.4%)	201 (2.1%)	266 (1.6%)	245 (1.4%)
Prior suicidal ideation	10 (0.0%)	1 (0.0%)	1 (0.0%)	2 (0.0%)	6 (0.0%)
Prior suicide attempt	27 (0.1%)	7 (0.1%)	6 (0.1%)	7 (0.0%)	7 (0.0%)
-	1100 10		0 0050	2202 2452	

<sup>a</sup>Morning: 0500–1059; afternoon: 1100–1659; evening: 1700–2259; night: 2300–0459. Abbreviation: NVDRS = National Violent Death Reporting System.

Nighttime suicide risk varied by age and alcohol intoxication, 2 groups with reduced executive function. Individuals aged 15–24 years experienced a 3-fold greater nighttime risk for suicide, possibly because nocturnal wakefulness may test and overwhelm the limited executive functions of an immature prefrontal cortex.<sup>37</sup> As the prefrontal cortex matures, executive functions should strengthen and risk should diminish, as was seen in the smaller increase in risk among those aged 25–34 years. Risk for homicide did not vary by age, but young adults accounted for more than half of all homicide victims, so there may have been insufficient age-related variance to detect a pattern. The fact that homicide deaths were so highly concentrated among young adults, including overnight deaths, suggests that young adults may place themselves in positions of danger (possibly due to impaired risk/reward assessment) that older adults would avoid. Indeed, circadian misalignment appears to increase sensitivity to reward anticipation and reduce sensitivity to reward loss, particularly in young adults.<sup>14–17,45</sup> There was also an unexpected finding of increased suicide risk among older adults at 6:00 AM. This peak is outside the hypothesized window of risk and may stem from sleep inertia, a period





<sup>a</sup>Raw case counts for suicide (A) and homicide (B) from the NVDRS were binned by clock hour and scaled to the percent of the maximum bin count (grey bars). Percent population wakefulness for each clock hour was then estimated from the ATUS (light blue shaded area). Robust Poisson regression models then estimated the IRR of suicide and homicide by clock hour. Models are unadjusted (yellow) and adjusted for age, sex, race, ethnicity, military service, currently depressed, current partner conflict, prior suicidal ideation, and prior suicide attempts (red). The shaded bands around the IRRs represent 95% Cls. The black dotted horizontal line represents an IRR of 1, meaning neither increased nor decreased risk compared to the 24-hour average. Data are double-plotted for clarity.

Abbreviations: ATUS = American Time Use Survey, IRR = incidence risk ratio, NVDRS = National Violent Death Reporting System.

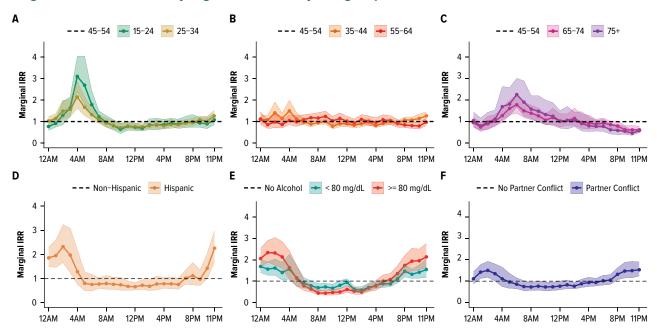
of impaired alertness and cognition following arousal. Older adults are more vulnerable to sleep inertia<sup>46</sup> and may experience greater deficits in executive function upon awakening that contribute to heightened suicide risk.

Alcohol intoxication correlated with greater nighttime risk for suicide and homicide. Less than half of suicides involve alcohol intoxication,47 but alcohol acutely increases suicide risk<sup>48,49</sup> and represents a potent risk factor for some individuals. Alcohol also disrupts executive functions,40 and individuals may use alcohol to enhance positive affect and/or combat negative affect,<sup>50-53</sup> which decline and rise at night, respectively.<sup>10-13</sup> Under the Mind after Midnight hypothesis, an individual experiencing nocturnal wakefulness may experience greater-than-normal negative mood (emotional dysregulation), overestimate the relief that consuming alcohol might provide (impaired risk/reward processing), and fail to control their impulses to drink (executive dysfunction). Although drinking may provide short-term relief, these benefits dissolve into greater mood dysregulation and

behavioral disinhibition, thus increasing the risk for suicide or homicide. Indeed, Chakravorty and colleagues<sup>41</sup> found that most alcohol-related suicides occur at night, and the 2.3-fold greater rise in nighttime risk for suicide among those with an elevated BAL may represent the synergistic effects of nocturnal wakefulness and acute intoxication. Similar mechanisms may affect homicide victims by impairing their ability to perceive or mitigate the risk of imminent bodily harm. Lack of BAL data for homicide perpetrators, however, limits further interpretation.

Two groups demonstrated disproportionate overnight risk that is not clearly related to neurocognition. Hispanic individuals showed a roughly 2-fold greater risk for suicide from 11:00 PM to 3:00 AM than non-Hispanic individuals. Hispanic individuals may experience greater risk from nocturnal wakefulness due to more prevalent sleep disturbances, sleep apnea, and insufficient sleep,<sup>54–56</sup> as well as differences in rates of shift work or neighborhood environmental conditions (eg, noise). Women experienced an elevated risk for death by homicide between 4:00 AM and 9:00 AM. This finding is

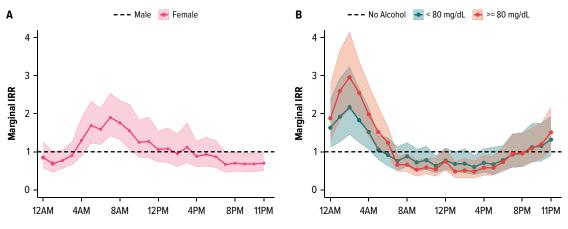
Figure 3.



#### Figure 2. Marginal IRRs for Suicide by Significant Time-by-Subgroup Interactions<sup>a</sup>

<sup>a</sup>Hourly IRRs varied by age (A–C), ethnicity (D), blood alcohol level (E), and current partner conflict (F). The reference groups are individuals aged 45–54 years (the age group with the greatest number of suicides), non-Hispanics, those with a negative blood alcohol level, and those with no current partner conflict. The shaded bands reflect 95% CIs, and the black dotted horizontal line represents an IRR of 1. Abbreviation: IRR = incidence risk ratio.

### Marginal IRRs for Homicide by Significant Time-by-Subgroup Interactions<sup>a</sup>



<sup>a</sup>Hourly IRRs varied by sex (A) and blood alcohol level (B). The reference groups were males and those with a negative blood alcohol level. The shaded bands reflect 95% CIs, and the black dotted horizontal line represents an IRR of 1. Abbreviation: IRR = incidence risk ratio.

difficult to interpret; the risk period is substantially delayed compared to other groups, and there are no clear neurobiological reasons why women would experience greater risk at this time. A more detailed examination of individual homicide cases may be needed to identify sex-specific factors that contribute to risk, although one possibility may involve opportunity to approach potential victims during early working hours.

Several null results are worth discussing. First, in contrast with McCarthy and colleagues,<sup>6</sup> individuals with military service did not differ substantially in their

suicide risk patterns. There are technical differences between studies, but the most likely explanation is methodological: McCarthy et al analyzed veterans and civilians separately rather than as a formal interaction, which is the recommended approach.<sup>57</sup> Second, there was no significant interaction between cannabis intoxication and suicide timing. Cannabis testing may reflect consumption up to 30 days previously, and thus a positive result may not reflect acute intoxication. Acute cannabis intoxication is known to impair aspects of executive functions, especially impulse control, and thus an analysis of subjects with acute intoxication may yield different results.<sup>58,59</sup> Third, there was no difference in risk among those with and without prior suicide attempts. One explanation is methodological-cases where data on prior suicide attempts were missing were inappropriately categorized as not having prior suicide attempts. Alternatively, past suicidal ideation/attempts are distal risk factors that do not contribute to acute changes in suicide risk. Finally, individuals with current depressed mood did not experience increased nighttime suicide risk. One possibility is that the NVDRS measure of current depressed mood may be too broad or inaccurate to capture individuals experiencing dysregulated mood at the time of death. This is especially relevant given the lack of psychiatric diagnoses (eg, bipolar disorder or borderline personality disorder). Alternatively, nocturnal wakefulness may create an antidepressant effect through partial or total sleep deprivation.<sup>60,61</sup> Mood regulation may vet contribute to risk during nocturnal wakefulness, but its effect was not evident here.

The neurobiological mechanisms discussed here do not act in isolation. Disrupted sleep is a transdiagnostic marker of psychiatric illness,62 and nocturnal wakefulness in vulnerable individuals may further increase the risk for dysregulated behaviors. Bipolar disorder, for instance, is characterized by altered rest/ activity patterns,63 and acute loss of sleep due to nocturnal wakefulness may precipitate mania.64 The hypervigilance and nightmares of posttraumatic stress disorder may produce nocturnal wakefulness65,66 in addition to predisposing patients to dysregulated emotional and behavioral responses. In both cases, disrupted sleep may play a role in suicidal behaviors.<sup>67-69</sup> Nocturnal wakefulness also occurs during an altered psychosocial landscape in which social services are closed, friends and family are asleep, and other coping strategies are unavailable. The modest increase in overnight suicide risk among individuals with partner conflicts may emerge from limited emotional selfregulation and feelings of thwarted belongingness and social isolation that contribute to suicidal thinking.42,70 Nocturnal wakefulness may also create inadvertent opportunities for dysregulated behaviors: a violent altercation may progress to homicide because no one is present to call law enforcement, or a teenager

may attempt suicide because their caregivers are asleep. Indeed, social services that might prevent dysregulated behaviors (eg, calling a therapist or peer support and escaping to a friend's house) may be unavailable at night.

The strengths of this study include the sample size and breadth of the NVDRS data as well as representative estimates of wakefulness from the ATUS. That said, the present study has a few limitations. Individuals with suicidal behavior are more likely to be awake at night,<sup>8,9</sup> and estimating population wakefulness from the ATUS may skew risk profiles since habitual sleep timing may differ by the presence of suicidal thinking. Future studies should derive estimates of wakefulness from more representative sources. Additionally, the NVDRS occasionally coded missing data as a negative response (ie, current depressed mood or partner conflict, prior suicidal ideation, or suicide attempt). This inappropriately obscures missingness and raises statistical power, so comparisons on these factors may not be accurate. Indeed, substantial missingness requires caution in interpreting some results (eg, alcohol and cannabis), as missing data may obscure significant patterns in hour-by-hour risk.

The outcomes presented here should be considered population-level estimates of risk that cannot be applied directly to individual cases because of the limitations of the available case data. As highlighted above, psychiatric disorders involve substantial disruptions in sleep and increase risk for suicide, and thus affected individuals may have greater vulnerability to nocturnal wakefulness. However, data related to psychiatric diagnoses within the NVDRS are derived from forensic reports (and thus susceptible to reporting bias) and demonstrated severe missingness (80% or more for all diagnoses). The consequence of this is that (1) the data do not accurately reflect who does or does not have a particular mental condition, and (2) there are insufficient data to adequately evaluate variations in hourly risk patterns for these conditions. Psychotropic substances ranging from drugs of abuse to prescription medications (particularly benzodiazepines and sedative-hypnotics) can also affect both sleep/wake patterns and suicide risk.<sup>71</sup> Autopsy testing for substances beyond alcohol and cannabis was not reported until 2013, however, and thus these data are not sufficiently representative (or present large enough subsamples) for further analysis. Finally, individual-level data on sleep continuity, chronotype, medical diagnoses, and socioeconomic status were not available for analysis, even though these factors are known to affect sleep and suicide risk.

#### Conclusion

Nearly 19% of suicides and 36% of homicides occur at night, a time when individuals may be sleep-deprived and awake when their circadian rhythms are promoting sleep. In accordance with the Mind after Midnight hypothesis, the present study demonstrated a disproportionate nighttime risk for suicide and homicide after accounting for population wakefulness. Additional studies are needed to clarify whether evidence-based interventions to improve sleep and reduce nocturnal wakefulness can reduce risks and, in so doing, prevent these tragic outcomes.

#### Article Information

Published Online: May 29, 2024. https://doi.org/10.4088/JCP.23m15207 © 2024 Physicians Postgraduate Press, Inc.

Submitted: November 30, 2023; accepted February 19, 2024.

**To Cite:** Tubbs AS, Fernandez F-X, Klerman EB, et al. Risk for suicide and homicide peaks at night: findings from the National Violent Death Reporting System, 35 states, 2003–2017. *J Clin Psychiatry*. 2024;85(2):23m15207.

Author Affiliations: Department of Psychiatry, University of Arizona College of Medicine–Tucson, Tucson, Arizona (Tubbs, Karp, Watkins, Grandner); Evelyn F. McKnight Brain Institute, Department of Psychology, University of Arizona, Tucson, Arizona (Fernandez): Department of Neurology, Massachusetts General Hospital, Boston, Massachusetts (Klerman); Division of Sleep Medicine, Harvard Medical School, Boston, Massachusetts (Klerman); Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, Pennsylvania (Basner, Chakravorty, Perlis).

Drs Tubbs and Fernandez are co-first authors, and Drs Perlis and Grandner are cosenior authors.

**Corresponding Author:** Andrew S. Tubbs, MD, PhD, Department of Psychiatry, University of Arizona College of Medicine–Tucson, 1501 N. Campbell Ave, Ste 7326, Tucson, AZ 85724 (atubbs@arizona.edu).

Relevant Financial Relationships: In the past 2 years, Dr Tubbs reports fees from the American Academy of Sleep Medicine and the American Council of Life Insurers. Dr Klerman reports consulting for the American Academy of Sleep Medicine Foundation, Circadian Therapeutics, National Sleep Foundation, Sleep Research Society Foundation, and Yale University Press; receives travel support from the European Biological Rhythms Society, EPFL Pavilions, and World Sleep Society; and serves on Scientific Advisory Board (unpaid) for Chronsulting. Her partner is the founder, director, and chief scientific officer of Chronsulting. Dr Karp receives grant support from Janssen. He has the potential for equity in Aifred Health for scientific advising. He has served as a scientific advisor to Biogen. He receives remuneration from the Journal of Clinical Psychiatry and the American Journal of Geriatric Psychiatry for service on the editorial boards. Dr Chakravorty reports research support from the NSF and NeuroFlow Inc. Dr Perlis reports research support from Axsome, grant support from NIH, and consulting income from Nexalin, Anavex, and Avecho and that he has participated in CE/CME and is a founding partner in Hypknowledge LLC. Dr Grandner reports consulting fees from Idorsia, Eisai, Jazz Pharmaceuticals, Merck, Fitbit, Natrol, SmartyPants Vitamins, Athleta, National Sleep Foundation, American Sleep Medicine Foundation, and Canyon Ranch; received grants from NIH, Kemin Foods, Jazz Pharmaceuticals, and CeraZ; received publishing royalties from Elsevier; and received speaking honoraria and/or travel support from New York University, University of Maryland, Stavis & Cohen, University of Miami, Clinical Education Alliance, and Jazz Pharmaceuticals. Drs Fernandez and Basner and Ms Watkins have no disclosures.

Funding/Support: The work presented in this article received no direct funding.

**Previous Presentation:** Data previously presented at the 2022 SLEEP meeting; June 4–8, 2022; Charlotte, North Carolina, and the 2023 SLEEP meeting; June 3–7, 2023; Indianapolis, Indiana.

Acknowledgments: The authors would like to acknowledge Catie Holt and Sabrina Arevalo, BS, at the University of Arizona for their assistance in preparing this manuscript.

ORCID: Andrew S. Tubbs: https://orcid.org/0000-0003-3799-5972; Michael A. Grandner: https://orcid.org/0000-0002-4626-754X; Fabian-Xosé Fernandez: https://orcid.org/0000-0002-4288-5383; Elizabeth B. Klerman: https://orcid.org/0000-0002-7402-3171; Mathias Basner: https://orcid.org/0000-0002-8453-0812; Michael L. Perlis: https://orcid.org/0000-0002-6806-759X

#### References

- Harris LM, Huang X, Linthicum KP, et al. Sleep disturbances as risk factors for suicidal thoughts and behaviours: a meta-analysis of longitudinal studies. *Sci Rep.* 2020;10(1):13888.
- Liu RT, Steele SJ, Hamilton JL, et al. Sleep and suicide: a systematic review and meta-analysis of longitudinal studies. *Clin Psychol Rev.* 2020;81:101895.
- Tubbs AS, Fernandez FX, Grandner MA, et al. The mind after midnight: nocturnal wakefulness, behavioral dysregulation, and psychopathology. *Front Netw Physiol*. 2022;1:830338.
- Perlis ML, Grandner MA, Chakravorty S, et al. Suicide and sleep: is it a bad thing to be awake when reason sleeps? *Sleep Med Rev.* 2016;29:101–107.
- Mansfield DR, Wasgewatta S, Reynolds A, et al. Nocturnal wakefulness and suicide risk in the Australian population. J Clin Psychiatry. 2022;83(4):21m14275.
- McCarthy MS, Hoffmire C, Brenner LA, et al. Sleep and timing of death by suicide among U.S. Veterans 2006–2015: analysis of the American Time Use Survey and the National Violent Death Reporting System. *Sleep*. 2019;42(8):zsz094.
- Ballard ED, Vande Voort JL, Bernert RA, et al. Nocturnal wakefulness is associated with next-day suicidal ideation in major depressive disorder and bipolar disorder. J Clin Psychiatry. 2016;77(6):825–831.
- Tubbs AS, Fernandez FX, Johnson DA, et al. Nocturnal and morning wakefulness are differentially associated with suicidal ideation in a nationally representative sample. J Clin Psychiatry. 2021;82(6):20m13820.
- Tubbs AS, Fernandez FX, Perlis ML, et al. Suicidal ideation is associated with nighttime wakefulness in a community sample. *Sleep.* 2021;44(1):zsaa128.
- Emens JS, Berman AM, Thosar SS, et al. Circadian rhythm in negative affect: implications for mood disorders. *Psychiatry Res.* 2020;293:113337.
- Dzogang F, Lightman S, Cristianini N. Circadian mood variations in Twitter content. Brain Neurosci Adv. 2017;1:2398212817744501.
- Golder SA, Macy MW. Diurnal and seasonal mood vary with work, sleep, and daylength across diverse cultures. *Science*. 2011;333(6051):1878–1881.
- Wirz-Justice A. Diurnal variation of depressive symptoms. *Dialogues Clin Neurosci.* 2008;10(3):337–343.
- Hasler BP, Soehner AM, Wallace ML, et al. Experimentally imposed circadian misalignment alters the neural response to monetary rewards and response inhibition in healthy adolescents. *Psychol Med.* 2021;52(16):1–9.
- Hasler BP, Forbes EE, Franzen PL. Time-of-day differences and short-term stability of the neural response to monetary reward: a Pilot Study. *Psychiatry Res.* 2014;224(1):22–27.
- Venkatraman V, Huettel SA, Chuah LYM, et al. Sleep deprivation biases the neural mechanisms underlying economic preferences. *J Neurosci.* 2011;31(10): 3712–3718.
- Venkatraman V, Chuah YML, Huettel SA, et al. Sleep deprivation elevates expectation of gains and attenuates response to losses following risky decisions. *Sleep.* 2007;30(5):603–609.
- Muto V, Jaspar M, Meyer C, et al. Local modulation of human brain responses by circadian rhythmicity and sleep debt. *Science*. 2016;353(6300): 687–690.
- Ly JOM, Gaggioni G, Chellappa SL, et al. Circadian regulation of human cortical excitability. *Nat Commun.* 2016;7:11828.
- Verweij IM, Romeijn N, Smit DJ, et al. Sleep deprivation leads to a loss of functional connectivity in frontal brain regions. *BMC Neurosci.* 2014;15:88.
- Niendam TA, Laird AR, Ray KL, et al. Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. *Cogn Affect Behav Neurosci*. 2012;12(2):241–268.
- Killgore WDS. Effects of sleep deprivation on cognition. *Prog Brain Res.* 2010; 185:105–129.
- Goel N, Rao H, Durmer JS, et al. Neurocognitive consequences of sleep deprivation. Semin Neurol. 2009;29(4):320–339.
- Schmidt C, Collette F, Cajochen C, et al. A time to think: circadian rhythms in human cognition. *Cogn Neuropsychol.* 2007;24(7):755–789.
- Keilp JG, Gorlyn M, Russell M, et al. Neuropsychological function and suicidal behavior: attention control, memory and executive dysfunction in suicide attempt. *Psychol Med.* 2013;43(3):539–551.
- Szanto K, Galfalvy H, Vanyukov PM, et al. Pathways to late-life suicidal behavior: cluster analysis and predictive validation of suicidal behavior in a sample of older adults with major depression. J Clin Psychiatry. 2018;79(2):17m11611.
- Clark L, Dombrovski AY, Siegle GJ, et al. Impairment in risk-sensitive decisionmaking in older suicide attempters with depression. *Psychol Aging*. 2011;26(2): 321–330.
- Gibbs LM, Dombrovski AY, Morse J, et al. When the solution is part of the problem: problem solving in elderly suicide attempters. *Int J Geriatr Psychiatry*. 2009;24(12):1396–1404.

- Voros V, Tenyi T, Nagy A, et al. Crisis concept re-loaded?—The recently described suicide-specific syndromes may help to better understand suicidal behavior and assess imminent suicide risk more effectively. *Front Psychiatry*. 2021;12: 598923.
- Schuck A, Calati R, Barzilay S, et al. Suicide Crisis Syndrome: a review of supporting evidence for a new suicide-specific diagnosis. *Behav Sci Law.* 2019; 37(3):223–239.
- Meijers J, Harte JM, Meynen G, et al. Differences in executive functioning between violent and non-violent offenders. *Psychol Med*. 2017;47(10):1784–1793.
  Meijers J, Harte JM, Jonker FA, et al. Prison brain? Executive dysfunction in
- prisoners. Front Psychol. 2015;6:43.
- 33. Gibbens TCN. Sane and insane homicide. *J Crim Law Criminology Police Sci.* 1958;49(2):110–115.
- Messner SF, Tardiff K. The social ecology of urban homicide: an application of the "routine activities" approach. *Criminology*. 1985;23(2):241–267.
- Sisti D, Rocchi MBL, Maccio A, et al. The epidemiology of homicide in Italy by season, day of the week and time of day. *Med Sci Law.* 2012;52(2): 100–106.
- Kelsay JD, Tillyer MS, Tillyer R, et al. The violent victimization of children, adolescents, adults, and the elderly: situational characteristics and victim injury. *Violence Vict.* 2017;32(2):342–361.
- Teffer K, Semendeferi K. Human prefrontal cortex: evolution, development, and pathology. *Prog Brain Res.* 2012;195:191–218.
- Hisler GC, Rothenberger SD, Clark DB, et al. Is there a 24-hour rhythm in alcohol craving and does it vary by sleep/circadian timing?. *Chronobiol Int.* 2021;38(1): 109–121.
- Logan RW, Hasler BP, Forbes EE, et al. Impact of sleep and circadian rhythms on addiction vulnerability in adolescents. *Biol Psychiatry*. 2018;83(12):987–996.
- Day AM, Kahler CW, Ahern DC, et al. Executive functioning in alcohol use studies: a brief review of findings and challenges in assessment. *Curr Drug Abuse Rev.* 2015;8(1):26–40.
- Chakravorty S, Smith RV, Perlis ML, et al. Circadian pattern of deaths due to suicide in intoxicated alcohol-dependent individuals. *J Clin Psychiatry*. 2018;79(6): 17m11800.
- Van Orden KA, Witte TK, Cukrowicz KC, et al. The interpersonal theory of suicide. Psychol Rev. 2010;117(2):575–600.
- Tubbs AS, Perlis ML, Basner M, et al. Relationship of nocturnal wakefulness to suicide risk across months and methods of suicide. *J Clin Psychiatry*. 2020;81(2): 19m12964.
- Perlis ML, Grandner MA, Brown GK, et al. Nocturnal wakefulness as a previously unrecognized risk factor for suicide. J Clin Psychiatry. 2016;77(6):e726–1733.
- Byrne JEM, Tremain H, Leitan ND, et al. Circadian modulation of human reward function: is there an evidentiary signal in existing neuroimaging studies?. *Neurosci Biobehav Rev.* 2019;99:251–274.
- Silva EJ, Duffy JF. Sleep inertia varies with circadian phase and sleep stage in older adults. *Behav Neurosci*. 2008;122(4):928–935.
- Cherpitel CJ, Borges GLG, Wilcox HC. Acute alcohol use and suicidal behavior: a review of the literature. *Alcohol Clin Exp Res.* 2004;28(5 suppl):18s–28s.
- Branas CC, Richmond TS, Ten Have TR, et al. Acute alcohol consumption, alcohol outlets, and gun suicide. *Subst Use Misuse*. 2011;46(13):1592–1603.
- Borges G, Loera CR. Alcohol and drug use in suicidal behaviour. *Curr Opin* Psychiatry. 2010;23(3):195–204.
- Sutker PB, Tabakoff B, Goist KC Jr, et al. Acute alcohol intoxication, mood states and alcohol metabolism in women and men. *Pharmacol Biochem Behav.* 1983; 18(suppl 1):349–354.

- Simons JS, Emery NN, Simons RM, et al. Effects of alcohol, rumination, and gender on the time course of negative affect. *Cogn Emot.* 2017;31(7):1405–1418.
- Centanni SW, Bedse G, Patel S, et al. Driving the downward spiral: alcoholinduced dysregulation of extended amygdala circuits and negative affect. *Alcohol Clin Exp Res.* 2019;43(10):2000–2013.
- Bresin K, Mekawi Y, Verona E. The effect of laboratory manipulations of negative affect on alcohol craving and use: a meta-analysis. *Psychol Addict Behav.* 2018; 32(6):617–627.
- Chen X, Wang R, Zee P, et al. Racial/Ethnic differences in sleep disturbances: the multi-ethnic study of atherosclerosis (MESA). *Sleep.* 2015;38(6):877–888.
- Hnin K, Mukherjee S, Antic NA, et al. The impact of ethnicity on the prevalence and severity of obstructive sleep apnea. *Sleep Med Rev.* 2018;41:78–86.
- Tubbs AS, Ghani SB, Valencia D, et al. Racial/ethnic minorities have greater declines in sleep duration with higher risk of cardiometabolic disease: an analysis of the U.S. National Health Interview Survey. *Sleep Epidemiol.* 2022;2:100022.
- Brookes ST, Whitley E, Peters TJ, et al. Subgroup analyses in randomised controlled trials: quantifying the risks of false-positives and false-negatives. *Health Technol Assess*. 2001;5(33):1–56.
- Oomen PP, van Hell HH, Bossong MG. The acute effects of cannabis on human executive function. *Behav Pharmacol.* 2018;29(7):605–616.
- Crean RD, Crane NA, Mason BJ. An evidence based review of acute and longterm effects of cannabis use on executive cognitive functions. *J Addict Med.* 2011; 5(1):1–8.
- Dopierala E, Rybakowski J. Sleep deprivation as a method of chronotherapy in the treatment of depression. *Psychiatr Pol.* 2015;49(3):423–433.
- Dallaspezia S, Benedetti F. Sleep deprivation therapy for depression. *Curr Top Behav Neurosci.* 2015;25:483–502.
- Freeman D, Sheaves B, Waite F, et al. Sleep disturbance and psychiatric disorders. *Lancet Psychiatry*. 2020;7(7):628–637.
- 63. Melo MCA, Abreu RLC, Linhares Neto VB, et al. Chronotype and circadian rhythm in bipolar disorder: a systematic review. *Sleep Med Rev.* 2017;34:46–58.
- Lewis KS, Gordon-Smith K, Forty L, et al. Sleep loss as a trigger of mood episodes in bipolar disorder: individual differences based on diagnostic subtype and gender. *Br J Psychiatry*. 2017;211(3):169–174.
- McLay RN, Klam WP, Volkert SL. Insomnia is the most commonly reported symptom and predicts other symptoms of post-traumatic stress disorder in U.S. service members returning from military deployments. *Mil Med.* 2010;175(10): 759–762.
- Germain A. Sleep disturbances as the hallmark of PTSD: where are we now? Am J Psychiatry. 2013;170(4):372–382.
- Betts KS, Williams GM, Najman JM, et al. The role of sleep disturbance in the relationship between post-traumatic stress disorder and suicidal ideation. J Anxiety Disord. 2013;27(7):735–741.
- Palagini L, Cipollone G, Masci I, et al. Insomnia symptoms predict emotional dysregulation, impulsivity and suicidality in depressive bipolar II patients with mixed features. *Compr Psychiatry*. 2019;89:46–51.
- Benard V, Etain B, Vaiva G, et al. Sleep and circadian rhythms as possible trait markers of suicide attempt in bipolar disorders: an Actigraphy Study. J Affect Disord. 2019;244:1–8.
- Chu C, Buchman-Schmitt JM, Stanley IH, et al. The interpersonal theory of suicide: a systematic review and meta-analysis of a decade of cross-national research. *Psychol Bull.* 2017;143(12):1313–1345.
- Tubbs AS, Fernandez FX, Ghani SB, et al. Prescription medications for insomnia are associated with suicidal thoughts and behaviors in two nationally representative samples. *J Clin Sleep Med*. 2021;17(5):1025–1030.