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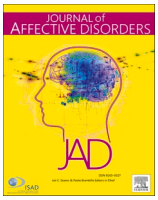

The sleep-anxiety dysregulation model of alcohol use disorder risk: A nine-year longitudinal machine learning study

Nur Hani Zainal [a,*], Natalia Van Doren [b]

[a] National University of Singapore, Department of Psychology, Kent Ridge Campus, Singapore
[b] University of California at San Francisco, Department of Psychiatry and Behavioral Sciences, San Francisco, CA, USA

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ABSTRACT

Background: Sleep disturbances are a known risk factor for alcohol use, yet their long-term predictive value for alcohol use disorder (AUD)–especially in the context of co-occurring anxiety symptoms–remains understudied. The present study thus applied machine learning with internal validation to evaluate how sleep disturbances predict nine-year AUD symptoms in midlife adults. It also introduces the *Sleep-Anxiety Dysregulation Model of AUD Risk*, which posits that sleep and anxiety symptoms confer shared vulnerability via disrupted arousal regulation.

Method: Community-dwelling midlife adults ($N = 1{,}054$) completed clinical interviews, self-reports, and a seven-day actigraphy protocol to assess demographics, psychiatric symptoms, anxiety severity, subjective sleep, and objective actigraphy sleep indices. A five-fold nested cross-validated random forest identified potentially nonlinear and interactive predictors. The baseline model included 41 variables.

Results: The final multivariable model explained over two-fifths of the variance in nine-year AUD symptoms ($R^2 = 42.7\%$, 95% confidence intervals [40.1%–45.8%]). Key baseline predictors of nine-year AUD severity included lower rest-stage activity, sleep discontinuity and fragmentation patterns, and decreased active wake-stage physical movement. Other baseline predictors comprised younger age, higher generalized anxiety disorder, major depression, and panic disorder severity. No subjective sleep disturbances predicted nine-year AUD symptoms.

Conclusions: Results underscore the shared contribution of sleep and anxiety disturbances to long-term AUD risk. The proposed *Sleep-Anxiety Dysregulation Model of AUD Risk* offers an integrative framework suggesting that AUD symptoms may emerge via chronic arousal dysregulation, including heightened physiological reactivity. Externally validating this model may inform preventive strategies targeting distal risk processes underlying AUD.

1. Introduction

Alcohol use disorder (AUD) involves excessive drinking that impairs academic, social, and occupational functioning (American Psychiatric Association, 2013). Approximately 12.7% of the U.S. population met criteria for 12-month AUD, and nearly 80 % had consumed alcohol in the past decade (Grant et al., 2017). AUD is linked to mental health comorbidities (Castillo-Carniglia et al., 2019), including anxiety disorders (Smith & Randall, 2012) and physical conditions (AshaRani et al., 2022), contributing to reduced quality of life and earlier mortality (Lu et al., 2022). Given its chronicity and burden, identifying early psychological and physiological vulnerabilities–'*distal risk factors*'–is vital for prevention and treatment.

AUD is frequently viewed through the lens of proximal triggers–such as craving, stress, or relapse cues–but a growing body of evidence suggests that long-term risk may originate in more chronic, under-recognized patterns of physiological and psychological dysregulation (Cox and Olatunji, 2016; Dvorak et al., 2014; Gondre-Lewis et al., 2016). In particular, persistent sleep disturbances and anxiety symptoms–two commonly co-occurring yet under-treated conditions–may contribute to neuroadaptive vulnerability that heightens long-term risk for AUD through shared disruption of arousal systems (Koob and Colrain, 2020). However, few studies have examined how co-occurring sleep and anxiety symptoms predict long-term AUD outcomes, particularly among

* Corresponding author at: National University of Singapore (NUS), Department of Psychology, Faculty of Arts and Social Sciences, Block AS4, Office #03-25, 9 Arts Link, 117570, Singapore.
E-mail address: hanizainal@nus.edu.sg (N.H. Zainal).

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midlife and older adults, a population often overlooked in AUD prevention research (Patrick et al., 2023). Few have incorporated objective (actigraphy) and subjective sleep measures or used machine learning (ML) approaches to detect nonlinear, interacting risk factors across extended timeframes. Thus, the present study addresses these gaps by testing the *Sleep-Anxiety Dysregulation Model of AUD Risk* in a nine-year prospective sample, with implications for identifying modifiable at-risk individuals before full-syndrome heightened AUD severity emerges.

Sleep disturbances may serve as key distal risk factors for increased AUD through biopsychosocial mechanisms. They frequently co-occur with anxiety symptoms (Cox and Olatunji, 2016), which share neurobiological pathways with sleep regulation (Harvey et al., 2011) and elevate the risk of maladaptive alcohol use (Brockdorf et al., 2022). Yet, few longitudinal models have explored their combined impact on long-term increased AUD risk in midlife. Neurologically, chronic sleep disruption may impair gamma-aminobutyric acid (GABA) and glutamate systems involved in reward, arousal, and anxiety regulation, increasing alcohol cravings, use, and tolerance for sleep regulation (Koob and Colrain, 2020; Lindberg et al., 2018). Cognitively, sleep fragmentation–often intensified by anxiety-driven hyperarousal–can heighten emotional reactivity, impair executive function, and fuel risk-taking, reinforcing a cycle of alcohol use to self-medicate poor sleep and distress, further degrading sleep quality (Panin and Peana, 2019). Although alcohol may offer short-term sedative effects (Gardiner et al., 2025; Gilman et al., 2008), withdrawal can induce hyperarousal, worsening anxiety, and sleep (Hartwell et al., 2015). These dynamics may be exacerbated by academic or occupational stress, which, when coupled with anxiety and sleep issues, dysregulate the HPA axis and perpetuate alcohol misuse (Forrester et al., 2019). Genetic predispositions may further heighten sensitivity to sleep-anxiety dysregulation, amplifying long-term increased AUD risk (Chakravorty et al., 2023).

Most empirical data assessing the links between sleep disturbances and AUD symptoms have been cross-sectional (He et al., 2019; Hussain et al., 2022), hindering directional inferences (Blackwell and Glynn, 2018). Additionally, existing longitudinal studies on this topic have mainly focused on college students and youth populations. For instance, daytime dysfunction, issues falling asleep, and later weekday and weekend bedtimes in childhood (Sabatier et al., 2025) and early adolescence (Hasler et al., 2022; Troxel et al., 2021) preceded more alcohol misuse in late adolescence or young adulthood. Similarly, shorter time dozing before rising preceded more alcohol consumption through executive functioning impairments in school-going youths (Warren et al., 2017). Moreover, poorer sleep quality among those with vs. without AUD persisted across five years in teenagers (Hasler et al., 2014). Similarly, in young community adult samples, alcohol misuse might acutely enhance subjective sleep efficiency across days (Miller et al., 2021), but experiencing insomnia and hypersomnia increased the odds of AUD three years later by 1.72 to 3.92 times (Breslau et al., 1996). Relatedly, higher insomnia symptoms worsened alcohol misuse across several months among U.S. Veterans (Davis et al., 2022) but not across days among nurses (Thompson et al., 2024). However, these studies rarely account for co-occurring psychiatric symptoms, particularly anxiety, which are strongly linked to both sleep disruption and AUD. This limits our understanding of how these symptoms may jointly contribute to increased long-term risk of AUD. Although progress in this area has been made, there has been a dearth of studies on how sleep disturbances predict long-term AUD symptoms across years in midlife and older community adult populations. Further, few studies have employed multivariable approaches capable of capturing the complex, interacting contributions of joint risk factors (such as anxiety and sleep) over extended periods.

Efforts to identify which unique sleep disturbances function as distal AUD symptom risk factors should integrate subjective and objective measures of sleep disturbances. However, most existing literature has relied on self-report measures of sleep disturbances (He et al., 2019), such as daily diaries (e.g., Miller et al., 2021) and the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Subjective self-reports indicate individuals' viewpoints of sleep quality and are influenced by cognitive and emotional factors, including anxiety, which has been shown to impact alcohol use experiences, including relapse risk, while in recovery (Dzierzewski et al., 2022; Smith et al., 2014). Sleep quality indices might include sleep efficiency (proportion of total sleep time [TST] to total time in bed), sleep onset latency (SOL; time taken to fall asleep), and wake after sleep onset (WASO; total wakefulness period after sleep onset but before final awakening; Shrivastava et al., 2014). Actigraphy wearables capture the same sleep quality indices more objectively, and actigraphy data have been shown to diverge from self-reports in the context of AUD research (Brooks et al., 2012). Sleep disturbance markers, such as TST, may be overestimated in self-reports, with actigraphy indices instead indicating extended wakefulness in individuals with or at risk for future AUD symptoms (Brooks et al., 2020; Geoghegan et al., 2012). Dual measurements of sleep disturbances, using both actigraphy and self-reports, are thus necessary to identify the relative contributions of cognitively driven sleep appraisals and actigraphy-captured physiological adaptations of sleep (Brooks et al., 2021). Taken together, objective actigraphy and subjective self-report markers of sleep disturbances might precede more long-term AUD symptoms across years in midlife and older adult samples.

However, linear model assumptions using traditional ordinary least squares (OLS) hinder the advancement of studying sleep disturbances and anxiety as distal risk factors of AUD symptoms. Traditional OLS regression often fails to account for multicollinearity among unique yet interrelated sleep predictors, and it also struggles to detect potential nonlinearities (Farahani et al., 2010) or complex higher-order interactions (Sheetal et al., 2023). These considerations are integral since the relationship between distinct sleep disturbance indicators and alcohol misuse, intoxication, cravings, tolerance, and related symptoms could be nonlinear (Zheng et al., 2024) and dependent on one another (Tracy et al., 2021). Data-driven ML methods could overcome these challenges, especially when relevant theories inform the predictor set (Elhai and Montag, 2020; Yarkoni and Westfall, 2017). Ensemble ML approaches, such as random forest (RF), optimize the bias-variance trade-off, detect nonlinear relations, identify higher-order interactions, and examine if such patterns generalize to unseen data (Fife and D'Onofrio, 2023). These goals could be achieved by using methods that separate model training and testing datasets, such as nested cross-validation (NCV), which prevents data leakage and overfitting (issues in generalizing patterns to unseen data; de Rooij and Weeda, 2020; Song et al., 2021). Further, ML approaches fall within the class of precision psychiatry techniques (Williams et al., 2024), where calls have been made to investigate their promises and pitfalls in AUD research (Ebrahimi et al., 2021). To this end, harnessing ML approaches would be a step toward developing an actionable clinical prognostic calculator to inform optimal prevention programs and tertiary treatments for those with or at risk for long-term AUD symptoms.

Addressing gaps in the literature, this study used ML to identify which sleep disturbances and anxiety symptoms predict AUD symptoms nine years later in midlife and older adults. First, we hypothesized that our multivariable model, which included actigraphy, sleep self-reports, and anxiety severity, would demonstrate good predictive performance, defined as an *R*-squared (R^2) value of $\geq$10% (Gao, 2023). Second, we expected both sleep disturbance and anxiety variables to consistently emerge as linear and nonlinear predictors of higher nine-year AUD severity. As detailed below, we adjusted for clinical, demographic, and related covariates to minimize concerns about data mining and *p*-hacking (Wegener et al., 2024).

2. Method

2.1. Study design

Data from the present study were derived from the publicly available Midlife Development in the United States (MIDUS) database (Ryff et al., 2019a; Ryff et al., 2021; Ryff et al., 2019b). Participants were followed twice over nine years. Sleep disturbance, AUD, comorbid psychiatric symptoms, and sociodemographic data were collected from 2004 to 2006 (Wave 1; W1). Data on AUD severity were collected again from 2013 to 2014 (Wave 2; W2). Among the 1,054 participants who participated in W1 of the data collection protocol that provided adequate relevant data for the present study, 167 (15.8%) dropped out by not completing the W2 AUD severity measure. Dropouts were significantly older than completers ($M = 59.5$, $SD = 14.9$ vs. $M = 54.7$, $SD = 11.0$; t(degrees of freedom [df] = 192.7) = −3.89, $p < .001$) and comprised a higher percentage of individuals who racially identified as non-White (26.9% vs. 14.8%; $\chi^2(df = 1) = 8.43$, $p = .004$). However, dropouts and completers were not statistically different in terms of self-reported sex (males: 17.4%; females: 14.6%; $\chi^2(df = 1) = 1.38$, $p = .241$) and education level (formal college education: 15.0%; up to high school education: 17.3%; $\chi^2(df = 3) = 0.94$, $p = .817$).

2.2. Participant attributes

Community-dwelling adults ($N = 1{,}054$) offered voluntary informed consent to participate in the MIDUS study (Ryff et al., 2019a; Ryff et al., 2021; Ryff et al., 2019b). Ethical approval for the MIDUS study was obtained from participating universities, and no additional approval was required for this secondary data analysis. Participants were primarily middle-aged adults ($M = 55.32$, $SD = 11.78$, range = 34–84), with a slight majority being women (577 [54.74%]) compared to men (477 [45.26%]). Educational attainment varied: 214 (20.31%) held a university or postgraduate degree, whereas 840 (79.69%) had a high school education or less, or declined to report. Racially, most participants were White (988 [93.73%]); the remainder identified as Black, Asian, Native American, Pacific Islander, or Multiracial (66 [6.27%]).

2.3. Procedures

2.3.1. Protocol across W1 and W2

Participants completed surveys assessing AUD symptoms using the Michigan Alcohol Screening Test (MAST) at W1 and W2 (Selzer, 1971), sleep quality with the PSQI at W1 (Buysse et al., 1989), and SUD symptoms at W1 using a self-report developed by MIDUS (Turiano et al., 2012). They also underwent clinical interviews assessing potential confounders–generalized anxiety disorder (GAD), major depressive disorder (MDD), and panic disorder (PD) symptoms–using the Composite International Diagnostic Interview-Short Form (CIDI-SF; Kessler et al., 1998), aligned with Diagnostic and Statistical Manual-Third Edition-Revised (DSM-III-R; Kessler et al., 2006). Additionally, participants attended a two-day MIDUS-organized site visit to complete biomarker assessments, including instructions for the actigraphy protocol (Love et al., 2010). The MIDUS project organized data collection across several universities by carrying out standardized protocols to facilitate alignment in actigraphy and self-report sleep measures at each site. All data were primarily overseen, managed, and integrated by the chief MIDUS coordinating center, which is situated at the University of Michigan's Inter-University Consortium at the Institute for Political and Social Research (ICPSR). ICPSR and the MIDUS center support harmonization and cross-site evaluations while preserving stringent quality checks and controls across the research network (Radler and Love, 2018).

2.3.2. Objective sleep actigraphy protocol

To standardize actigraphy procedures, MIDUS researchers instructed participants to begin wearing the Actiwatch (Mini-Mitter, Philips) on the Tuesday after their site visit (Križan and Hisler, 2019; Uysal et al., 2019). Participants wore the device continuously for seven days, through Tuesday morning. The actigraphy passively recorded sleep efficiency, SOL, WASO, TST, activity counts, movement intensity, wake time, and other sleep-wake markers during wake, rest, and sleep stages (Hisler and Krizan, 2017; Teas and Friedman, 2021). Sleep and wake bouts were also tracked across these stages. Participants used daily diaries to mark the start and end of rest periods (Bhat et al., 2024); missing entries were supplemented using adjacent time points, following best-practice guidelines for actigraphy data (Owens et al., 2017).

2.4. Measures

2.4.1. W1 and W2 AUD symptoms

AUD severity was assessed using an adapted version of the MAST self-report, which asked participants how often they experienced specific AUD symptoms in the past year (Selzer, 1971). Symptoms included: (i) mental health issues (e.g., depression, paranoia, bizarre thoughts); (ii) intense cravings or urges; (iii) prolonged periods of excessive drinking or recovery; (iv) increased alcohol tolerance; and (v) risk of serious harm. Each symptom was coded as *present* (1) or *absent* (0), yielding scores from 0 to 5. This scale demonstrated acceptable internal consistency (Cronbach's α = .70 at W1, .77 at W2) and strong construct validity (Zainal et al., 2024).

2.4.2. W1 GAD symptoms

Participants completed the CIDI-SF interview assessing GAD symptoms over the past year, rating how often they experienced worries more frequently than most people, ranging from *never* (1) to *most days* (4). Symptoms included: (i) restlessness; (ii) nervousness; (iii) irritability; (iv) trouble falling or (v) staying asleep; (vi) concentration difficulties; (vii) memory lapses; (viii) fatigue; (ix) low stamina; and (x) muscle tension or soreness (score range = 4–40). The scale demonstrated excellent internal consistency (α = .98), reliability, and construct validity (Gigantesco and Morosini, 2008; Ng et al., 2024).

2.4.3. W1 MDD symptoms

Participants reported the *presence* (1) or *absence* (0) of past-year MDD symptoms related to depressed mood or anhedonia using the CIDI-SF interview (Gigantesco and Morosini, 2008). Symptoms included: (i) loss of interest; (ii) fatigue; (iii) appetite changes; (iv) sleep disturbances; (v) concentration issues; (vi) feelings of worthlessness; and (vii) recurrent thoughts of death. Scores ranged from 0 to 14. This measure demonstrated high internal consistency (α = .93) and strong construct validity (Zainal and Newman, 2021; Zainal and Newman, 2022).

2.4.4. W1 PD symptoms

Participants completed the CIDI-SF, which assessed past-year PD symptoms, indicating the *presence* (1) or *absence* (0) of symptoms experienced during unexpected panic attacks in safe situations (Wang et al., 2000). Symptoms included: (i) rapid heart rate; (ii) chest discomfort; (iii) sweating; (iv) trembling; (v) chills or hot flashes; and (vi) derealization. Scores ranged from 0 to 6. The scale demonstrated good internal consistency (α = .86) and strong construct validity (Bakhshaie et al., 2016).

2.4.5. W1 SUD symptoms

Participants completed survey items assessing past-year SUD symptoms, indicating *presence* (1) or *absence* (0) of nonmedical use of substances such as sedatives, stimulants, painkillers, antidepressants, inhalants, marijuana, cocaine, hallucinogens, or heroin (Zvolensky et al., 2015). One item captured any use, whereas others assessed unplanned use, functional impairment, risk of harm, mental health impact, and tolerance. Scores ranged from 0 to 18. The scale showed high internal consistency (α = .81) and strong construct validity (Bakhshaie

et al., 2015; Zainal et al., 2024).

2.4.6. W1 subjective sleep quality

Participants completed the PSQI, which assessed seven dimensions of subjective sleep quality: (i) daytime dysfunction; (ii) habitual sleep inefficiency; (iii) sleep disturbances; (iv) TST; (v) SOL; (vi) sleep medication use; and (vii) overall sleep quality (Buysse et al., 1989; Carpenter and Andrykowski, 1998). All items were scored 0–3, except habitual sleep inefficiency (0–4). The PSQI demonstrated acceptable internal consistency (α = .70) and good construct validity (Hinz et al., 2017; Liu et al., 2021).

2.4.7. W1 childhood trauma

Since prior trauma could serve as a potential confounder, we statistically adjusted for this variable in all multivariable models. The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994) was used to assess childhood maltreatment encounters across six dimensions: emotional abuse, physical abuse, sexual abuse, emotional neglect, physical neglect, as well as minimization and denial. Participants responded to each item on a 5-point Likert scale (1 = *never true* to 5 = *very often true*), generating scores that could range from 28 to 140. The CTQ has shown high internal consistency (α = .79–.94) and strong retest-reliability (Bernstein et al., 1994). CTQ scores have also demonstrated good convergent validity and strong discriminant validity (Bernstein and Fink, 1998; Hoeboer et al., 2025).

2.5. Data analysis

All data management and analyses were conducted using *R* (R Core Team, 2025). RF nonparametric imputation was employed to address missing data, given its ability to capture complex interactions and nonlinearities that exceed the capabilities of standard parametric methods (Golino and Gomes, 2016; Shah et al., 2014). Continuous variables were standardized ($M = 0$, $SD = 1$), and nominal variables were one-hot encoded (James et al., 2013). Feature engineering was performed separately within training and testing folds under a five-fold nested cross-validation (5F-NCV) framework to prevent data leakage and overfitting (Qiu, 2024). Inner loops handled feature engineering, tuning, and training; outer loops conducted model testing. The W1 predictor set included 42 variables, such as actigraphy scores averaged across seven days, with full descriptives in Table 1. Covariates were selected based on prior research: age (Daskalopoulou et al., 2018), sex (Freeman et al., 2022), education (Assari and Lankarani, 2016), GAD, MDD, and PD symptoms (Kushner et al., 2000; Rudenstine et al., 2020), as well as childhood trauma (Shin et al., 2019).

Seven multivariable ML models were evaluated using the *nestedcv* package, with RF emerging as the best performer (Lewis et al., 2023). RF integrates decision trees with stopping rules to reduce overfitting

Table 1
Descriptive statistics of variables in the W1 variables to predict W2 AUD severity.

	M/n	(*SD*)/(%)	Minimum	Maximum	Skewness	Kurtosis
W1 age (years)	55.32	(11.78)	34	84	0.30	−0.67
W1 men vs. women	477	(45.26)	–	–	–	–
W1 college-educated	214	(20.31)	–	–	–	–
White	988	(93.73)	–	–	–	–
W1 MDD severity	0.85	(2.34)	0	14	2.82	7.36
W1 GAD severity	11.69	(6.85)	8	32	1.48	0.53
W1 PD severity	0.43	(1.18)	0	6	2.90	7.84
W1 AUD severity	0.07	(0.37)	0	4	7.03	56.43
W1 SUD severity	0.61	(1.89)	0	18	4.11	21.71
W1 CTQ severity	64.24	(6.72)	44	98	0.68	2.75
W1 PSQI daytime dysfunction	0.81	(0.67)	0.00	3.00	0.49	0.21
W1 PSQI habitual sleep inefficiency	0.72	(1.16)	0.00	4.00	1.47	0.90
W1 PSQI sleep disturbances	1.28	(0.56)	0.00	3.00	0.61	0.47
W1 PSQI total sleep time (TST)	0.78	(0.75)	0.00	3.00	0.82	0.51
W1 PSQI sleep onset latency (SOL)	0.88	(0.92)	0.00	3.00	0.86	−0.12
W1 PSQI sleep medication use	0.57	(1.07)	0.00	3.00	1.57	0.79
W1 PSQI subjective poor sleep quality	0.97	(0.68)	0.00	3.00	0.46	0.52
W1 mean activity counts (resting phase)	31.44	(18.88)	6.90	120.42	1.82	4.02
W1 maximum activity counts (resting phase)	683.04	(207.62)	201.43	1420.33	0.83	0.82
W1 wake time (resting phase)	69.01	(34.19)	22.33	234.07	1.38	2.38
W1% of wake time (resting phase)	14.89	(7.38)	5.50	41.96	1.51	2.22
W1 average wake bouts (resting phase)	38.15	(13.52)	10.71	95.71	1.13	2.59
W1 average sleep bouts (resting phase)	12.50	(6.06)	3.29	81.16	4.32	35.83
W1 total activity counts (sleep phase)	7713.94	(4604.40)	1002.33	28,696.57	1.76	4.20
W1 mean activity counts (sleep phase)	18.66	(12.03)	2.59	74.10	1.92	4.39
W1 maximum activity counts (sleep phase)	510.46	(176.68)	185.43	1297.33	0.91	1.30
W1 average sleep onset latency (sleep phase)	25.77	(23.08)	0.21	128.57	1.87	3.58
W1 total sleep time (sleep phase)	13.46	(14.94)	0.50	84.92	2.28	5.61
W1 sleep efficiency (sleep phase)	81.61	(9.57)	44.27	93.61	−1.52	2.50
W1 wake after sleep onset (sleep phase)	45.57	(21.87)	8.83	139.86	1.33	2.51
W1 wake time (sleep phase)	45.65	(22.13)	8.83	139.86	1.37	2.74
W1% of wake time (sleep phase)	10.79	(5.69)	2.28	31.08	1.49	2.06
W1 average wake bouts (sleep phase)	32.06	(10.59)	10.14	71.33	0.59	0.13
W1 average sleep bouts (sleep phase)	14.25	(7.43)	4.26	81.44	4.37	32.35
W1 total activity counts (active phase)	328,289.45	(105,549.46)	62,573.67	620,944.17	0.36	−0.01
W1 mean activity counts (active phase)	335.93	(108.24)	65.49	660.60	0.42	0.29
W1 maximum activity counts (active phase)	1376.26	(364.51)	429.83	2406.83	0.38	−0.10
W1 wake time (active phase)	818.20	(105.14)	504.92	1085.75	−0.49	0.07
W1% of wake time (active phase)	83.23	(9.21)	48.62	97.64	−1.02	0.94
W1 average wake bouts (active phase)	62.58	(30.14)	7.33	158.17	0.58	−0.14
W1 average sleep bouts (active phase)	2.73	(1.46)	1.22	19.29	7.31	77.14
W2 AUD severity	0.13	(0.55)	0.00	5.00	5.53	35.03

Note. W1, wave 1 (2004–2006); W2, wave 2 (2013–2014); AUD, alcohol use disorder; MDD, major depressive disorder; GAD, generalized anxiety disorder; PD, panic disorder; SUD, substance use disorder; CTQ, Childhood Trauma Questionnaire; PSQI, Pittsburgh Sleep Quality Index.

(Breiman, 2001; Lechner and Okasa, 2024). The default number of trees was 500. The '*mtry*' parameter, or number of predictors randomly selected at each split, ranged from 2 to $\sqrt{}$(the total number of predictors) (Chen et al., 2023). Two splitting rules were tested: '*variance*' (default) and '*extra trees*' (Mishra et al., 2021). The minimum terminal node size was set at 5, 10, or 15 to restrict further splits. A 5F-NCV framework ensured separation of tuning and testing to prevent overfitting (Lewis et al., 2023). Predictor importance was assessed using permutation importance, which favors variables that most reduce tree impurity (Fife and D'Onofrio, 2023).

Model performance was evaluated using R^2, root mean squared error (RMSE), and mean absolute error (MAE) point estimates (Pargent et al., 2023). To quantify uncertainty, 95% confidence intervals (CIs) were computed via 1,000 bootstrap resamples, with narrower CIs indicating greater precision (Noma et al., 2021). Hypothesis 1 was supported if R^2 exceeded 10% (Gao, 2023). Model calibration assessed the alignment between predicted and actual outcomes (Lindhiem et al., 2018). Calibration plots displayed a dashed red line for perfect calibration (intercept = 0, slope = 1) and a bold blue line to show deviations. Brier scores, representing mean squared error, indicated better calibration with lower values (Fokkema et al., 2022).

To test Hypothesis 2 and visualize linear, nonlinear, and interactive effects of each multivariable predictor on W2 AUD severity, we used two explainable artificial intelligence (XAI) methods: partial dependence plots (PDPs; Molnar, 2022) and Shapley additive explanations (SHAP; Lundberg and Lee, 2017). PDPs, generated via the *pdp* package (Greenwell, 2017), show each predictor's global marginal effect while averaging over all other predictors. Because PDPs lack participant-level insights, SHAP bee swarm plots were also created to display both global and local effects (Lundberg et al., 2020). Using the *kernelshap* package (Mayer and Watson, 2024), SHAP visualized the distribution of each predictor's impact: rows represented predictors ranked by absolute SHAP value, with red (positive) and blue (negative) points indicating the direction and density of individual-level effects.

The dataset is already publicly available at the MIDUS repository (https://tinyurl.com/icpsr-midus). The authors do not have permission to re-share the data with manuscript submission but are instead instructed by the MIDUS team to cite the MIDUS repository, as we have already done in our manuscript. *R* analytic codes and scripts are also available upon reasonable request.

3. Results

Hypothesis 1. Multivariable predictive ML model performance.

Table 2 presents performance metrics for all seven ML algorithms. The RF model performed best, yielding the highest R^2 (42.7%, 95% CI [40.1%–45.8%]) and lowest RMSE (0.199, 95% CI [0.174–0.226]) and MAE (0.098, 95% CI [0.088–0.109]). As shown in Fig. S1, the calibration plot indicated close alignment between predicted and actual values, aside from minor over- and underpredictions. The Brier score was low (0.010), as was the calibration intercept, which was close to 0 (−0.023), and the calibration slope, which approached 1 (1.284), suggesting generally good model calibration. Overall, results supported Hypothesis 1, which anticipated strong multivariate model performance.

Hypothesis 2. Linear and complex relations between W1 predictors and W2 AUD severity.

Fig. 1 presents PDPs for the top 20 W1 predictors of W2 AUD severity; Fig. 2 shows corresponding SHAP bee swarm plots. These predictors included 5 psychopathology variables, 1 demographic variable, and 14 actigraphy-indexed variables, with their relative importance denoted by the number (#) in parentheses. With respect to W1 psychopathology variables, higher symptom severity of AUD (#1), GAD (#2), MDD (#4), PD (#5), and SUD (#6). For the 1 demographic variable, younger age (#3) was associated with higher W2 AUD severity. For rest stage variables at W1, lower total activity counts (#7), high and low percentage of wake time (#8), longer wake time (#10), higher average movement counts (#16), as well as high and low maximum movement counts (#19), predicted greater W2 AUD severity. For sleep stage variables at W1, fewer wake bouts (#9), higher average sleep bouts (#13), greater maximum activity counts (#14), shorter TST (#15), and lower sleep efficiency (#17) predicted higher W2 AUD severity. For active wake stage variables at W1, fewer movement counts (#11), wake bouts (#12), higher sleep bouts (#18), and lower percentage of wake time (#20) predicted more W2 AUD symptoms. Overall, these findings were fully aligned with Hypothesis 2, which anticipated that linear and nonlinear patterns of W1 anxiety and sleep disturbance variables would predict higher W2 AUD severity.

Table 2
Multivariate ML model performance metrics of W1 variables predicting W2 AUD severity.

Model	RMSE (95 % CI)	MAE (95 % CI)	R^2 (95 % CI)
LASSO	0.255 (0.239–0.271)	0.121 (0.115–0.128)	0.058 (0.024–0.090)
Ridge	0.254 (0.239–0.271)	0.121 (0.114–0.128)	0.064 (0.034–0.094)
Elastic net	0.254 (0.239–0.270)	0.121 (0.114–0.128)	0.065 (0.032–0.097)
Decision trees	0.252 (0.219–0.286)	0.119 (0.105–0.133)	0.079 (0.011–0.152)
Random forest	0.199 (0.174–0.226)	0.098 (0.088–0.109)	0.427 (0.401–0.458)
GBM	0.249 (0.217–0.282)	0.120 (0.107–0.134)	0.105 (0.059–0.156)
SVM	0.202 (0.172–0.235)	0.071 (0.060–0.083)	0.409 (0.376–0.446)

Note. ML, machine learning; W1, wave 1 (2004–2006); W2, wave 2 (2013–2014); AUD, alcohol use disorder; RMSE, root mean squared error; CI, confidence interval; MAE, mean absolute error; R^2, *R*-squared; LASSO, least absolute shrinkage and selection operator; GBM, gradient boosting machine; SVM, support vector machine. All ML models were based on five-fold nested cross-validation, and the model performance metrics were derived from aggregating across all outer-loop folds.

4. Discussion

Our study tested the prognostic utility of a multivariate model predicting nine-year AUD symptoms with a high-dimensional data set that comprised sleep disturbances (objective and subjective) and anxiety severity vis-à-vis clinical and demographic confounders. Encouragingly, this multivariate model had moderate predictive power with practical significance (Rights and Cole, 2018), accounting for 42.7%, 95% CIs (40.1%–45.8%) of the proportion of variance of nine-year AUD severity. Five top psychopathology predictors comprised AUD, GAD, and PD severity, as well as MDD and SUD symptoms. Fourteen top predictors were purely actigraphy-indexed sleep disturbances, while subjective appraisals of sleep disturbances based on the PSQI did not emerge as significant predictors. Younger age also predicted higher nine-year AUD severity. We propose that biopsychosocial mechanisms partly account for these outcomes, and we refer to our theory as the '*Sleep-Anxiety Dysregulation Model of AUD Risk*' (Fig. 3) to spur further research that may bridge the gap between clinical psychological theory and practice.

Concordant with the theorized *Sleep-Anxiety Dysregulation Model of AUD Risk*, the outcomes showed that heightened W1 anxiety-linked psychopathology, namely GAD, MDD, PD, and SUD symptoms, predicted higher AUD severity nine years later. These findings underscore the idea that shared physiological arousal systems may contribute to cross-cutting risk for excessive alcohol consumption across time (He et al., 2019). The finding that AUD severity at W1 emerged as the topmost predictor of future AUD severity also aligned with homotypic continuity models of risk (Nadel and Thornberry, 2017; Speranza et al., 2023).

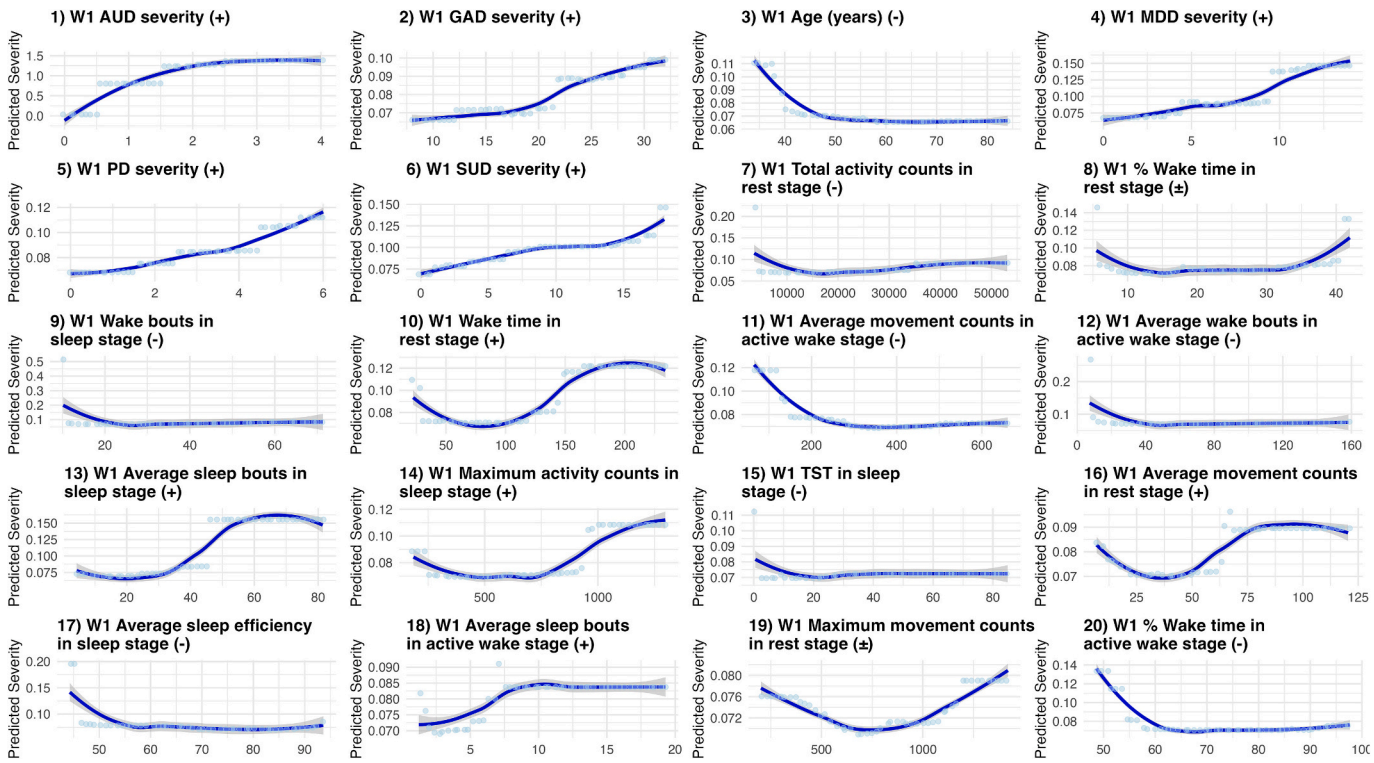

Fig. 1. Partial dependence plots of W1 sleep disturbance variables predicting W2 AUD severity
Note. W1, wave 1 (2004–2006); W2, wave 2 (2013–2014); AUD, alcohol use disorder; GAD, generalized anxiety disorder; MDD, major depressive disorder; PD, panic disorder; SUD, substance use disorder; TST, total sleep time.

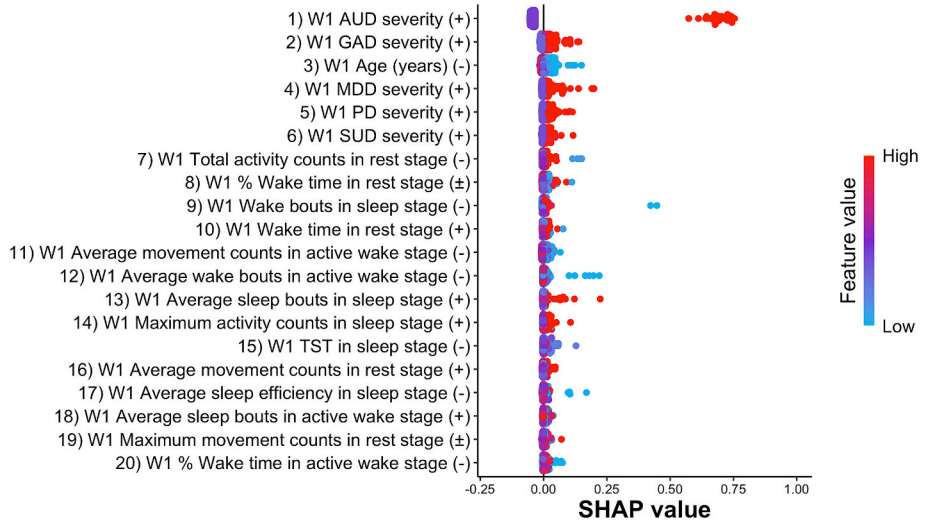

Fig. 2. SHAP bee swarm plot of W1 sleep disturbance variables predicting W2 AUD severity
Note. SHAP, Shapley additive explanations; W1, wave 1 (2004–2006); W2, wave 2 (2013–2014); AUD, alcohol use disorder; GAD, generalized anxiety disorder; MDD, major depressive disorder; PD, panic disorder; SUD, substance use disorder; TST, total sleep time.

Beyond mental disorders, 14 actigraphy-based variables in active wake, rest, and sleep stages forecasted nine-year AUD severity. These outcomes might be implicated by circadian rhythm misalignment, restorative sleep impairments, and somatic hyperarousal mediated by imbalances in the GABA or glutamate pathways (Nam et al., 2012) or an overactive HPA axis (Koob and Colrain, 2020). These complex, U-shaped relations, such as both low and high wake time percentages in rest stages predicted more AUD symptoms, highlight the expected nonlinear patterns. For example, during rest stages, minimal physical activity, high and low percentages of wake time, and longer wake duration predicted more future AUD symptoms. This pattern might have reflected anticipatory somatic arousal during planned rest intervals and autonomic imbalances, such as overactivation of the sympathetic branch compared to the parasympathetic branch (Koob and Colrain, 2020). Likewise, lower sleep efficiency and shorter TST during sleep stages predicted more nine-year AUD symptoms. Such disrupted non-restorative sleep patterns might be explained by deficits in emotion regulation and reward processing (Martindale et al., 2017; Yang et al., 2024). Postponements in dim light melatonin onset (DLMO; Burgess et al., 2022) may contribute to evening chronotype patterns captured by

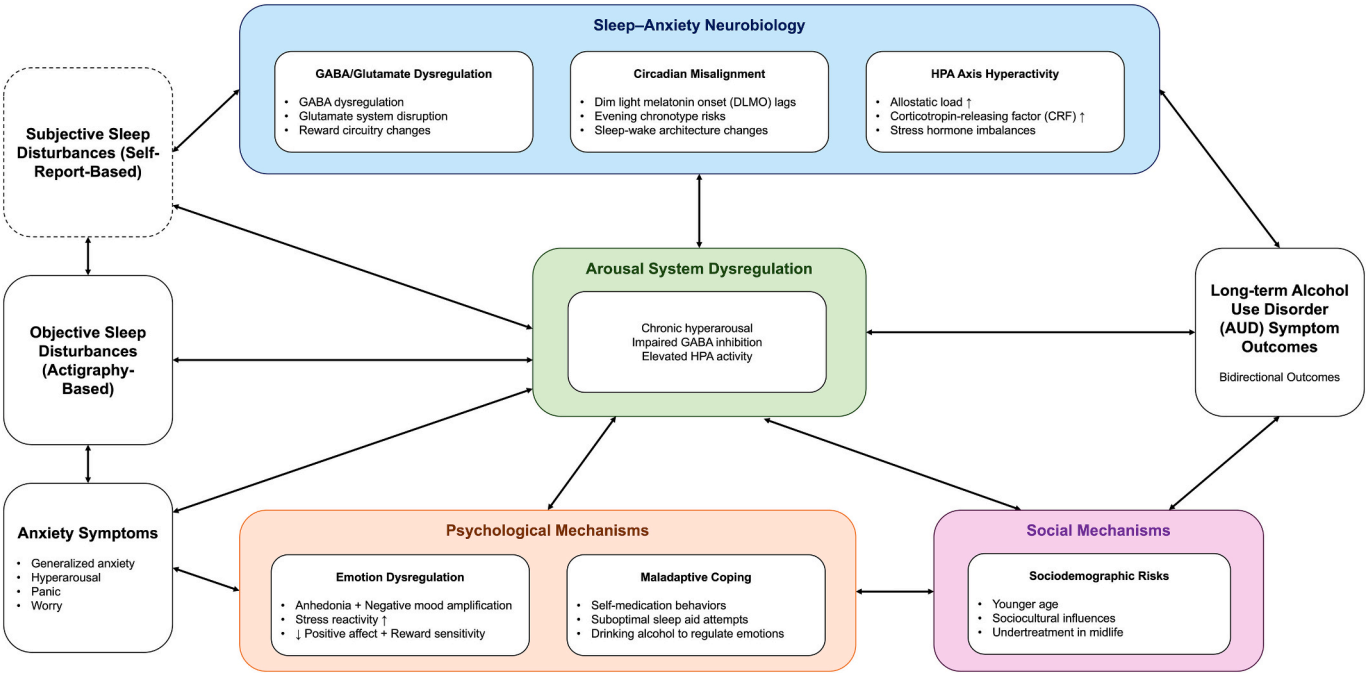

Fig. 3. Theoretical pathways based on the sleep-anxiety dysregulation model of alcohol use disorder risk
Note. GABA, gamma-aminobutyric acid; HPA, hypothalamus-pituitary axis.

actigraphy, potentially negatively affecting sleep onset and prolonging circadian misalignment that sustains AUD symptoms. Active wake stage predictors, including fewer wake bouts and lower physical activity counts, as well as more sleep bouts, likely indicated issues with delayed or evening chronotype and sleep-wake boundary regulation. These outcomes might be attributed to impulse control problems that could be persistent in those with or at risk for increased alcohol use (Sirtoli et al., 2023). Higher allostatic load, cumulative wear-and-tear from persistent physiological burdens and stressors, could aggravate sleep-anxiety dysregulation and prolong alcohol misuse via continued HPA axis activation (Guidi et al., 2021). Collectively, these bidirectional and nonlinear patterns expand on prior work by identifying actigraphy-based indices of circadian disturbances and rest-stage arousal factors as essential predictors of nine-year AUD severity. They emphasize the theory's primary hypothesis that persistent sleep-wake circadian disturbances and anxiety issues jointly contribute to long-term excessive alcohol use.

Additionally, younger adults were more susceptible to drinking more alcohol in the long term than their middle-aged and older adult counterparts. This observation might be due to several developmental considerations. Those who started drinking excessively earlier on might be more likely to accrue more binge drinking episodes over time, gradually leading to more deeply ingrained, maladaptive alcohol use habits in the long run (Merline et al., 2008). Earlier alcohol use could also prolong decision-making deficits, externalizing behaviors, and impulse control issues (Nurnberger Jr. et al., 2019). Such entrenched alcohol use patterns might evolve into alcohol dependence that could be difficult to "mature out of" as these individuals grow older (Hingson et al., 2006). Together, this finding highlights the importance of early identification and intervention.

Another noteworthy outcome was that objective, actigraphy-based indices, rather than subjective PSQI-based self-reports of sleep disturbances, predicted higher AUD severity nine years later. A plausible account relates to the capacity of actigraphy to capture physiological aspects of active wake, rest, and sleep stages that are often missed by subjective self-reports of sleep (Brooks et al., 2012). Across long durations, sleep self-reports may be influenced by memory, mood, and personal biases (Piekarski et al., 2022), which can be better accounted for by psychiatric comorbidities, such as GAD and PD symptoms, that have emerged as top predictors. Comparatively, actigraphy markers might do a better job of capturing circadian rhythms, such as sleep continuity and fragmentation, that converging evidence would suggest are essential for predicting future AUD severity (Brooks et al., 2020). To this end, actigraphy markers could be an improvement over sleep self-reports by identifying sleep disturbances that would be subtle yet meaningful indicators of persistent alcohol use.

Our findings and theory should be interpreted under some limitations. First, our *Sleep-Anxiety Dysregulation Model of AUD Risk* posits reciprocal, longitudinal associations between sleep disturbances, anxiety symptoms, and AUD symptoms, aligned with existing literature (Helaakoski et al., 2022). However, since our goal was to examine the etiological importance of sleep disturbances and anxiety symptoms in predicting long-term AUD symptoms, future studies should focus on the opposite pathway of how AUD symptoms precede and predict sleep disturbances. Second, genetic factors (Hatoum et al., 2022) and related confounders should be adjusted in future longitudinal studies assessing the proposed theoretical tenets. Third, later investigations should explore the boundary conditions–distinct measures, time-lag durations, sample attributes, and analytic approach–within which sleep disturbances and anxiety predict long-term AUD symptoms. Fourth, external validation is required before an actionable prognostic calculator can be built and implemented in clinical and routine care settings (Oliver, 2022). Fifth, as no diagnostic measures of AUD were administered, we were unable to ascertain the proportion of individuals with clinical levels of AUD at W1 and W2. Future replication studies should thus include and model AUD diagnostic status. Simultaneously, we believe our use of the MAST as a dimensional severity measure remains justified and appropriate for understanding AUD risk and trajectories in this population-based cohort. Nonetheless, the study's strengths included the nine-year time lag, large community sample, and robust multivariate NCV ML analyses.

Several clinical implications merit consideration if future studies were to replicate similar findings in diverse populations and offer evidence for external validation (Collins et al., 2024). Our findings may point to a modifiable risk phenotype–characterized by chronic sleep problems, anxiety symptoms, and maladaptive coping–that may precede

the onset of AUD, and for whom early, tailored intervention may prevent the escalation into chronic AUD. By identifying individuals with this pattern early, particularly in midlife when both sleep and anxiety disturbances often become entrenched yet remain under-recognized, there may be a critical opportunity to intervene before the development of chronic AUD. This interpretation builds on and expands beyond traditional self-medication models by showing how subjective sleep dysfunction and anxiety co-occurrence predict long-term alcohol risk through **alcohol**-specific biobehavioral pathways rather than more general alcohol use vulnerability. Although most existing interventions targeting sleep in the context of AUD focus on relapse prevention among individuals already diagnosed with the disorder (cf. meta-analysis by Miller et al., 2017), the present findings highlight the need for early, targeted prevention strategies. Specifically, adults presenting with chronic sleep disturbances and anxiety symptoms, particularly those with evening chronotypes or misaligned sleep-wake patterns, may represent a high-risk subgroup for whom brief, tailored interventions could be particularly impactful. Behavioral sleep interventions that incorporate biofeedback (Penzlin et al., 2015), progressive muscle relaxation (Murphy et al., 2019), and sleep restriction (Geoffroy et al., 2020) may be adapted to concurrently address anxiety-related hyperarousal and alcohol expectancies related to sleep (e.g., using alcohol as a sedative). It might simultaneously improve sleep and decrease AUD risk in the long run. Delivering these interventions via digital health platforms or just-in-time adaptive interventions (Zainal et al., 2025) could enhance accessibility and support the real-time use of skills in daily life. Embedding such approaches into primary care or behavioral health screening settings, before the emergence of full-syndrome AUD, offers a promising public health strategy (Williamson et al., 2022). Overall, findings may support a shift toward proactively addressing sleep-anxiety dysregulation as a modifiable pathway for AUD prevention rather than solely as a maintenance factor in those already in treatment.

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Preprints

This manuscript has not been posted on any preprint server or elsewhere.

CRediT authorship contribution statement

Nur Hani Zainal: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Natalia Van Doren:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Investigation, Data curation, Conceptualization.

Ethical standards

Our study received approval from the institutional review boards (IRB) of universities that participated in the Midlife Development in the United States (MIDUS) project. Informed consent was obtained from participants per IRB requirements at Harvard University, Georgetown University, the University of California at Los Angeles, and the University of Wisconsin at Madison. Since this study used a publicly available dataset, it was exempt from IRB approval.

Declaration of Generative AI and AI-assisted technologies in the writing process

The authors confirm that no generative AI tools were used in the writing process of this manuscript.

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Declaration of competing interest

The authors have no financial or personal relationships with organizations or individuals that could bias the work reported in this study.

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References

American Psychiatric Association, A. P. A, 2013. Diagnostic and Statistical Manual of Mental Disorders (DSM-5; 5th ed.). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596.

AshaRani, P.V., Karuvetil, M.Z., Brian, T.Y.W., Satghare, P., Roystonn, K., Peizhi, W., Subramaniam, M., 2022. Prevalence and correlates of physical comorbidities in alcohol use disorder (AUD): a pilot study in treatment-seeking population. Int. J. Ment. Heal. Addict. 1-18. https://doi.org/10.1007/s11469-021-00734-5.

Assari, S., Lankarani, M.M., 2016. Education and alcohol consumption among older Americans; Black-White differences. Front. Public Health 4, 67. https://doi.org/10.3389/fpubh.2016.00067.

Bakhshaie, J., Zvolensky, M.J., Goodwin, R.D., 2015. Cigarette smoking and the onset and persistence of depression among adults in the United States: 1994-2005. Compr. Psychiatry 60, 142–148. https://doi.org/10.1016/j.comppsych.2014.10.012.

Bakhshaie, J., Zvolensky, M.J., Goodwin, R.D., 2016. Cigarette smoking and the onset and persistence of panic attacks during mid-adulthood in the United States: 1994-2005. J. Clin. Psychiatry 77 (1), e21–e24. https://doi.org/10.4088/JCP.14m09290.

Bernstein, D.P., Fink, L., 1998. Childhood Trauma Questionnaire: A Retrospective Self-Report Manual. The Psychological Corporation.

Bernstein, D.P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., Ruggiero, J., 1994. Initial reliability and validity of a new retrospective measure of child abuse and neglect. Am. J. Psychiatry 151 (8), 1132–1136. https://doi.org/10.1176/ajp.151.8.1132.

Bhat, A.C., Diaz, J.A., Lee, S.A., Almeida, D.M., Lee, S., 2024. Associations between recession hardships and subjective and objective sleep measures in the midlife in the United States study: race and gender differences. Front. Sleep 3. https://doi.org/10.3389/frsle.2024.1403818.

Blackwell, M., Glynn, A.N., 2018. How to make causal inferences with time-series cross-sectional data under selection on observables. Am. Polit. Sci. Rev. 112 (4), 1067–1082. https://www.jstor.org/stable/26592097.

Breiman, L., 2001. Random forests. Mach. Learn. 45 (1), 5–32. https://doi.org/10.1023/A:1010933404324.

Breslau, N., Roth, T., Rosenthal, L., Andreski, P., 1996. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol. Psychiatry 39 (6), 411–418. https://doi.org/10.1016/0006-3223(95)00188-3.

Brockdorf, A.N., Haws, J.K., Brock, R.L., Gratz, K.L., Messman, T.L., DiLillo, D., 2022. Drinking to cope as a mechanism between sleep problems and heavy episodic drinking among trauma survivors. Psychol. Addict. Behav. 36 (4), 387–396. https://doi.org/10.1037/adb0000709.

Brooks, A.T., Krumlauf, M.C., Whiting, B.P., Clark, R.J., Wallen, G.R., 2012. Are you sleeping? Pilot comparison of self-reported and objective measures of sleep quality and duration in an inpatient alcoholism treatment program. Subst. Abus. 6, 135–139. https://doi.org/10.4137/SART.S10385.

Brooks, A.T., Raju, S., Barb, J.J., Kazmi, N., Chakravorty, S., Krumlauf, M., Wallen, G.R., 2020. Sleep regularity index in patients with alcohol dependence: daytime napping and mood disorders as correlates of interest. Int. J. Environ. Res. Public Health 17 (1). https://doi.org/10.3390/ijerph17010331.

Brooks, A.T., Kazmi, N., Yang, L., Tuason, R.T., Krumlauf, M.C., Wallen, G.R., 2021. Sleep-related cognitive/behavioral predictors of sleep quality and relapse in individuals with alcohol use disorder. Int. J. Behav. Med. 28 (1), 73–82. https://doi.org/10.1007/s12529-020-09901-9.

Burgess, H.J., Rizvydeen, M., Kikyo, F., Kebbeh, N., Tan, M., Roecklein, K.A., Cao, D., 2022. Sleep and circadian differences between light and heavy adult alcohol drinkers. Alcohol. Clin. Exp. Res. 46 (7), 1181–1191. https://doi.org/10.1111/acer.14872.

Buysse, D.J., Reynolds 3rd, C.F., Monk, T.H., Berman, S.R., Kupfer, D.J., 1989. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res. 28 (2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4.

Carpenter, J.S., Andrykowski, M.A., 1998. Psychometric evaluation of the Pittsburgh sleep quality index. J. Psychosom. Res. 45 (1), 5–13. https://doi.org/10.1016/s0022-3999(97)00298-5.

Castillo-Carniglia, A., Keyes, K.M., Hasin, D.S., Cerda, M., 2019. Psychiatric comorbidities in alcohol use disorder. Lancet Psychiatry 6 (12), 1068–1080. https://doi.org/10.1016/S2215-0366(19)30222-6.

Chakravorty, S., Kember, R.L., Mazzotti, D.R., Dashti, H.S., Toikumo, S., Gehrman, P.R., Kranzler, H.R., 2023. The relationship between alcohol- and sleep-related traits: Results from polygenic risk score and Mendelian randomization analyses. Drug Alcohol Depend. 251, 110912. https://doi.org/10.1016/j.drugalcdep.2023.110912.

Chen, J.-J., Liu, L.-F., Chang, S.-M., Lu, C.-P., 2023. Identifying the top determinants of psychological resilience among community older adults during COVID-19 in Taiwan: a random forest approach. Mach. Learn. Appl. 14. https://doi.org/10.1016/j.mlwa.2023.100494.

Collins, G.S., Dhiman, P., Ma, J., Schlussel, M.M., Archer, L., Van Calster, B., Riley, R.D., 2024. Evaluation of clinical prediction models (part 1): from development to external validation. BMJ 384, e074819. https://doi.org/10.1136/bmj-2023-074819.

Cox, R.C., Olatunji, B.O., 2016. A systematic review of sleep disturbance in anxiety and related disorders. J. Anxiety Disord. 37, 104–129. https://doi.org/10.1016/j.janxdis.2015.12.001.

Daskalopoulou, C., Stubbs, B., Kralj, C., Koukounari, A., Prince, M., Prina, A.M., 2018. Associations of smoking and alcohol consumption with healthy ageing: a systematic review and meta-analysis of longitudinal studies. BMJ Open 8 (4), e019540. https://doi.org/10.1136/bmjopen-2017-019540.

Davis, J.P., Prindle, J., Saba, S.K., DiGuiseppi, G.T., Hummer, J., Lee, D.S., Pedersen, E.R., 2022. What's sleep got to do with it? Longitudinal associations between insomnia, PTSD, and alcohol use among U.S. Veterans. Addict. Behav. 132, 107358. https://doi.org/10.1016/j.addbeh.2022.107358.

Dvorak, R.D., Sargent, E.M., Kilwein, T.M., Stevenson, B.L., Kuvaas, N.J., Williams, T.J., 2014. Alcohol use and alcohol-related consequences: associations with emotion regulation difficulties. Am. J. Drug Alcohol Abuse 40 (2), 125–130. https://doi.org/10.3109/00952990.2013.877920.

Dzierzewski, J.M., Ravyts, S.G., Martin, C.E., Polak, K.M., Nielson, S.A., Pomm, D., Svikis, D.S., 2022. Sleep disturbance and problematic alcohol use: examination of sex and race differences. Front. Sleep 1. https://doi.org/10.3389/frsle.2022.1014610.

Ebrahimi, A., Wiil, U.K., Schmidt, T., Naemi, A., Nielsen, A.S., Shaikh, G.M., Mansourvar, M., 2021. Predicting the risk of alcohol use disorder using machine learning: a systematic literature review. IEEE Access 9, 151697–151712. https://doi.org/10.1109/access.2021.3126777.

Elhai, J.D., Montag, C., 2020. The compatibility of theoretical frameworks with machine learning analyses in psychological research. Curr. Opin. Psychol. 36, 83–88. https://doi.org/10.1016/j.copsyc.2020.05.002.

Farahani, H.A., Rahiminezhad, A., Same, L., immannezhad, K., 2010. A comparison of partial least squares (PLS) and ordinary least squares (OLS) regressions in predicting of couples mental health based on their communicational patterns. Procedia. Soc. Behav. Sci. 5, 1459–1463. https://doi.org/10.1016/j.sbspro.2010.07.308.

Fife, D.A., D'Onofrio, J., 2023. Common, uncommon, and novel applications of random forest in psychological research. Behav. Res. Methods 55 (5), 2447–2466. https://doi.org/10.3758/s13428-022-01901-9.

Fokkema, M., Iliescu, D., Greiff, S., Ziegler, M., 2022. Machine learning and prediction in psychological assessment. Eur. J. Psychol. Assess. 38 (3), 165–175. https://doi.org/10.1027/1015-5759/a000714.

Forrester, S.N., Leoutsakos, J.M., Gallo, J.J., Thorpe Jr., R.J., Seeman, T.E., 2019. Association between allostatic load and health behaviours: a latent class approach. J. Epidemiol. Community Health 73 (4), 340–345. https://doi.org/10.1136/jech-2018-211289.

Freeman, L.K., Miller, M.B., Simenson, A., Sparrow, E., Costa, A.N., Musich, M., Curtis, A.F., 2022. Sex differences in associations between alcohol use and sleep in mid-to-late life. Sleep Med. 100, 298–303. https://doi.org/10.1016/j.sleep.2022.08.016.

Gao, J., 2023. R-squared (R2) – how much variation is explained? Res. Methods Med. Health Sci. 5 (4), 104–109. https://doi.org/10.1177/26320843231186398.

Gardiner, C., Weakley, J., Burke, L.M., Roach, G.D., Sargent, C., Maniar, N., Halson, S.L., 2025. The effect of alcohol on subsequent sleep in healthy adults: a systematic review and meta-analysis. Sleep Med. Rev. 80, 102030. https://doi.org/10.1016/j.smrv.2024.102030.

Geoffroy, P.A., Lejoyeux, M., Rolland, B., 2020. Management of insomnia in alcohol use disorder. Expert Opin. Pharmacother. 21 (3), 297–306. https://doi.org/10.1080/14656566.2019.1705279.

Geoghegan, P., O'Donovan, M.T., Lawlor, B.A., 2012. Investigation of the effects of alcohol on sleep using actigraphy. Alcohol Alcohol. 47 (5), 538–544. https://doi.org/10.1093/alcalc/ags054.

Gigantesco, A., Morosini, P., 2008. Development, reliability and factor analysis of a self-administered questionnaire which originates from the World Health Organization's composite international diagnostic interview - short form (CIDI-SF) for assessing mental disorders. Clin. Pract. Epidemiol. Ment. Health 4 (1), 8. https://doi.org/10.1186/1745-0179-4-8.

Gilman, J.M., Ramchandani, V.A., Davis, M.B., Bjork, J.M., Hommer, D.W., 2008. Why we like to drink: a functional magnetic resonance imaging study of the rewarding and anxiolytic effects of alcohol. J. Neurosci. 28 (18), 4583–4591. https://doi.org/10.1523/JNEUROSCI.0086-08.2008.

Golino, H.F., Gomes, C.M.A., 2016. Random forest as an imputation method for education and psychology research: its impact on item fit and difficulty of the Rasch model. Int. J. Res. Method Educ. 39 (4), 401–421. https://doi.org/10.1080/1743727x.2016.1168798.

Gondre-Lewis, M.C., Warnock, K.T., Wang, H., June Jr., H.L., Bell, K.A., Rabe, H., June Sr., H.L., 2016. Early life stress is a risk factor for excessive alcohol drinking and impulsivity in adults and is mediated via a CRF/GABA(A) mechanism. Stress 19 (2), 235–247. https://doi.org/10.3109/10253890.2016.1160280.

Grant, B.F., Chou, S.P., Saha, T.D., Pickering, R.P., Kerridge, B.T., Ruan, W.J., Hasin, D.S., 2017. Prevalence of 12-month alcohol use, high-risk drinking, and DSM-IV alcohol use disorder in the United States, 2001-2002 to 2012-2013: results from the National Epidemiologic Survey on alcohol and related conditions. JAMA Psychiatry 74 (9), 911–923. https://doi.org/10.1001/jamapsychiatry.2017.2161.

Greenwell, B.M., 2017. Pdp: an R package for constructing partial dependence plots. R J. 9 (1), 421–436. https://doi.org/10.32614/RJ-2017-016.

Guidi, J., Lucente, M., Sonino, N., Fava, G.A., 2021. Allostatic load and its impact on health: a systematic review. Psychother. Psychosom. 90 (1), 11–27. https://doi.org/10.1159/000510696.

Hartwell, E.E., Bujarski, S., Glasner-Edwards, S., Ray, L.A., 2015. The association of alcohol severity and sleep quality in problem drinkers. Alcohol Alcohol. 50 (5), 536–541. https://doi.org/10.1093/alcalc/agv046.

Harvey, A.G., Murray, G., Chandler, R.A., Soehner, A., 2011. Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. Clin. Psychol. Rev. 31 (2), 225–235. https://doi.org/10.1016/j.cpr.2010.04.003.

Hasler, B.P., Martin, C.S., Wood, D.S., Rosario, B., Clark, D.B., 2014. A longitudinal study of insomnia and other sleep complaints in adolescents with and without alcohol use disorders. Alcohol. Clin. Exp. Res. 38 (8), 2225–2233. https://doi.org/10.1111/acer.12474.

Hasler, B.P., Graves, J.L., Wallace, M.L., Claudatos, S., Franzen, P.L., Nooner, K.B., Clark, D.B., 2022. Self-reported sleep and circadian characteristics predict alcohol and cannabis use: a longitudinal analysis of the National Consortium on alcohol and neurodevelopment in adolescence study. Alcohol. Clin. Exp. Res. 46 (5), 848–860. https://doi.org/10.1111/acer.14808.

Hatoum, A.S., Winiger, E.A., Morrison, C.L., Johnson, E.C., Agrawal, A., 2022. Characterisation of the genetic relationship between the domains of sleep and circadian-related behaviours with substance use phenotypes. Addict. Biol. 27 (4), e13184. https://doi.org/10.1111/adb.13184.

He, S., Hasler, B.P., Chakravorty, S., 2019. Alcohol and sleep-related problems. Curr. Opin. Psychol. 30, 117–122. https://doi.org/10.1016/j.copsyc.2019.03.007.

Helaakoski, V., Kaprio, J., Hublin, C., Ollila, H.M., Latvala, A., 2022. Alcohol use and poor sleep quality: a longitudinal twin study across 36 years. Sleep Adv. 3 (1), zpac023. https://doi.org/10.1093/sleepadvances/zpac023.

Hingson, R.W., Heeren, T., Winter, M.R., 2006. Age of alcohol-dependence onset: associations with severity of dependence and seeking treatment. Pediatrics 118 (3), e755–e763. https://doi.org/10.1542/peds.2006-0223.

Hinz, A., Glaesmer, H., Brahler, E., Loffler, M., Engel, C., Enzenbach, C., Sander, C., 2017. Sleep quality in the general population: psychometric properties of the Pittsburgh sleep quality index, derived from a German community sample of 9284 people. Sleep Med. 30, 57–63. https://doi.org/10.1016/j.sleep.2016.03.008.

Hisler, G., Krizan, Z., 2017. Anger tendencies and sleep: poor anger control is associated with objectively measured sleep disruption. J. Res. Pers. 71, 17–26. https://doi.org/10.1016/j.jrp.2017.08.009.

Hoeboer, C.M., Bodor, N., Oprel, D.A.C., de Kleine, R.A., Schoorl, M., van Minnen, A., van der Does, W., 2025. Validation of the childhood trauma questionnaire (CTQ) in the context of trauma-focused treatment. Child Maltreat., 10775595251328611 https://doi.org/10.1177/10775595251328611.

Hussain, J., Ling, L., Alonzo, R.T., Rodrigues, R., Nicholson, K., Stranges, S., Anderson, K.K., 2022. Associations between sleep patterns, smoking, and alcohol use among older adults in Canada: insights from the Canadian longitudinal study on aging (CLSA). Addict. Behav. 132, 107345. https://doi.org/10.1016/j.addbeh.2022.107345.

James, G., Witten, D., Hastie, T., Tibshirani, R., 2013. An introduction to statistical learning, (Vol. 103).. Springer, New York.

Kessler, R.C., Andrews, G., Mroczek, D., Ustun, B., Wittchen, H.-U., 1998. The World Health Organization composite international diagnostic interview - short-form (CIDI-SF). Int. J. Methods Psychiatr. Res. 7 (4), 171–185. https://doi.org/10.1002/mpr.47.

Kessler, R.C., Andrews, G., Mroczek, D., Ustun, B., Wittchen, H.U., 2006. The World Health Organization composite international diagnostic interview short-form (CIDI-SF). Int. J. Methods Psychiatr. Res. 7 (4), 171–185. https://doi.org/10.1002/mpr.47.

Koob, G.F., Colrain, I.M., 2020. Alcohol use disorder and sleep disturbances: a feed-forward allostatic framework. Neuropsychopharmacology 45 (1), 141–165. https://doi.org/10.1038/s41386-019-0446-0.

Križan, Z., Hisler, G., 2019. Personality and sleep: neuroticism and conscientiousness predict behaviourally recorded sleep years later. Eur. J. Pers. 33 (2), 133–153. https://doi.org/10.1002/per.2191.

Kushner, M.G., Abrams, K., Borchardt, C., 2000. The relationship between anxiety disorders and alcohol use disorders: a review of major perspectives and findings. Clin. Psychol. Rev. 20 (2), 149–171. https://doi.org/10.1016/s0272-7358(99)00027-6.

Lechner, M., Okasa, G., 2024. Random forest estimation of the ordered choice model. Empir. Econ. 68 (1), 1–106. https://doi.org/10.1007/s00181-024-02646-4.

Lewis, M.J., Spiliopoulou, A., Goldmann, K., Pitzalis, C., McKeigue, P., Barnes, M.R., 2023. Nestedcv: an R package for fast implementation of nested cross-validation with embedded feature selection designed for transcriptomics and high-dimensional data. Bioinform. Adv. 3(1), vbad048. https://doi.org/10.1093/bioadv/vbad048.

Lindberg, D., Andres-Beck, L., Jia, Y.-F., Kang, S., Choi, D.-S., 2018. Purinergic signaling in neuron-astrocyte interactions, circadian rhythms, and alcohol use disorder. Front. Physiol. 9. https://doi.org/10.3389/fphys.2018.00009.
Lindhiem, O., Petersen, I.T., Mentch, L.K., Youngstrom, E.A., 2018. The importance of calibration in clinical psychology. Assessment 27 (4), 840–854. https://doi.org/10.1177/1073191117752055.
Liu, D., Kahathuduwa, C., Vazsonyi, A.T., 2021. The Pittsburgh sleep quality index (PSQI): psychometric and clinical risk score applications among college students. Psychol. Assess. 33 (9), 816–826. https://doi.org/10.1037/pas0001027.
Love, G.D., Seeman, T.E., Weinstein, M., Ryff, C.D., 2010. Bioindicators in the MIDUS national study: protocol, measures, sample, and comparative context. J. Aging Health 22 (8), 1059–1080. https://doi.org/10.1177/0898264310374355.
Lu, J., Yang, Y., Cui, J., Xu, W., Wu, C., Li, J., Li, X., 2022. Alcohol use disorder and its association with quality of life and mortality in Chinese male adults: a population-based cohort study. BMC Public Health 22 (1), 789. https://doi.org/10.1186/s12889-022-13146-4.
Lundberg, S.M., Lee, S.I., 2017. A unified approach to interpreting model predictions. Adv. Neural Inf. Proces. Syst. 30, 4768–4777. https://doi.org/10.5555/3295222.3295230.
Lundberg, S.M., Erion, G., Chen, H., DeGrave, A., Prutkin, J.M., Nair, B., Lee, S.I., 2020. From local explanations to global understanding with explainable AI for trees. Nat. Mach. Intell. 2 (1), 56–67. https://doi.org/10.1038/s42256-019-0138-9.
Martindale, S.L., Hurley, R.A., Taber, K.H., 2017. Chronic alcohol use and sleep homeostasis: risk factors and neuroimaging of recovery. J. Neuropsychiatry Clin. Neurosci. 29 (1). https://doi.org/10.1176/appi.neuropsych.16110307. A6-5.
Mayer, M., Watson, D., 2024. kernelshap: Kernel SHAP. In: R package version 0.7.0. https://CRAN.R-project.org/package=kernelshap.
Merline, A., Jager, J., Schulenberg, J.E., 2008. Adolescent risk factors for adult alcohol use and abuse: stability and change of predictive value across early and middle adulthood. Addiction 103 (1), 84–99. https://doi.org/10.1111/j.1360-0443.2008.02178.x.
Miller, M.B., Donahue, M.L., Carey, K.B., Scott-Sheldon, L.A.J., 2017. Insomnia treatment in the context of alcohol use disorder: a systematic review and meta-analysis. Drug Alcohol Depend. 181, 200–207. https://doi.org/10.1016/j.drugalcdep.2017.09.029.
Miller, M.B., Freeman, L.K., Deroche, C.B., Park, C.J., Hall, N.A., McCrae, C.S., 2021. Sleep and alcohol use among young adult drinkers with insomnia: a daily process model. Addict. Behav. 119, 106911. https://doi.org/10.1016/j.addbeh.2021.106911.
Mishra, S., Tripathy, H.K., Kumar Thakkar, H., Garg, D., Kotecha, K., Pandya, S., 2021. An explainable intelligence driven query prioritization using balanced decision tree approach for multi-level psychological disorders assessment. Front. Public Health 9, 795007. https://doi.org/10.3389/fpubh.2021.795007.
Molnar, C., 2022. Interpretable Machine Learning: A Guide for Making Black-Box Models Explainable, 2nd ed. https://christophm.github.io/interpretable-ml-book/cite.html.
Murphy, J.G., Dennhardt, A.A., Martens, M.P., Borsari, B., Witkiewitz, K., Meshesha, L.Z., 2019. A randomized clinical trial evaluating the efficacy of a brief alcohol intervention supplemented with a substance-free activity session or relaxation training. J. Consult. Clin. Psychol. 87 (7), 657–669. https://doi.org/10.1037/ccp0000412.
Nadel, E.L., Thornberry, T.P., 2017. Intergenerational consequences of adolescent substance use: patterns of homotypic and heterotypic continuity. Psychol. Addict. Behav. 31 (2), 200–211. https://doi.org/10.1037/adb0000248.
Nam, H.W., McIver, S.R., Hinton, D.J., Thakkar, M.M., Sari, Y., Parkinson, F.E., Choi, D. S., 2012. Adenosine and glutamate signaling in neuron-glial interactions: implications in alcoholism and sleep disorders. Alcohol. Clin. Exp. Res. 36 (7), 1117–1125. https://doi.org/10.1111/j.1530-0277.2011.01722.x.
Ng, M.H.S., Zainal, N.H., Newman, M.G., 2024. Positive reappraisal coping mediates the relationship between parental abuse and lack of affection on adulthood generalized anxiety severity. J. Anxiety Disord. 102, 102826. https://doi.org/10.1016/j.janxdis.2024.102826.
Noma, H., Shinozaki, T., Iba, K., Teramukai, S., Furukawa, T.A., 2021. Confidence intervals of prediction accuracy measures for multivariable prediction models based on the bootstrap-based optimism correction methods. Stat. Med. 40 (26), 5691–5701. https://doi.org/10.1002/sim.9148.
Nurnberger Jr., J.I., Yang, Z., Zang, Y., Acion, L., Bierut, L., Bucholz, K., Schuckit, M., 2019. Development of alcohol use disorder as a function of age, severity, and comorbidity with externalizing and internalizing disorders in a young adult cohort. J. Psychiatry Brain Sci. 4. https://doi.org/10.20900/jpbs.20190016.
Oliver, D., 2022. The importance of external validation to advance precision psychiatry. Lancet Reg. Health Eur. 22, 100498. https://doi.org/10.1016/j.lanepe.2022.100498.
Owens, S.L., Hunte, H.E.R., Sterkel, A., Johnson, D.A., Johnson-Lawrence, V., 2017. Association between discrimination and objective and subjective sleep measures in the midlife in the United States study adult sample. Psychosom. Med. 79 (4), 469–478. https://doi.org/10.1097/PSY.0000000000000428.
Panin, F., Peana, A.T., 2019. Sleep and the pharmacotherapy of alcohol use disorder: unfortunate bedfellows. A systematic review with meta-analysis. Front. Pharmacol. 10, 1164. https://doi.org/10.3389/fphar.2019.01164.
Pargent, F., Schoedel, R., Stachl, C., 2023. Best practices in supervised machine learning: a tutorial for psychologists. Adv. Methods Pract. Psychol. Sci. 6 (3), 25152459231162559. https://doi.org/10.1177/25152459231162559.
Patrick, M.E., Pang, Y.C., Jang, B.J., Arterberry, B.J., Terry-McElrath, Y.M., 2023. Alcohol use disorder symptoms reported during midlife: results from the monitoring the future study among US adults at modal ages 50, 55, and 60. Subst. Use Misuse 58 (3), 380–388. https://doi.org/10.1080/10826084.2022.2161826.
Penzlin, A.I., Siepmann, T., Illigens, B.M., Weidner, K., Siepmann, M., 2015. Heart rate variability biofeedback in patients with alcohol dependence: a randomized controlled study. Neuropsychiatr. Dis. Treat. 11, 2619–2627. https://doi.org/10.2147/NDT.S84798.
Piekarski, D., Sullivan, E.V., Pfefferbaum, A., Zahr, N.M., 2022. Poor subjective sleep predicts compromised quality of life but not cognitive impairment in abstinent individuals with alcohol use disorder. Alcohol 103, 37–43. https://doi.org/10.1016/j.alcohol.2022.07.001.
Qiu, J., 2024. An analysis of model evaluation with cross-validation: techniques, applications, and recent advances. Adv. Econ. Manag. Polit. Sci. 99 (1), 69–72. https://doi.org/10.54254/2754-1169/99/2024ox0213.
R Core Team, 2025. R: A language and environment for statistical computing. In: (Version 4.4.1) R Foundation for Statistical Computing. https://www.R-project.org/.
Radler, B.T., Love, G.D., 2018. Behind the scenes in integrative health science: Understanding and negotiating data management challenges. In: Ryff, C.D., Krueger, R.F. (Eds.), The Oxford Handbook of Integrative Health Science. Oxford University Press, pp. 23–34. https://doi.org/10.1093/oxfordhb/9780190676384.013.2.
Rights, J.D., Cole, D.A., 2018. Effect size measures for multilevel models in clinical child and adolescent research: new R-squared methods and recommendations. J. Clin. Child Adolesc. Psychol. 47 (6), 863–873. https://doi.org/10.1080/15374416.2018.1528550.
de Rooij, M., Weeda, W., 2020. Cross-validation: a method every psychologist should know. Adv. Methods Pract. Psychol. Sci. 3 (2), 248–263. https://doi.org/10.1177/2515245919898466.
Rudenstine, S., Espinosa, A., Kumar, A., 2020. Depression and anxiety subgroups across alcohol use disorder and substance use in a national epidemiologic study. J. Dual Diagn. 16 (3), 299–311. https://doi.org/10.1080/15504263.2020.1784498.
Ryff, C., Almeida, D., Ayanian, J., Binkley, N., Carr, D.S., Coe, C., Williams, D., 2019a. Midlife in the United States (MIDUS 3), 2013–2014 Inter-University Consortium for Political and Social Research [Distributor]. https://doi.org/10.3886/ICPSR36346.v7.
Ryff, C.D., Seeman, T., Weinstein, M., 2019b. Midlife in the United States (MIDUS 2): Biomarker Project, 2004–2009 Inter-University Consortium for Political and Social Research [Distributor]. https://doi.org/10.3886/ICPSR29282.v9.
Ryff, C.D., Almeida, D.M., Ayanian, J.Z., Carr, D.S., Cleary, P.D., Coe, C., Williams, D.R., 2021. Midlife in the United States (MIDUS 2), 2004–2006 Inter-University Consortium for Political and Social Research [Distributor]. https://doi.org/10.3886/ICPSR04652.v8.
Sabatier, T., Kousignian, I., Gomajee, R., Barry, K.M., Melchior, M., Mary-Krause, M., 2025. Sleep disturbances during childhood can predict adult alcohol consumption: a longitudinal cohort study. Subst. Use Misuse 60 (4), 487–495. https://doi.org/10.1080/10826084.2024.2434674.
Selzer, M.L., 1971. The Michigan alcoholism screening test: the quest for a new diagnostic instrument. Am. J. Psychiatry 127 (12), 1653–1658. https://doi.org/10.1176/ajp.127.12.1653.
Shah, A.D., Bartlett, J.W., Carpenter, J., Nicholas, O., Hemingway, H., 2014. Comparison of random forest and parametric imputation models for imputing missing data using MICE: a CALIBER study. Am. J. Epidemiol. 179 (6), 764–774. https://doi.org/10.1093/aje/kwt312.
Sheetal, A., Jiang, Z., Di Milia, L., 2023. Using machine learning to analyze longitudinal data: a tutorial guide and best-practice recommendations for social science researchers. Appl. Psychol. 72 (3), 1339–1364. https://doi.org/10.1111/apps.12435.
Shin, S.H., Jiskrova, G.K., Wills, T.A., 2019. Childhood maltreatment and alcohol use in young adulthood: the role of self-regulation processes. Addict. Behav. 90, 241–249. https://doi.org/10.1016/j.addbeh.2018.11.006.
Shrivastava, D., Jung, S., Saadat, M., Sirohi, R., Crewson, K., 2014. How to interpret the results of a sleep study. J. Community Hosp. Intern. Med. Perspect. 4 (5), 24983. https://doi.org/10.3402/jchimp.v4.24983.
Sirtoli, R., Mata, G.D.G., Rodrigues, R., Martinez-Vizcaino, V., Lopez-Gil, J.F., Guidoni, C. M., Mesas, A.E., 2023. Is evening chronotype associated with higher alcohol consumption? A systematic review and meta-analysis. Chronobiol. Int. 40 (11), 1467–1479. https://doi.org/10.1080/07420528.2023.2256899.
Smith, N., Hill, R., Marshall, J., Keaney, F., Wanigaratne, S., 2014. Sleep related beliefs and their association with alcohol relapse following residential alcohol detoxification treatment. Behav. Cogn. Psychother. 42 (5), 593–604. https://doi.org/10.1017/S1352465813000465.
Smith, J.P., Randall, C.L., 2012. Anxiety and alcohol use disorders: comorbidity and treatment considerations. Alcohol Research: Current Reviews 34 (4), 414–431.
Song, Q.C., Tang, C., Wee, S., 2021. Making sense of model generalizability: a tutorial on cross-validation in R and shiny. Adv. Methods Pract. Psychol. Sci. 4 (1), 2515245920947067. https://doi.org/10.1177/2515245920947067.
Speranza, A.M., Liotti, M., Spoletini, I., Fortunato, A., 2023. Heterotypic and homotypic continuity in psychopathology: a narrative review. Front. Psychol. 14. https://doi.org/10.3389/fpsyg.2023.1194249.
Teas, E., Friedman, E., 2021. Sleep and functional capacity in adults: cross-sectional associations among self-report and objective assessments. Sleep Health 7 (2), 198–204. https://doi.org/10.1016/j.sleh.2020.12.001.
Thompson, L.M., Slavish, D.C., Messman, B.A., Dietch, J.R., Kelly, K., Ruggero, C., Blumenthal, H., 2024. Alcohol use predicts longer but more fragmented sleep: a daily diary study of alcohol, sleep, and PTSD in nurses. Int. J. Behav. Med. https://doi.org/10.1007/s12529-024-10308-z.
Tracy, E.L., Reid, K.J., Baron, K.G., 2021. The relationship between sleep and physical activity: the moderating role of daily alcohol consumption. Sleep 44 (10). https://doi.org/10.1093/sleep/zsab112.

Troxel, W.M., Rodriguez, A., Seelam, R., Tucker, J.S., Shih, R.A., Dong, L., D'Amico, E.J., 2021. Longitudinal associations of sleep problems with alcohol and cannabis use from adolescence to emerging adulthood. Sleep 44 (10). https://doi.org/10.1093/sleep/zsab102.

Turiano, N.A., Whiteman, S.D., Hampson, S.E., Roberts, B.W., Mroczek, D.K., 2012. Personality and substance use in midlife: conscientiousness as a moderator and the effects of trait change. J. Res. Pers. 46 (3), 295–305. https://doi.org/10.1016/j.jrp.2012.02.009.

Uysal, A., Aykutoglu, B., Ascigil, E., 2019. Basic psychological need frustration and health: prospective associations with sleep quality and cholesterol. Motiv. Emot. 44 (2), 209–225. https://doi.org/10.1007/s11031-019-09806-5.

Wang, P.S., Berglund, P., Kessler, R.C., 2000. Recent care of common mental disorders in the United States: prevalence and conformance with evidence-based recommendations. J. Gen. Intern. Med. 15 (5), 284–292. https://doi.org/10.1046/j.1525-1497.2000.9908044.x.

Warren, C.M., Riggs, N.R., Pentz, M.A., 2017. Longitudinal relationships of sleep and inhibitory control deficits to early adolescent cigarette and alcohol use. J. Adolesc. 57, 31–41. https://doi.org/10.1016/j.adolescence.2017.03.003.

Wegener, D.T., Pek, J., Fabrigar, L.R., 2024. Accumulating evidence across studies: consistent methods protect against false findings produced by p-hacking. PloS One 19 (8), e0307999. https://doi.org/10.1371/journal.pone.0307999.

Williams, L.M., Carpenter, W.T., Carretta, C., Papanastasiou, E., Vaidyanathan, U., 2024. Precision psychiatry and research domain criteria: implications for clinical trials and future practice. CNS Spectr. 29 (1), 26–39. https://doi.org/10.1017/S1092852923002420.

Williamson, A.A., Okoroji, C., Cicalese, O., Evans, B.C., Ayala, A., Harvey, B., Mindell, J. A., 2022. Sleep well! An adapted behavioral sleep intervention implemented in urban primary care. J. Clin. Sleep Med. 18 (4), 1153–1166. https://doi.org/10.5664/jcsm.9822.

Yang, P., Weng, J., Huang, X., 2024. Sleep features in alcohol use disorder: a systematic review and meta-analysis of polysomnographic findings in case-control studies. Eur. J. Psychiatry 38 (2). https://doi.org/10.1016/j.ejpsy.2023.100231.

Yarkoni, T., Westfall, J., 2017. Choosing prediction over explanation in psychology: lessons from machine learning. Perspect. Psychol. Sci. 12 (6), 1100–1122. https://doi.org/10.1177/1745691617693393.

Zainal, N.H., Newman, M.G., 2021. Within-person increase in pathological worry predicts future depletion of unique executive functioning domains. Psychol. Med. 51 (10), 1676–1686. https://doi.org/10.1017/S0033291720000422.

Zainal, N.H., Newman, M.G., 2022. Inflammation mediates depression and generalized anxiety symptoms predicting executive function impairment after 18 years. J. Affect. Disord. 296, 465–475. https://doi.org/10.1016/j.jad.2021.08.077.

Zainal, N.H., Soh, C.P., Van Doren, N., 2024. Daily stress reactivity and risk appraisal mediates childhood parental abuse predicting adulthood psychopathology severity: an 18-year longitudinal mediation analysis. J. Affect. Disord. 358, 138–149. https://doi.org/10.1016/j.jad.2024.04.068.

Zainal, N.H., Liu, X., Leong, U., Yan, X., Chakraborty, B., 2025. Bridging innovation and equity: advancing public health through just-in-time adaptive interventions. Annu. Rev. Public Health 46 (1), 43–68. https://doi.org/10.1146/annurev-publhealth-071723-103909.

Zheng, J.W., Ai, S.Z., Chang, S.H., Meng, S.Q., Shi, L., Deng, J.H., Shi, J., 2024. Association between alcohol consumption and sleep traits: observational and Mendelian randomization studies in the UK biobank. Mol. Psychiatry 29 (3), 838–846. https://doi.org/10.1038/s41380-023-02375-7.

Zvolensky, M.J., Bakhshaie, J., Sheffer, C., Perez, A., Goodwin, R.D., 2015. Major depressive disorder and smoking relapse among adults in the United States: a 10-year, prospective investigation. Psychiatry Res. 226 (1), 73–77. https://doi.org/10.1016/j.psychres.2014.11.064.