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Exploring the relationship between sleep patterns, alcohol and other substances consumption in young adults: Insights from wearables and Mobile surveys in the National Consortium on alcohol and NeuroDevelopment in adolescence (NCANDA) cohort

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ABSTRACT

Introduction: The use of psychotropic substances has negative short- and long-term health outcomes, including complex direct and indirect effects on sleep and sleep-cardiovascular function. Here, we investigate daily relationships between self-reported substance use and objective measures of sleep and sleep-related heart rate (HR) in community-dwelling young adults.

Methods: Fifty-five healthy young adults (M^{age} = 23.1 ± 2.29 y, 30 female) in the National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA) study completed a 28-day ecological momentary assessment protocol, including remote sleep and HR measurements via Fitbit devices, as well as daily app-based self-reports of alcohol and other substance use.

Results: A total of 1459 days of data were collected. Caffeine was the most frequent substance used, followed by alcohol, cannabis, nicotine, and other drugs. The analysis showed that substance use was associated with delays in sleep start and end time, reduced sleep duration and efficiency, and increased wake after sleep onset. Increases in sleep heart rate were associated with prior-day alcohol use.

Discussion: Substance use negatively influences sleep and sleep HR. These preliminary data highlight the potential value of using remote multimodal data collection to investigate the daily relationships between substance use and sleep in young adults, in an ecological setting.

1. Introduction

The consumption of psychotropic substances is common among young adults. It involves the use of substances with varying levels of legality across countries, including alcohol, caffeine, nicotine, heroin, cocaine, etc. According to the annual report of the Substance Abuse and Mental Health Services Administration (SAMHSA) from 2022, young adults (aged 18 to 25) represent an at-risk cohort for substance abuse. (SAMHSA, 2022) Besides caffeine, which is by far the most used substance worldwide, (Mahoney et al., 2019; Lone et al., 2023; Riera-Sampol et al., 2022) in 2022 in the U.S., alcohol was the most commonly used substance among young adults (17.5 million, 50.2 %), followed by

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nicotine (10.4 million, 30.0 %), cannabis (9.0 million, 25.9 %), and other illicit drugs like cocaine (1.3 million, 3.7 %), methamphetamine (161.000, 0.5 %), and heroin (55.000, 0.2 %). (SAMHSA, 2022)

All these substances may have detrimental effects across multiple health domains. For example, heavy alcohol consumption has a profound impact on cardiovascular function and is associated with increased cardiovascular risks. (Biddinger et al., 2022) Between 2015 and 2019, almost twenty-five thousand people died of heart complications due to alcohol use in the US. (CDC Centers for Disease Control and Prevention, 2023) The link between alcohol and cardiovascular health is complex with multiple factors thought to play a role in the relationship, notably including sleep. Alcohol has different effects across distinct sleep processes. A first experimental study conducted by Feige and colleagues (Feige et al., 2006) clearly showed a strong hypnagogic effect of alcohol, with reduced polysomnographic (PSG) sleep onset latency (i.e., minutes required to get asleep) and increased slow wave sleep (i.e., the so-called 'deep sleep') in the first part of the night. However, these initial sleep "improvements" were counter-balanced by a rebound effect characterized by suppression of rapid-eye-movement (REM) sleep (i.e., sleep periods characterized by muscle atonia and physiological activity almost resembling wakefulness) in the first part of the night and increased stage-1 sleep (i.e., the 'lightest' phase of non-REM sleep) in the second part. Consistent with Feige et al., (Feige et al., 2006) several studies over the years further proved the negative impact of alcohol on both sleep macro- and micro-structure. Indeed, in healthy adults not only has alcohol been proven to reduce total sleep time (i.e., time spent sleeping) and sleep efficiency (i.e., proportion of time spent sleeping over time in bed), but also to reduce REM sleep and increase wakefulness. (Sagawa et al., 2011; Arnedt et al., 2011; Pabon et al., 2022) Interestingly, while the above studies showed the possible negative effect of nighttime alcohol consumption on subsequent sleep, an open question is whether this effect changes depending on the time between consumption and sleep onset. The few studies on the topic suggested more pronounced effects when alcohol is taken in the nighttime compared to the afternoon. (Van Reen et al., 2011a; Yules et al., 1966) Sleep alterations were also reported after caffeine consumption, consisting of an overall reduction in sleep efficiency and total sleep time (see Gardiner et al., (Gardiner et al., 2023) for a meta-analysis of experimental studies). Similar effects were reported for nicotine. (Jaehne et al., 2009; Jaehne et al., 2015) Cannabis seems to particularly affect REM sleep by reducing its duration across the night. (Babson et al., 2017) Evidence of the negative effects of substance consumption has also been reported by actigraphic studies, which detected a negative effect of alcohol (Fucito et al., 2018; Miller et al., 2021) and cannabis (Bell et al., 2022) on sleep efficiency. Survey studies also reported an association between worse sleep quality and alcohol, (Graupensperger et al., 2021) caffeine, (Riera-Sampol et al., 2022) and nicotine. (Kang and Bae, 2021) Conversely, mixed results were found for cannabis. (Graupensperger et al., 2021; Drazdowski et al., 2021) It is important to note that the magnitude of these effects may depend on several factors, including dose and time of substance use, metabolism of individuals, and demographic variables.

Less investigated are the specific effects of alcohol and other substance use on sleep cardiovascular restoration. Sagawa et al. (Sagawa et al., 2011) conducted a laboratory study in a sample of 10 young male adults to investigate the effects of alcohol on sleep and heart rate (HR) variability (HRV). Specifically, participants underwent three non-consecutive nights of alcohol-dose administration, followed by sleep PSG recording and HRV monitoring. Results showed a dose-dependent effect of alcohol, with increased HR and decreased HRV associated with higher doses. A similar experiment was conducted by de Zambotti and colleagues (de Zambotti et al., 2021) in a sample of 26 healthy participants (30–60 y). The authors compared three nights of PSG recordings in which different doses of alcohol were administered before bedtime. Both low (1 standard drink for women and 2 for men) and high (3 standard drinks for women and 4 for men) doses of alcohol strongly affected sleep CV function. Specifically, HR was higher during the first 4 h of the low-dose sleep night and during the first 6 h of the high-dose sleep night. Lastly, in women, systolic blood pressure showed increases in the morning hours after high-dose alcohol consumption. Others have reported that nicotine and caffeine also affect sleep CV function by enhancing sympathetic activity during sleep. (Choi et al., 2017; Bonnet et al., 2005) Still largely unknown is the synergistic effect of alcohol and other substances on sleep cardiovascular function.

In the current study, we adopted a 28-day ecological momentary assessment (EMA) design to further investigate the relationship between alcohol and other substance use with sleep in a sample of healthy young adults from the US National Consortium on Alcohol and NeuroDevelopment in Adolescence (NCANDA) longitudinal study. A combination of passive wearable acquisition with daily surveys (high-density daily data sampling) is particularly well-suited for studying real-time relationships between biopsychological factors and behaviors. (Brannon et al., 2016) Recent studies have exploited its potential by studying the interactions between daily sleep and adolescent behaviors, (Menghini et al., 2023) and daily HR variability in smokers. (Bodin et al., 2017) However, to our knowledge, the combination of high density wearable and survey data collection has not been used for an analysis of the relationship between substance use and sleep patterns in young adults.

Considering the evidence in the literature, here we hypothesize that alcohol and other substances (i.e., caffeine, nicotine, cannabis, and other drugs) are associated with negative outcomes in sleep behavior (i.e., sleep schedules such as bedtime and wake times), sleep macro-structure (i.e., night-level sleep parameters such as sleep duration and sleep efficiency), and sleep-related HR.

2. Method

2.1. Participants

Fifty-five (55) healthy young adults (Mean age = 23.24 ± 2.42 years, 30 female, 25 male) who were participating in the 8th and 9th year follow-ups of the NCANDA study (Brown et al., 2015) at the SRI International site, took part in a 28-day Ecological Momentary Assessment (EMA) of sleep, alcohol, and other substance use. Data were collected between January and April 2022. Inclusion criteria for the NCANDA study at baseline included an age range between 12.0 and 21.9 years, absence of medical and psychiatric disorders, absence of developmental disorders, and absence of substance dependence (Brown et al., 2015). Inclusion criteria to be involved in the 28-day EMA, were to be enrolled in the main NCANDA study, age > 18 years, absence of medical, psychiatric, and substance use disorders (assessed at the annual NCANDA assessment), and to have a Fitbit-compatible phone.

The Advara Institutional Review Board and the SRI International Institutional Review Board approved the study protocol. Participants in NCANDA completed consent if they were older than 18 years, or assent, with a parent completing consent, if they were younger than 18 years. In order to participate in the EMA and Fitbit portion of the study, they completed an additional specific written consent form and received additional monetary compensation.

See Table 1, for demographics and sample characterization. Data were obtained through the Research Electronic Data Capture (REDCap) data management online platform. (Harris et al., 2009; Harris et al., 2019) These data were part of the public data release NCANDA_RELEASE_BASE_REDCAP_MEASUREMENTS_V07, whose collection and distribution were supported by the National Institute on Alcohol Abuse and Alcoholism funding AA021697, AA021695, AA021692, AA021696, AA021681, AA021690, and AA021691.

BMI = Body Mass Index; EMA = Ecological Momentary Assessment; PSQI = Pittsburgh Sleep Quality Index (Buysse et al., 1989).

Table 1
Sample characterization.

		Women	Men	Total
Sample, n.		30	25	55
Age, years, M (SD) (range = 19–28)		23.40 (2.49)	22.80 (2.02)	23.24 (2.42)
Race				
	Asian, n.	7	3	10
	White, n.	21	20	41
	African-American/ Black	0	2	2
	Native American/ American Indian	0	1	1
	More than one race, n.	1	0	1
Ethnicity				
	Hispanic or Latino, n.	2	5	7
Habitual Sleep and Habitual Alcohol Consumption, M (SD)				
	PSQI (range = 1–10)	3.82 (2.01)	4.44 (2.28)	4.14 (2.14)
	Alcohol in the past year, n. of drinks	68.31 (62.60)	62.17 (52.31)	65.53 (57.70)
Body Composition				
	BMI, kg x m-2, M (SD)	24.19 (4.71)	24.52 (4.11)	24. 43 (4.36)
	Healthy (BMI: 18.5 to <25), n.	20	14	34
	Overweight (BMI: 25 to <30), n.	7	8	15
	Obese (BMI: 30 or higher), n.	3	3	6
Daily Alcohol and Substances Consumption Over 28-day EMA Assessment, M (SD)				
	Alcohol drinks (n. days/total)	0.24 (0.43)	0.19 (0.35)	0.22 (0.42)
	Morning	0.00 (0.02)	0.00 (0.01)	0.00 (0.01)
	Afternoon	0.04 (0.06)	0.04 (0.10)	0.04 (0.08)
	Evening	0.22 (0.23)	0.15 (0.18)	0.18 (0.21)
	Night	0.14 (0.16)	0.16 (0.18)	0.15 (0.17)
	Alcohol drinks *per* day (n. of drink/total days)	0.85 (0.87)	0.86 (1.33)	0.85 (1.09)
	Caffeine (n. days/ total)	0.65 (0.48)	0.57 (0.50)	0.61 (0.49)
	Nicotine (n. days/ total)	0.11 (0.31)	0.08 (0.28)	0.10 (0.30)
	Cannabis (n. days/ total)	0.12 (0.33)	0.17 (0.37)	0.14 (0.35)
	Other Drugs (n. days/ total)	0.00 (0.06)	0.01 (0.33)	0.01 (0.08)

2.2. Procedure

Participants were allowed to freely decide when starting the EMA protocol in accordance with their own schedule. Specifically, most of the participants started the data collection protocol on Friday (27.5 %), 20 % on Tuesday, and 14.5 % on Monday, Wednesday, and Thursday, whereas only 3.5 % and 5.5 % started on Saturday and Sunday, respectively. The distribution of the days of the week across the 28-day EMA was comparable (Sunday = 14 %, Monday = 14.2 %, Tuesday = 14.3 %, Wednesday = 14.8 %, Thursday = 14.1 %, Friday = 14.6 %, Saturday = 14 %). During the EMA protocol, participants were asked to wear a Fitbit Charge 3 (FC3) or a Fitbit Charge 4 (FC4) (Fitbit, LLC., San Francisco, CA) 24/7 on their non-dominant wrist, to synch the device with the Fitbit App as well as to respond to a survey each morning through the mNCANDA mobile phone app. (Cummins et al., 2021) The Fitabase platform (Small Steps Labs LLC., San Diego, CA) was used to support Fitbit data collection and management. Furthermore, participants were guided to turn off the notifications and data feedback of the device and not to run any updates or change the settings. On each morning, participants were asked to sync the Fitbit device with the paired App. The entire EMA procedure is displayed in Fig. 1.

All instructions and devices were provided during an in-person lab appointment and/or via online meetings. On these occasions, participants were also guided to install Fitbit and mNCANDA apps on their mobile phones using ad-hoc e-mail addresses with no personally identifying information.

2.3. Measures

2.3.1. Fitbit

Sleep, sleep HR, and physical activity measures were obtained via FC3 and FC4 devices (firmware versions: 8.20001.96.29–20,001.88.11). These consumer-grade devices have been repeatedly evaluated against reference standard PSG. (Haghayegh et al., 2019; de Zambotti et al., 2019; Lim et al., 2023) Here, we largely followed the recommendations for data management, use, and interpretation of wearable data, as introduced by de Zambotti et al. (de Zambotti et al., n.d.)

Fitbit data (sleep and sleep HR) were exported from Fitabase to be pre-processed before analysis. We first applied the Menghini et al. (Menghini et al., 2023) analytic pipeline to precisely identify nocturnal sleep periods and filter atypical/unreliable cases. Subsequently, we used high-granularity (30-s epoch-by-epoch, EBE) data to derive the outcomes for each identified sleep period starting between 06:00 PM and 06:00 AM, with a minimum sleep duration of $\geq$3 h. Based on EBE sleep classification, we retrieved the following outcomes (see Menghini et al. (Menghini et al., 2021)): sleep period start (SPS, hh.mm, i.e., the timestamp of the first epoch included in the sleep period), sleep onset (SO, hh.mm, i.e., the timestamp of the first epoch classified as sleep), and end time (SPE, hh:mm, i.e., the timestamp of the last epoch classified as sleep), sleep period duration (SPD, hours, i.e., the time between SPS and SPE), total sleep time (TST, hours, i.e., time spent sleeping between SPS and SPE), wake after sleep onset (WASO, i.e., hours, time spent awake between SO and SPE), sleep efficiency (SE, %, i.e., percentage of time spent asleep between SPS and SPE), the percentages (over TST) of "light," "deep," and REM sleep, in addition to sleep midpoint (hh.mm, i.e., the timestamp of the halfway point between SO and SPE). Furthermore, HR per minute was used to compute mean HR (bpm) in non-REM (NREM-HR) and REM sleep (REM-HR).

2.3.2. mNCANDA survey

The mNCANDA mobile-phone survey consisted of ad-hoc questions to be answered each morning from 07:00 am (after a signal time) via the mNCANDA app. (Cummins et al., 2021) It took around 1–2 min to be completed, with no time limit. From the signal time, participants could complete the survey at any time of the day. Specifically, participants were asked to indicate the number of alcoholic drinks consumed the day before (How many alcoholic drinks did you have yesterday?), and the timing (When did you drink? Morning (05:00–12:00), Afternoon (12:00–17:00), Evening (17:00–21:00), Night (21:00–05:00)), and if they used cannabis (Did you use marijuana yesterday? Yes/No), other drugs (Did you use another drug yesterday? Yes/No), nicotine/tobacco (Did you use nicotine/tobacco yesterday? Yes/No), and caffeine (Did you drink caffeine yesterday? Yes/No). The last question checked whether the participants synchronized their Fitbit that morning (Did you sync your Fitbit today? Yes/No).

The completion rate before 1:00 pm was 95 %, and the remaining 5 % completed the interview by 6:00 pm.

After each submission, the corresponding anonymized dataset was

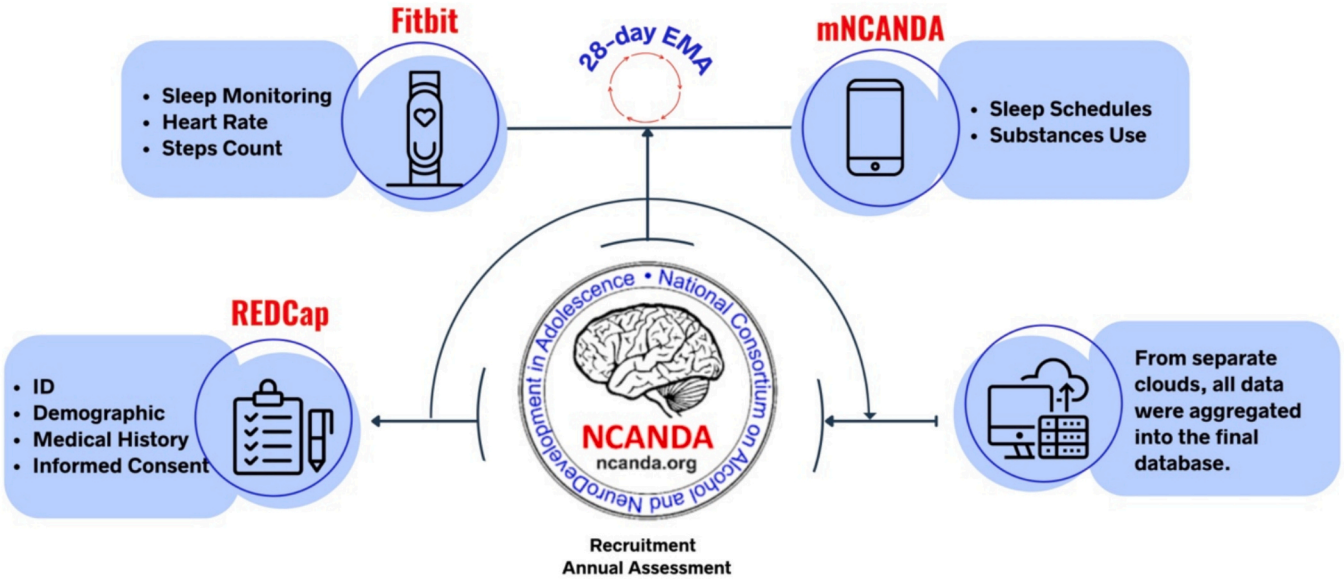

Fig. 1. Schematic description of the preliminary phases and the ecological momentary assessment (EMA) protocol of the study.

automatically loaded into the mNCANDA database and associated with the participant's identification code.

2.4. Statistical analyses

We investigated the relationships between alcohol and other substance use with Fitbit measures (sleep and sleep HR) by using linear mixed-effect regression models with the restricted maximum likelihood (REML) estimator. This approach is the most suitable for daily observations nested within participants. (Bates et al., 2014) Specifically, we specified a separate hierarchical set of models for each Fitbit measure (i.e., dependent variables). In each set of models, we firstly inserted demographic (biological sex, age, and body mass index [BMI]) and alcohol use variables as predictors, with the number of alcoholic drinks consumed each day inserted at both within- (i.e., person-mean centered) and between-person level (i.e., person means). In the second step, we included all the other substance use variables (yes/no for nicotine, cannabis, caffeine, other drugs) if they improved the model. Participants were included as random effects. For each predictor, we conducted a stepwise evaluation by considering both the Akaike Information Criterion (AIC) and Schwartz's Bayesian Information Criterion (BIC), including only the ones generating the lower AIC and BIC. This approach (i.e., stepwise) generates models with a different number of predictors, but greatly increases the goodness-of-fit of the model.

All analyses were conducted by using Jamovi 2.3.21., (The jamovi project, 2023) a statistical software based on R syntax. The chi-square (χ^2) Test was used in the case of categorical measures. Descriptive data were reported as Mean (Standard Deviation), except when otherwise specified. Confidence intervals (CI) were also reported when appropriate. The intraclass correlation coefficient (ICC) was used for each measure as an index of the person- vs day-level variability.

3. Results

3.1. Relationships between substance use, sleep timing, sleep stages, and sleep cardiac activity measures

By considering the whole sample at the within-person level, the linear mixed-effect model analysis yielded several associations between substance use and Fitbit outcomes.

Overall, the sleep variables showed higher within-individual variability (ICC ranging from 0.21 to 0.44) than HR variables (ICC ranging from 0.71 to 0.74). Furthermore, higher within-individual variability was found in alcohol use (ICC = 0.28) than in other substance use (ICC from 0.66 to 0.60 for caffeine, nicotine, and cannabis use).

Regarding sleep timing behaviors, delays in both sleep onset (AIC = 3954, BIC = 4037, R2 = 0.43, ICC = 0.41) and sleep period start (AIC = 3948, BIC = 4057, R2 = 0.43, ICC = 0.42) were predicted by nighttime alcohol and nicotine consumption. Sleep midpoint (AIC = 3583, BIC = 3682, R2 = 0.46, ICC = 0.44) and sleep period end (AIC = 3952, BIC = 4105, R2 = 0.40, ICC = 0.37) were negatively predicted by other drugs consumption (Table 2), indicating an association between other drug use and delayed sleep schedules.

Other drugs consumption was also associated with reduced sleep duration. Indeed, other drugs consumption negatively predicted both sleep period duration (AIC = 13,051, BIC = 13,055, R2 = 0.27, ICC = 0.23) and total sleep time (AIC = 12,783, BIC = 12,785, R2 = 0.27, ICC = 0.23) (Table 3). Increased wake after sleep onset (WASO) was predicted by cannabis consumption (AIC = 9826, BIC = 9848, R2 = 0.22, ICC = 0.22), whereas afternoon alcohol predicted lower sleep efficiency (AIC = 5865, BIC = 5962, R2 = 0.29, ICC = 0.31), indicating a more disrupted sleep associated with cannabis and alcohol consumption (Table 3). Light sleep duration was positively predicted by older age (AIC = 10,588, BIC = 10,501, R2 = 0.43, ICC = 0.37), whereas REM sleep duration (AIC = 9286, BIC = 9308, R2 = 0.25, ICC = 0.26) was predicted by both female gender and younger age. No significant predictor emerged for deep sleep duration (AIC = 8688, BIC = 8728, R2 = 0.22, ICC = 0.21) (Table 4).

Sleep cardiac activity showed significant associations with alcohol consumption. Specifically, increases in both NREM-HR (AIC = 5962, BIC = 6024, R2 = 0.73, ICC = 0.71) and REM-HR (AIC = 5876, BIC = 5929, R2 = 0.75, ICC = 0.74) were predicted by nighttime alcohol use. Furthermore, increased NREM-HR and REM-HR were also associated with higher BMI (Table 5).

4. Discussion

The current study used a high-density data collection protocol (28 days of remote assessments) via consumer-grade wearable devices and digital surveys, to explore the relationships between alcohol and other substances use and sleep and sleep cardiac function in young healthy adults.

Study outcomes indicated that, in young adults, drinking alcohol up to low/moderate doses (below NIAA definition of heavy drinking, i.e., ≥

Table 2
Unstandardized parameters estimated by linear mixed-effect model of the relationship between alcohol and other substance use and Fitbit sleep timing measures (N = 1459 obs. From 55 participants).

	Sleep Period Start		Sleep Period End		Sleep Onset		Sleep Midpoint	
Predictors	b(SE)	95 % CI	b(SE)	95 % CI	b (SE)	95 % CI	b (SE)	95 % CI
Intercept	24.16 (0.16)	23.85, 24.48	8.03 (0.16)	7.72, 8.33	24.27 (0.16)	23.96, 24.57	28.14 (0.14)	27.86, 28.43
Sex (F-M)	−0.48 (0.32)	−1.11, 0.16	−0.14 (0.15)	−0.76, 0.49	−0.50 (0.32)	−1.13, 0.13	−0.30 (0.29)	−0.87, 0.27
Age	−0.14 (0.07)	−0.29, 0.00	−0.08 (0.07)	−0.23, 0.06	−0.14 (0.07)	−0.29, 0.00	−0.10 (0.06)	−0.24, 0.02
BMI	0.02 (0.04)	−0.06, 0.09	−0.01 (0.04)	−0.08, 0.06	0.01 (0.03)	−0.06, 0.09	−0.00 (0.03)	−0.07, 0.07
Alcoholic Drink/Day (within)	−0.06 (0.04)	−0.15, 0.03	0.06 (0.04)	−0.03, 0.14	−0.03 (0.04)	−0.11, 0.04	0.00 (0.03)	−0.06, 0.06
Alcoholic Drink/Day (between)	−0.12 (0.15)	−0.43, 0.18	−0.24 (0.16)	−0.54, 0.06	−0.10 (0.15)	−0.41, 0.21	−0.17 (0.14)	−0.45, 0.10
Morning Alcohol	−0.16 (0.70)	−1.55, 1.23	−0.25 (0.71)	−1.64, 1.13				
Afternoon Alcohol	−0.19 (0.25)	−0.68, 0.29	−0.39 (0.25)	−0.88, 0.10	−0.24 (0.25)	−0.73, 0.25	−0.28 (0.21)	−0.70, 0.13
Evening Alcohol	0.14 (0.14)	−0.14, 0.41	−0.05 (0.14)	−0.33, 0.22				
Night Alcohol	0.43 (0.17)	0.08, 0.77*	0.10 (0.17)	−0.24, 0.45	0.38 (0.17)	0.05, 0.72*	0.27 (0.14)	−0.01, 0.55
Cannabis					−0.16 (0.18)	−0.53, 0.19		
Other Drugs	−0.05 (0.27)	−1.05, 0.94	−1.65 (0.51)	−2,65, −0,66***			−0.82 (0.43)	−1.66, 0.01*
Nicotine	0.69 (0.28)	0.15, 1.24*	0.25 (0.29)	−0.08, 0.48	0.70 (0.28)	0.16, 1.25*	0.42 (0.25)	−0.06, 0.91
Caffeine			0.19 (0.14)	−0.32, 0.83				

* $= p \leq 0.05$.
*** $= p \leq 0.001$.

Table 3
Unstandardized parameters estimated by the linear mixed-effect model of the relationship between alcohol and other substance use and Fitbit sleep structure measures (N = 1459 obs. From 55 participants).

	Sleep Period Duration		Total Sleep Time		Sleep Efficiency		Wake After Sleep Onset	
Predictors	b(SE)	95 % CI	b(SE)	95 % CI	b (SE)	95 % CI	b (SE)	95 % CI
Intercept	471.83 (7.57)	457.00, 486.66	419.38 (6.59)	406.46, 432.30	89.03 (0.30)	88.44, 89.62	46.06 (1.50)	43.11, 49.00
Sex (F-M)	21.95 (15.32)	−8.07, 51.97	18.06 (13.37)	−8.14, 44.26	−0.65 (0.59)	−1.82, 0.51	4.97 (3.04)	−0.99, 10.95
Age	4.32 (3.56)	−2.66, 11.30	2.79 (3.12)	−3.32, 8.90	−0.14 (0.14)	−0.41, 0.13	1.09 (0.70)	−0.29, 2.48
BMI	−1.69 (1.81)	−5.24, 1.85	−1.54 (1.57)	−4.62, 1.55	0.02 (0.07)	−0.11, 0.16	−0.07 (0.36)	−0.77, 0.63
Alcoholic Drink/Day (within)	2.60 (1.80)	−0.92, 6.13	3.88 (2.07)	−0.18, 7.93	0.01 (0.07)	−0.12, 0.15	1.01 (0.54)	−0.05, 2.07
Alcoholic Drink/Day (between)	−6.13 (7.63)	−21.09, 8.82	−7.19 (6.69)	−20.29, 6.92	−0.01 (0.29)	−0.58, 0.55	−1.03 (1.52)	−4.02, 1.95
Morning Alcohol					−2.35 (1.70)	−5.69, 0.98	12.22 (10.45)	−8.27, 32.72
Afternoon Alcohol					−1.39 (0.59)	−2.56, −0.23*		
Night Alcohol			−11.58 (9.51)	−30.23, 7.06			−4.77 (2.48)	−9.64, 0.10
Cannabis			17.70 (10.11)	−2.10, 37.51	−0.79 (0.43)	−1.64, 0.05	6.76 (2.53)	1.78, 11.73**
Other Drugs	−99.96 (32.47)	−160.59, −33.32**	−85.36 (28.76)	−141.73, −28.98**			−13.53 (7.50)	−28.24, 1.18
Nicotine	−7.97 (16.49)	−40.28, 24.34	−13.19 (14.71)	−42.03, 15.64				
Caffeine			12.13 (7.78)	−3.11, 27.37				

* $= p \leq 0.05$.
** $= p \leq 0.01$.

Table 4
Unstandardized parameters estimated by the linear mixed-effect model of the relationship between alcohol and other substance use and Fitbit sleep stages measures (N = 1459 obs. From 55 participants).

	Light Sleep Duration		Deep Sleep Duration		REM Sleep Duration	
Predictors	b (SE)	95 % CI	b (SE)	95 % CI	b (SE)	95 % CI
Intercept	248.14 (6.31)	235.77, 260.50	79.91 (1.83)	76.32, 83.51	89.13 (2.23)	84.57, 93.69
Sex (F-M)	7.43 (12.73)	−17.53, 32.38	−1.41 (3.71)	−8.68, 5.86	10.55 (4.69)	1.35, 19.74*
Age	7.09 (2.96)	1.29, 12.90*	−0.05 (0.86)	−1.75, 1.65	−2.60 (1.10)	−4.76, −0.44*
BMI	−1.40 (1.48)	−4.31, 1.51	0.26 (0.43)	−0.58, 1.11	−0.01 (0.55)	−1.08, 1.06
Alcoholic Drink/Day (within)	0.79 (1.20)	−1.56, 3.15	0.93 (0.67)	−0.37, 2.24	0.41 (0.65)	−0.86, 1.69
Alcoholic Drink/Day (between)	−7.02 (6.04)	−18.85, 4.80	0.51 (1.83)	−3.08, 4.11	−2.07 (2.31)	−6.61, 2.46
Morning Alcohol					−18.51 (14.80)	−47.52, 10.50
Afternoon Alcohol						
Night Alcohol			−4.66 (2.92)	−10.39, 1.07		
Other Drugs			−14.58 (9.54)	−33.28, 4.12		
Caffeine					5.74 (2.97)	−0.07, 11.56

* $= p \leq 0.05$.

Table 5
Unstandardized parameters estimated by the linear mixed-effect model of the relationship between alcohol and other substance use and Fitbit sleep cardiac activity measures ($N = 1459$ obs. From 55 participants).

	NREM Heart Rate		REM Heart Rate	
Predictors	b(SE)	95 % CI	b(SE)	95 % CI
Intercept	60.49 (1.05)	58.42, 62.56	61.65 (1.09)	59.51, 63.78
Sex (F-M)	1.66 (2.11)	−2.47, 5.80	1.85 (2.17)	−2.41, 6.11
Age	0.10 (0.49)	−0.85, 1.07	0.09 (0.50)	−0.89, 1.08
BMI	0.60 (0.25)	0.12, 1.09*	0.59 (0.25)	0.09, 1.08*
Alcoholic Drink/Day (within)	0.07 (0.16)	−0.24, 0.38	−0.01 (0.15)	−0.31, 0.28
Alcoholic Drink/Day (between)	1.04 (0.98)	−0.87, 2.96	0.58 (1.01)	−1.38, 2.56
Morning Alcohol	−4.03 (2.53)	−8.99, 0.93	−4.38 (2.42)	−9.13, 0.36
Afternoon Alcohol	−1.48 (0.93)	−3.30, 0.34	−0.84 (0.89)	−2.58, 0.90
Night Alcohol	1.44 (0.66)	0.14, 2.74*	1.26 (0.63)	0.02, 2.50*
Cannabis	−1.14 (0.72)	−2.55, 0.28		

* $= p \leq 0.05$.

4 drinks/day for women and $\geq$ 5 drinks/day for men (NIH National Institutes of Health, 2024)) and, more generally, any substance use was associated with negative outcomes in sleep behaviors, sleep structure, and sleep HR.

Overall participants reported less than one alcoholic drink consumed each day, with greater within-person variability than between-person variability in both alcoholic and sleep measures. As shown by other studies, besides the specific effect of alcohol consumption on sleep, alcohol variability has been related to higher sleep variability (Pasch et al., 2012; Heacock et al., 2022), an index that has been associated with several negative consequences on health, such as increased risk of obesity, metabolic syndrome, diabetes, and mortality. (Roenneberg et al., 2012; Koopman et al., 2017)

Regarding the relationship between alcohol use and sleep, alcohol use was associated with a delayed sleep period and afternoon alcohol consumption specifically was associated with lower sleep efficiency, denoting a negative effect on objective sleep quality. Considering the young age of our sample, the delayed sleep schedules associated with alcohol use might reflect the tendency to consume these types of substances later in the day, as others have shown (Singleton and Wolfson, 2009; Digdon and Landry, 2013), a tendency associated with other typically youthful nighttime behaviors (e.g., playing video games (De Rosa et al., 2024)). Surprisingly, the number of alcoholic drinks consumed or consumption of alcohol at night was not associated with poor sleep outcomes. In contrast, prior studies have linked alcohol consumption at night with poor sleep (Feige et al., 2006; Van Reen et al., 2011a; Yules et al., 1966). This paucity of evidence linking alcohol and more disrupted sleep should be viewed in light of the following considerations: 1) the effects of alcohol on sleep may be less pronounced in younger age, (Feige et al., 2006; Van Reen et al., 2011b) possibly due to their higher total concentration of body water and a more efficient enzymes' response; (Barry and Blow, 2016) 2) differently from PSG studies in which alcohol administration is controlled (as in de Zambotti et al. (de Zambotti et al., 2021)), here we are not aware of the precise number of drinks consumed right before bedtime; moreover, participants did not report excessive alcohol use, which may have limited the effects on subsequent sleep. Indeed, as reported by PSG studies, negative effects on sleep structure were mainly detected after pre-sleep heavy consumption (e.g., McCullar et al. (McCullar et al., 2024)); 3) The wrist is not the brain: despite some (but not all) controlled laboratory PSG findings indicating that pre-sleep alcohol consumption impacts the subsequent sleep, especially in the first part of the night, sleep estimate from wearables may not have a 1:1 correspondence with PSG-based sleep assessment. (Haghayegh et al., 2019; de Zambotti et al., 2019; Lim et al., 2023)

When looking at the relation between alcohol and sleep cardiac function, our data showed a positive association between nighttime alcohol use and increased NREM- and REM-HR during sleep. This evidence is consistent with polysomnographic studies that analyzed the impact of nocturnal alcohol on subsequent sleep HR, (Sagawa et al., 2011; de Zambotti et al., 2021) and denotes a possible dose-time-dependent effect of alcohol that future studies should further investigate. Although with lower incidence, even at young ages alcohol disruption of cardiac activity has been associated with several implications, such as cardio-metabolic dysfunction, (Du et al., 2017) macro- and microcirculation alterations, (Piano, 2017) and cardiac dysfunctional remodeling. (Rodrigues et al., 2018) Further studies should evaluate whether sleep cardiovascular restoration is long-term compromised in association with alcohol use, and particularly which specific factors associated with the pattern of alcohol and other substance use (e.g., frequency and amount of drinking, craving processes) are the main determinants of this altered sleep cardiovascular function.

In our study, other than alcohol, we further investigated the relationships between other substances (caffeine, nicotine, cannabis, other drugs), sleep, and sleep HR, but these results should be interpreted cautiously as we only considered whether participants used them over the day (yes/no) but not the specific amount of intake or the timing of use.

Along with alcohol, nicotine use was associated with delayed sleep onset and sleep period start time, in line with its sleep-detrimental stimulant effects. (Singh et al., 2023) Further studies should better disentangle the effects of substance intake from those of "late activity" on sleep timing, also considering the type of activity the participant engaged in the previous night, which we did not measure. Cannabis also showed a negative effect on sleep, by predicting increased WASO. Studies have shown mixed effects of cannabis on sleep. For example, while Drazdowski and colleagues found an association between cannabis consumption, reduced sleep efficiency, and daytime functioning (Drazdowski et al., 2021), Graupensperger and colleagues reported that the use of cannabis as a sleep aid was not associated with sleep issues or daytime dysfunctions in young adults. (Graupensperger et al., 2021) Other drug consumption was generally associated with a reduction of sleep duration, showing negative associations with sleep midpoint, sleep period time, and total sleep time. Although we did not specifically record the type of drug used, these findings depict a general negative effect of drugs on sleep. However, it should be considered that the frequency of other drug use was very low in our sample, and this might have biased the resulting relationships with sleep measures.

A few study limitations should be considered. Given the observational/correlational nature of the study, even though we hypothesized an association between prior substance use and subsequent sleep, we cannot draw a conclusion that these factors are causally linked. Other limitations concern the type of data collected from daily surveys. Specifically, we collected the number of alcoholic drinks consumed each day, which is an approximate measurement unit and does not consider the effective percentage of alcohol intake). Conversely, for other substances (caffeine, nicotine, cannabis, and other drugs) we only derived whether the participants used them or not for each day of data collection. In this regard, future studies conducted with wearable assessment should investigate the effects of other substances, possibly adopting an objective measure of their intake across the day. This could also be useful to evaluate further the hypothesized time-related effects of alcohol and substances on sleep. Future research should also better investigate the potential cumulative effects of consuming alcohol or other substances at multiple time windows (e.g., both afternoon and evening) vs. only once per day (e.g., afternoon only). Indeed, binge

drinking has been related to more negative consequences on sleep (Patrick et al., 2018). That said, our study relies on different strengths. Here we conducted a deeper evaluation by collecting data at high granularity with both passive-wearable (Fitbit) and daily survey measurements. This approach not only enables the calculation of average measures of sleep and HR but also to test the possible variations at the within-person level. Therefore, this type of methodological approach provides a more detailed analysis of the possible effects of substances in the short term and should be preferred in cases where it is necessary to conduct longitudinal investigations, not easily implemented in a laboratory setting.

5. Conclusions

As one of the first studies to analyze intensive longitudinal data from wearable devices and daily survey administration in young adults, we further demonstrated the importance of monitoring the effects on sleep of alcohol and substance consumption in this age group, as well as the benefits of using daily EMA. Indeed, our data showed that in young adults, substance consumption was negatively associated with delays in sleep timing and poorer sleep structure, with a specific effect of alcohol on sleep HR. Future studies should investigate the possible long-term consequences on sleep and cardiovascular health of alcohol and other substance consumption in young adults.

CRediT authorship contribution statement

Oreste De Rosa: Writing – review & editing, Writing – original draft, Visualization, Formal analysis. **Luca Menghini:** Writing – review & editing, Formal analysis, Data curation. **Erin Kerr:** Investigation, Data curation. **Eva Müller-Oehring:** Writing – review & editing, Supervision. **Kate Nooner:** Writing – review & editing, Supervision. **Brant P. Hasler:** Writing – review & editing, Supervision. **Peter L. Franzen:** Writing – review & editing, Supervision. **Duncan B. Clark:** Writing – review & editing, Supervision. **Sandra Brown:** Writing – review & editing, Supervision. **Susan F. Tapert:** Writing – review & editing, Supervision. **Kevin Cummins:** Writing – review & editing, Supervision. **Fiona C. Baker:** Writing – review & editing, Supervision, Methodology, Funding acquisition. **Massimiliano de Zambotti:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization.

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Financial arrangements

None.

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Data availability

The analysis is based on the data release NCANDA_RELEASE_BASE_REDCAP_MEASUREMENTS_V07 made accessible to the public according to the NCANDA Data Distribution [X]. X: National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA). Available online: https://www.niaaa.nih.gov/ national-consortium-alcohol-and-neurodevelopment-adolescence- ncanda (accessed on 7 July 2022). The data release was distributed according to the NCANDA Data Distribution agreement (https://www- niaaa-nih-gov.laneproxy.stanford.edu/ncanda-data-distribution- agreement), whose collection and distribution were supported by National Institutes of Health, USA funding [National Consortium on Alcohol and Neurodevelopment in Adolescence grant numbers: AA021697, AA021695, AA021692, AA021696, AA021681, AA021690, AA021691].

High-density aggregated measures of Fitbit and mNCANDA App data will be available upon reasonable request to the corresponding author.

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