INTRODUCTION

1.1 The relationship between sleep and caffeine

Insufficient sleep duration has become a growing health problem. According to the Centers for Disease Control and Prevention, more than one-third of American adults do not get enough sleep on a regular basis (Liu et al., 2014). Research has shown that there is an inverse relationship between caffeine consumption and sleep duration (e.g., Bonnar & Gradisar, 2015; Hicks, Hicks, Reyes, & Cheers, 1983; Hollingworth, 1912; etc.). Caffeine is also believed to prolong sleep latency, reduce sleep efficiency and worsen perceived sleep quality (Clark & Landolt, 2017), whereas caffeine abstinence has been demonstrated to be effective in improving sleep quality (Sin, Ho, & Chung, 2009). The caffeine and sleep relationship also has biologic bases in the brain. It is widely accepted that adenosine receptor agonists promote sleep and caffeine antagonizes adenosine receptors, thereby inhibiting adenosine's sleep-inducing effects (Clark & Landolt, 2017; Nehlig, Daval, & Debruyne, 1992). Thus, sleep hygiene advice on insomnia often suggests...
Although the relationship between caffeine and sleep has been intensively examined, a closer look at the literature indicated that most studies focused on the effect of caffeine on sleep (e.g., Kerpershoek, Antypa, & Van den Berg, 2018; Sanchez-Ortuno et al., 2005; Watson, Coates, Kohler, & Banks, 2016), not the inverse. Given the correlational nature of these studies, they actually cannot examine the direction of the relationship. On the other hand, experimental laboratory studies (e.g., Drake, Roehrs, Shambroom, & Roth, 2013; Hindmarch et al., 2000; Lloret-Linares et al., 2012; Shilo et al., 2002) are able to examine the causal effect of caffeine on sleep by recording participants’ sleep after administering certain amounts of caffeine, yet this effect may not be reflected in real life. People may not directly go to sleep after taking caffeine, and daily caffeine intake is often in the morning to prepare for the workday ahead (Lieberman, Agarwal, & Fulgoni, 2019). This pattern is especially prevalent in adults, whereas in adolescents (13–17 years) caffeine consumption is more evenly distributed throughout the day (Martyn, Lau, Richardson, & Roberts, 2018). It is questionable whether caffeine taken in the morning still has an effect in the evening (Youngberg, Karpov, Begley, Pollock, & Buysse, 2011). As mentioned earlier, adenosine increases the desire to sleep, and caffeine inactivates adenosine receptors. But about 5 to 7 hr after oral administration, our body will remove 50% of the caffeine concentration (Newton et al., 1981; Seng et al., 2009). Although the half-life of caffeine varies greatly from person to person (Seng et al., 2009), its effect usually does not last all day. Conversely, caffeine is often used to counteract sleepiness generated by sleep deprivation, jet lag and shift work (Carrier et al., 2009). Because a high proportion of the population uses caffeine to cope with evening work and jet lag, especially middle-aged adults (Centofanti et al., 2018), it is highly possible that the caffeine–sleep relationship may be bidirectional. Perhaps high daily intakes of caffeine impair sleep, and less sleep leads to more caffeine use to compensate for sleep loss or delayed sleep onset.

Portable devices such as actigraphs (Marino et al., 2013) allow researchers to monitor participants’ sleep in their home environment. Actigraphs have been widely used in sleep studies. For example, in a study by Hindmarch et al. (2000), 30 healthy volunteers (aged 19–36 years) received equal-volume drinks equivalent to either one or two cups of tea, coffee or water. Results indicated that increased levels of caffeine resulted in shorter sleep times as measured by actigraphs. Another study (Nova, Hernandez, Ptomey, & Zeitzer, 2012) found that estimations of integrated salivary caffeine concentration during sleep were correlated with wake after sleep onset (WASO), which was also measured by actigraphs. We can use actigraphs to continuously measure participants non-invasively in their natural environment, and the obtained intensive longitudinal data can help us examine the bidirectional relationship between sleep and caffeine. By tracking the same participant for several days, researchers can observe habitual sleep and caffeine intake simultaneously, analyse their dynamic relationship within a person to control for individual differences, and therefore overcome the limitations of non-experimental designs.

### 1.2 The role of age

Age is a known factor affecting sleep, caffeine intake and the relationship between the two. Research argues that as age increases, sleep quality worsens over time (Mander, Winer, & Walker, 2017). Caffeine intake increases with age and appears to be most prevalent in adults aged 50–64 years (Fray, Johnson, & Wang, 2005; Mitchell, Knight, Hockenberry, Teplinsky, & Hartman, 2014). Clark and Landolt (2017) found that the sleep of older adults may be more sensitive to caffeine compared to younger adults. Carrier et al. (2009) found that middle-aged subjects are particularly vulnerable to the circadian waking signal under the influence of caffeine; they showed greater decrements of sleep duration and sleep efficiency than young subjects. However, no study has compared the middle-age group with the elderly, and the impact of sleep on caffeine use has not yet been examined in different age groups.

### 1.3 Research questions and hypotheses

The effect of caffeine on sleep is well documented and has biological bases. However, mixed findings also exist. If we look closely at the literature, we can see that the laboratory studies that gave participants caffeine and then measured sleep often found a significant association between the two, whereas for surveys on habitual caffeine intake and sleep, there are many non-significant findings and methodological concerns (Chaudhary, Grandner, Jackson, & Chakravorty, 2016; James & Keane, 2007; Mniszek, 1988). For the inconsistency between findings from laboratories and surveys, it is possible that laboratory studies that instructed participants to take caffeine before bed may lack external validity, because in real life caffeine is often ingested in the morning and its effect may not last until bedtime. It is also possible that survey studies using a cross-sectional design have little control over individual differences and may also suffer from retrospective bias; therefore, the real effect may be masked by individual differences or measurement errors. Daily diary design helps overcome the limitations of both laboratory studies and cross-sectional surveys. This design allows researchers to observe participants on-site in their natural environment for a longer period of time, is able to capture and analyse within-person variations to fully control individual differences, and thus has the potential to establish Granger causality.

This study aimed to re-examine the relationship between habitual caffeine consumption and sleep duration using a daily diary design. Particularly, we examined the direction of the relationship and the role of age. Multiple statistical strategies were performed on the repeated daily measures, including the traditional static approaches and the cutting-edge dynamic approaches. By doing so, we wish to replicate findings in past cross-sectional
studies, as well as initiatively examine the within-person relationship between caffeine and sleep, and identify the direction of the relationship. The statistical strategies will be elaborated in the Methods section.

We tested the following two hypotheses regarding the direction of the relationship, each of which expects a static result and a dynamic result. H1: Caffeine intake influences sleep duration. Under this hypothesis, we predict that (a) today’s caffeine intake is negatively related to tomorrow’s sleep duration, and (b) a high level and/or an increasing rate of caffeine intake would drive people to decrease sleep duration in the future. H2: Sleep duration influences caffeine intake. Under this hypothesis, we predict that (a) tonight’s sleep duration is negatively related to tomorrow’s caffeine intake, and (b) a short and/or a decreasing rate of sleep duration would drive one to increase caffeine use in the future.

We also examined the role of age and tested four age-related hypotheses. According to the literature, we expected that (H3) as age increases, the average sleep duration decreases; and (H4) caffeine consumption should peak at the end of middle age, so we predicted that caffeine intake first increases with age and then decreases. For the relationship between caffeine and sleep, past research suggested that the negative impact of caffeine on sleep increases with age. However, this conclusion was based on the comparison between adults and adolescents; it is not clear whether this phenomenon can still be observed from early adulthood to later. This study compared the middle-aged adults with older adults, and we expected that (H5) sleep is more sensitive to caffeine among the elderly than the middle-aged. In the literature, little is known about whether the effects of sleep on caffeine consumption vary with age. Given that most older adults have retired, they may not need to stay alert for work, and we predicted that (H6) they are less likely to use caffeine to counteract sleep loss than middle-aged adults.

Participants were assessed/asked questions on their sleep quality, medical history and medications. According to the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), 167 participants were good sleepers (PSQI global score ≤ 5), 136 participants were poor sleepers (PSQI global score > 5) and 29 participants had missing values. There was no difference between the two age groups in their sleep quality, $M_{\text{middle}} = 6.42$ (standard deviation $SD = 3.78$), $M_{\text{old}} = 5.68$ ($SD = 3.11$), $t(301) = 1.867, p > .05$. The medical history question asked about 23 symptoms, including heart disease, high blood pressure, cancer, diabetes, alcoholism, depression, etc. A total of 156 participants reported no symptoms, 96 participants reported one diagnosed symptom and 80 participants reported more than one diagnosed symptom. The number of symptoms per capita did not differ between the two age groups, $M_{\text{middle}} = 0.93$ ($SD = 1.28$), $M_{\text{old}} = 0.91$ ($SD = 1.06$), $t(330) = 0.165, p > .05$. For the medicine data, 101 participants reported no use of medicine (72 in the middle-aged group and 29 in the elderly group) and 231 participants used at least one medicine. Because this study focused on sleep, we examined the diagnosis code for medicine and found that nine participants used prescription medicines and seven participants used over-the-counter medications for sleep/sleep aid. Among them, four participants used sleep/sleep aid medicines during the 7 days of diary study, so they were excluded from the data analysis. Given that a large number of participants were classified as poor sleepers or diagnosed with certain symptoms, we did not exclude participants based on their sleep quality or medical history, but controlled these conditions in the data analysis. The final sample for data analysis consisted of 328 participants (161 middle-aged and 167 elderly).

## 2 METHODS

### 2.1 Participants

The data used in the current study come from a large national database, the MIDUS Biomarker project (Ryff, Seeman, & Weinstein, 2013). Three different sites were used to collect these data, including the University of California at Los Angeles, University of Washington (UW) and Georgetown University. The whole dataset includes 1,255 participants. All data used in this study were collected at the second site, UW, because only the UW site collected the diary data. In total, 377 participants completed diary data during a 7-day period; 332 among them were coffee drinkers and were used in this analysis. Of the 332 participants, 138 were male and 194 were female. The average age was 56.78 years, with a standard deviation of 11.19, and ranged from 35 to 85. According to the average age, the whole sample was divided into two age groups: middle-aged adults (35–55 years, n = 163) and older adults (≥55 years, n = 169).

The sleep duration was calculated as the duration between the time participants fell asleep and the time they woke up. In this study, only night-time sleep was accounted for in sleep duration; day time naps were not counted. Sleep duration was measured by Actigraphy and daily sleep diary for seven consecutive days. Actigraphy is a non-invasive method of monitoring rest/activity cycles, and it can be used to roughly estimate sleep duration, sleep latency, waking during sleep and some other sleep parameters. In this study, participants wore an actigraph device, Actiwatch®, on their non-dominant wrist to actively measure sleep. Participants also self-reported bedtime and wake-up time in the daily sleep diary. They were asked “What time did you go to bed and begin trying to sleep?” and “What time did you wake up for the day and not return to sleep?” upon awakening. Their answers were used to help define the rest intervals in the actigraphy data. Incomplete/inconsistent data between device and diary

2.2 | Measures

### 2.2.1 Sleep duration

The sleep duration was calculated as the duration between the time participants fell asleep and the time they woke up. In this study, only night-time sleep was accounted for in sleep duration; day-time naps were not counted. Sleep duration was measured by Actigraphy and daily sleep diary for seven consecutive days. Actigraphy is a non-invasive method of monitoring rest/activity cycles, and it can be used to roughly estimate sleep duration, sleep latency, waking during sleep and some other sleep parameters. In this study, participants wore an actigraph device, Actiwatch®, on their non-dominant wrist to actively measure sleep. Participants also self-reported bedtime and wake-up time in the daily sleep diary. They were asked “What time did you go to bed and begin trying to sleep?” and “What time did you wake up for the day and not return to sleep?” upon awakening. Their answers were used to help define the rest intervals in the actigraphy data. Incomplete/inconsistent data between device and diary
were reviewed, adjusted or deleted. Only complete and clean data were used in the data analysis.

2.2.2 Caffeine intake

Caffeine use was measured daily by asking participants how many caffeinated drinks they had on that day. One cup of coffee or tea (8-ounce) or one can of fizzy drink (12-ounce) counts as one drink. One drink contains about 60–140 mg caffeine, depending on the type of drinks. In this study, because there were no data on the type of caffeinated drinks, we used the number of cups to represent the amount of caffeine consumed per day. Although the caffeine intake data were self-reported, there is research suggesting that the caffeine questionnaire was able to accurately predict salivary concentrations of caffeine (Nova et al., 2012). Among the 377 participants who completed the daily diary study, 45 participants reported no use of caffeine drinks at all, so they were removed from the data analysis. Among the 332 caffeine users, 13 participants consistently reported the same number of drinks across the 7 days, and 319 participants showed variations across days.

2.3 Data analysis strategies

There are several approaches to analyse longitudinal data. One strategy is to simply aggregate data within person, and then examine the correlation between the aggregated variables. This method is straightforward, but completely ignores intra-individual variations that are more meaningful for establishing causal relationships and providing intervention advice.

Multilevel modelling and growth curve modelling are also commonly used methods for longitudinal data analysis. The growth curve method is suitable for modelling development and substantive change over time. However, daily regulatory behaviours, such as sleep or caffeine consumption, often do not include such long-term growth. In comparison, multilevel models (also known as hierarchical linear models and mixed-effect models) focusing on controlling the shared residual in the same person are suitable for short-term fluctuations. In such a model, daily observations (Level 1) are nested in person (Level 2), and the association between the daily measures can be tested. The model used in this study is specified as Equation 1. To examine the role of age, Equation 1 was also tested within the middle-aged group and within the older group.

\[ \text{Sleep} = \beta_0 + \beta_1 \text{Sleep} + \beta_2 \text{Sleep} + \beta_3 \text{Caffeine} + \beta_4 \text{Caffeine} \]

\[ \text{To test H2: Level 1: Caffeine} = \beta_0 + \beta_1 \text{Sleep} + \beta_2 \text{Sleep} + \beta_3 \text{Caffeine} + \beta_4 \text{Caffeine} \]

3 RESULTS

3.1 The average level of caffeine intake and sleep duration

3.1.1 Aggregation across people

The average sleep duration and caffeine intake were calculated for each day of the week across people. As shown in Table 1 and Figure 1, the highest sleep duration happened on Friday night, with an average 6.370 hr, and the lowest on Wednesday night, with an average 6.145 hr. The highest caffeine intake happened on Friday, with an average of 2.418 cups per day per capita, and lowest on Saturday, with an average of 2.368 cups per day per capita. But please note that when individual differences are averaged out,
the differences between days are quite small, which is to be expected, and there is no overall trend showing differences between weekdays and weekends, nor sign of association between the two processes in Figure 1.

### 3.1.2 Aggregation across days

The average sleep duration and average caffeine intake were calculated for each participant. Correlation analysis indicated that the average sleep duration and the average caffeine intake were not significantly correlated, $r = -0.067$, $p > .05$, which provided no support for H1 and H2. The two age groups did not differ significantly in their average sleep duration ($M_{old} = 6.25$ hr, $SD_{old} = 1.06$; $M_{middle} = 6.19$ hr, $SD_{middle} = 1.07$), $t(326) = -0.484$, $p > .05$, which did not support H3. They also did not differ significantly in their average caffeine intake ($M_{old} = 2.35$ cups, $SD_{old} = 1.67$; $M_{middle} = 2.44$ cups, $SD_{middle} = 1.97$), $t(326) = 0.465$, $p > .05$. However, within the middle-aged group, caffeine intakes significantly increased with age, $b = 0.058$, SE = 0.029, $p = .048$, whereas within the older group, caffeine intakes significantly decreased with age, $b = -0.037$, SE = 0.016, $p = .021$. These results supported H4 and suggested that the average caffeine intakes first increased and then decreased with age.

### 3.2 The static association between caffeine intake and sleep duration

Multilevel regression was used to analyse the effect of today’s caffeine intake on tonight’s sleep (H1a) and the effect of tonight’s sleep on the next day’s caffeine intake (H2a). We used the lmer function of the lme4 packages (Bates, Maechler, Bolker, & Walker, 2015) in R. lmer allows controlling for the variance associated with random factors without data aggregation. Participants were specified as a random factor to control for their associated intra-class correlation. In this study, we compared three models: Model 1 allowed random intercept and random slope; Model 2 allowed random intercept only; and Model 3 was a regular linear regression where all participants had the same intercept and slope.

Regression coefficients are shown in Table 2. In the caffeine predicting sleep model, the variance of intercept across people was estimated as 1.033, the variance of the slope was estimated as 0.0002, and the variance of residuals was estimated as 1.213, 1.017, 2.167.

### TABLE 2 The static multilevel regression coefficients ($n = 328$)

<table>
<thead>
<tr>
<th>IV → DV</th>
<th>Model</th>
<th>Fixed effect</th>
<th>Random effect</th>
<th>Residual</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$b$</td>
<td>SE</td>
<td>$p$</td>
</tr>
<tr>
<td>Caffeine → Sleep</td>
<td>M1</td>
<td>-0.004</td>
<td>0.019</td>
<td>.848</td>
</tr>
<tr>
<td></td>
<td>M2</td>
<td>0.001</td>
<td>0.020</td>
<td>.961</td>
</tr>
<tr>
<td></td>
<td>M3</td>
<td>-0.026</td>
<td>0.015</td>
<td>.082</td>
</tr>
<tr>
<td>Sleep → Caffeine</td>
<td>M1</td>
<td>0.007</td>
<td>0.032</td>
<td>.820</td>
</tr>
<tr>
<td></td>
<td>M2</td>
<td>-0.014</td>
<td>0.022</td>
<td>.507</td>
</tr>
<tr>
<td></td>
<td>M3</td>
<td>-0.076</td>
<td>0.031</td>
<td>.014</td>
</tr>
</tbody>
</table>

Note: Caffeine’ stands for the caffeine intake on the next day. M1 indicates a random intercept and random slope model, M2 indicates a random intercept model, and M3 indicates a regular linear regression model with no random effect. The final models are highlighted in grey. DV, dependent variable; IV, independent variable; SE, standard error.
suggesting that there is no need to allow random slope. In the random intercept only model (M2), the effect of caffeine on sleep was not significant, $b = 0.001$, $SE = 0.020$, $p > .05$. Therefore, H1a was not supported. In the sleep predicting caffeine model, the variance of intercept across subjects was estimated as 9.843, the variance of the slope was estimated as 0.157, and the variance of residuals was estimated as 0.798. These results suggested the inclusion of a random intercept but not random slope. In the random intercept only model (M2), the effect of sleep on caffeine was not significant, $b = -0.014$, $SE = 0.022$, $p > .05$. Therefore, H2a was not supported. The multilevel regression analysis within each age group also did not reveal any significant effect, $p > .05$. We also controlled medical diseases (the number of symptoms from medical history) and sleep problems (PSQI global score) in the random intercept models; neither the predictors nor the covariates had any significant effect, $p > .05$.

### 3.3 The dynamic association between caffeine intake and sleep duration

The first derivatives (change rates) and the second derivatives (accelerations) of the two processes on each day were calculated using generalized local linear approximation (GLLA, Boker, Deboeck, Edler, & Keel, 2010). To test H1b, the acceleration of sleep duration was predicted by the current sleep level, sleep change rate, current caffeine level and caffeine change rate (Equation 2, first line). Similarly, to test H2b, the acceleration of caffeine intake was predicted by the current caffeine intake, caffeine change rate, current sleep duration and current sleep change rate (Equation 2, second line).

Table 3 shows the regression coefficients in the multilevel regressions on the above-described momentums with random intercept. There were significant self-regulating effects on sleep duration and caffeine intake. For example, the current level ($b = -0.659$, $SE = 0.015$, $p < .001$) and change rate ($b = -1.073$, $SE = 0.005$, $p < .001$) of sleep significantly predicted the future state of sleep. This self-regulating effect suggests that when people are away from their normal sleep time, there will be a strong force driving them back. For the coupling effect between caffeine and sleep, according to the results using the overall sample, caffeine level and change rate did not significantly predict the change tendency of sleep; therefore H1b was not supported. However, the current sleep level significantly predicted the change tendency of caffeine intake, $b = -0.026$, $SE = 0.012$, $p < .05$. A shorter sleep duration predicted a stronger tendency to increase caffeine consumption. H2b was supported. Then the same dynamic model was run within each age group, and we found that a short and/or reduced sleep duration significantly predicted more future caffeine use in the middle-aged group ($b = -0.040$, $SE = 0.018$, $p < .05$, and $b = -0.026$, $SE = 0.009$, $p < .01$) but not in the senior group ($b = -0.005$, $SE = 0.016$, $p > .05$, and $b = 0.014$, $SE = 0.009$, $p > .05$). For the middle-aged group, when the sleep duration was short, or shorter than before, people tended to increase caffeine consumption, and these effects remained or became even larger after controlling disease and sleep problems, $b = -0.046$, $SE = 0.019$, $p < .05$, and $b = -0.027$, $SE = 0.009$, $p < .01$.

### 4 DISCUSSION

#### 4.1 Main findings

This study examined the dynamic relationship between daily caffeine intake and sleep duration in middle-aged and older adults. Although caffeine is often considered as an obstructive factor for good sleep (Clark & Landolt, 2017), we did not find this association in this national dataset. There may be two possible reasons for this result. First, people may have become used to the amount of caffeine they regularly take and this habitual caffeine use does not affect their sleep duration (Dager et al., 1999; Hindmarch et al., 2000; Lovallo et al., 2005). Another possible explanation is that the timing of caffeine use may also influence its effect on sleep (Bonnet & Arand, 1992; Lieberman et al., 2019). As mentioned before, adults tend to drink coffee in the morning and youths tend to drink throughout the day (Martyn et al., 2018). The participants in this study were all adults, and if most of them ingest caffeine in the morning, its effect on night sleep would

<table>
<thead>
<tr>
<th>DV</th>
<th>IV</th>
<th>Overall sample</th>
<th>Middle-aged</th>
<th>Older adults</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$b$</td>
<td>$SE$</td>
<td>$b$</td>
</tr>
<tr>
<td>Sleep</td>
<td>Sleep</td>
<td>-0.659***</td>
<td>0.015</td>
<td>-0.667***</td>
</tr>
<tr>
<td></td>
<td>Sleep</td>
<td>-1.073***</td>
<td>0.005</td>
<td>-1.077***</td>
</tr>
<tr>
<td></td>
<td>Caffeine</td>
<td>0.005</td>
<td>0.014</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>Caffeine</td>
<td>0.002</td>
<td>0.007</td>
<td>-0.006</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Caffeine</td>
<td>-0.096***</td>
<td>0.008</td>
<td>-0.073***</td>
</tr>
<tr>
<td></td>
<td>Caffeine</td>
<td>-0.914***</td>
<td>0.008</td>
<td>-0.885***</td>
</tr>
<tr>
<td></td>
<td>Sleep</td>
<td>-0.026*</td>
<td>0.012</td>
<td>-0.040*</td>
</tr>
<tr>
<td></td>
<td>Sleep</td>
<td>-0.008</td>
<td>0.006</td>
<td>-0.026**</td>
</tr>
</tbody>
</table>

*p < .05.

**p < .01.

***p < .001.
be trivial. This study also tested the other direction of the relationship. In comparison with the abundant literature on the effect of caffeine on sleep, there is less research on the effect of sleep on caffeine intake. We found that lack of sleep was associated with more caffeine consumption in the future, which is consistent with past literature (Carrier et al., 2009; Roehrs & Roth, 2008; Watson et al., 2016), and added to the literature by finding that this phenomenon was only true among middle-aged people, not the elderly.

4.2 Method comparison

This study used three methods to analyse the repeated measures: data aggregation, multilevel linear regression and dynamical system analysis. Data aggregation can investigate individual differences on average scores, but the within-person variation is completely ignored. Multilevel linear regression, a common practice in longitudinal data analysis, helps determine the within-person synchronous correlation between two processes and allows personalized correlation/regression coefficients, yet it is still a static method. A known problem of static methods is a phase problem. There is often a time lag between the cause and the consequences, and the interval time is often uncertain and varies from person to person, so even if there is a causal effect, it cannot be detected in a static model with a zero or fixed time lag. In comparison, if we can estimate the tendency of the behaviour, even if the effect may not show up immediately, we should still be able to observe a change in acceleration. So, including momentums in the model may help solve the phase problem and detect associations that do not immediately happen after the leading process.

4.3 Limitations and future directions

Several limitations of this study should be addressed. In the data we used, the timing of caffeine consumption was not measured. Ludden and Wolfson (2010) investigated adolescents’ caffeine use and found that in their high-school student sample, 95% of participants reported recent caffeine use, and the typical time of use was in the evening. Our sample included middle-aged and senior people and they may consume caffeine at different times of the day. In the future, researchers can ask participants when they consume caffeinated products to clarify whether timing is the reason for the non-significant effect of caffeine intake on sleep. Second, individual traits were not included in this study. Previous research found that estimations of integrated salivary caffeine concentration during sleep were correlated with wake after sleep onset (WASO) most strongly in morning-type individuals (Nova et al., 2012). Further research can ask whether participants are morning-type people or night-type people or mixed. Moreover, this study did not investigate different types of caffeine drinks. Cluster analysis on adolescents (Ludden & Wolfson, 2010) found three groups of caffeine users: “low caffeine use”, “high soda use” and “mixed use”. Compared to the high-soda-use group, the mixed-use group reported more reasons for caffeine use, related to recovering from daytime sleepiness, getting energy and drinking more coffee. Future studies can ask participants what they drink and why they drink it. Considering caffeinated beverages have become popular in youth culture (e.g., energy drinks, coffee), gaining more insight into the differences in motives for caffeine use across the lifespan would help researchers understand why people in different age groups consume a certain type of caffeine and how that type of caffeinated drink influences sleep. In sum, more details on caffeine use may help us understand the coupled dynamics better.

CONFLICT OF INTEREST

No conflicts of interest declared.

AUTHOR CONTRIBUTIONS

YH contributed to the conception of the work, data analysis, result interpretation, manuscript drafting and critical revision. KS contributed to literature review, data analysis and manuscript drafting. DK contributed to literature review and critical revision. All authors approved the final version to be published.

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