



Original article

Caffeine, Daytime Sleepiness, and Conduct Problems Among Early Adolescents: A Longitudinal Analysis

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A B S T R A C T

Purpose: Recent research suggests that caffeine use may promote a range of adjustment difficulties among adolescents, particularly during the middle school years. The effects of caffeine are particularly concerning given the increased use of high-dosage caffeine products, such as energy drinks, among youth. We investigated the influence of caffeine use on trajectories of conduct problems among early adolescents. Daytime sleepiness was tested as mediator of caffeine's effect.

Methods: Hypotheses were tested with longitudinal (4 waves) data from 2,633 middle school students beginning in the Fall of sixth grade and concluding in the spring of seventh grade. All students in a single class cohort at 20 West Virginia middle schools were invited to participate.

Results: Controlling for family affluence and management practices and pubertal development, use of more than 100 mg of caffeine significantly predicted linear increases in conduct problems over time (Est. = 0.23, $p = .015$). Increases in daytime sleepiness partially mediated this effect (Est. = 0.22, $p = .002$).

Discussion: Caffeine use and daytime sleepiness are important vulnerability factors for the emergence of conduct problems.

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IMPLICATIONS AND
CONTRIBUTION

Recent evidence suggests that caffeine use affects adjustment among young adolescents. Little prospective research, however, investigates the effects of caffeine on conduct problem trajectories or mechanisms that may explain the effects of caffeine. Study results underscore the potential harms of caffeine use in early adolescence and demonstrate that both caffeine use and daytime sleepiness are vulnerability factors for conduct problems.

Caffeine is the most widely used psychoactive substance by children and adolescents in the United States [1]. Classified as a psychostimulant, caffeine produces psychomotor activating, reinforcing, and arousing effects, which are a product of the disinhibition of the brake that endogenous adenosine imposes on dopamine and arousal systems [2]. Regular caffeine

consumption leads to physical dependence, evidenced by abstinence-induced withdrawal symptoms including headache, fatigue, difficulty concentrating, mood disturbances, and flu-like symptoms [3]. Withdrawal symptoms typically occur 12–24 hours after abstinence and symptoms may persist for 2–9 days [4]. Notably, withdrawal symptoms may occur following abstinence from as little as 100 mg of daily caffeine intake, roughly the amount in a typical cup of filter coffee, a single espresso, or an 8oz energy drink. Dietary habits change significantly in adolescence (e.g., fewer home meals, less fruits and vegetables, higher consumption of fast foods). Whereas intake

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levels appear not to have changed appreciably over time, marked changes in consumption patterns have been observed with increased use of energy drinks [5].

Children and adolescents are regarded as at-risk populations for adverse effects from excessive caffeine intake [6]. There are several reasons for this, including their smaller body size and lack of tolerance to the effects of caffeine due to less consistent patterns of consumption. No universal guidelines for caffeine intake for children and adolescents exist. Health Canada [7] and the European Food Safety Authority [8] recommend daily intake of no more than 2.5 and 3.0 mg per kg of body weight, respectively; approximately 100–120 mg per day for 12-year-olds and 155–186 mg per day for 18-year-olds. Most children and adolescents consume caffeine regularly; the majority do not exceed the aforementioned recommended maxima [5]. The American Academy of Pediatrics (AAP) [9] and the American Academy of Child and Adolescent Psychiatry (AACAP) [10] advise zero caffeine consumption for children under 12 years and no more than 100 mg per day for adolescents aged 12–18 years.

The influence of caffeine on mood and behavior is a particular concern for children during the transition to adolescence (ages 10–12 years). Rapid development in physiological and neuro-cognitive systems compounded by changing social roles and a challenging school transition confers increased vulnerability to the effects of substances [11]. Caffeine use has been linked cross-sectionally to aggressive behavior [12–14], self-reported violence [15,16], and conduct disorder [15]. Prospective evidence that considers multiple confounding variables, however, is limited. Caffeine use may be a symptom of an underlying externalizing syndrome rather than a driver of problem behavior. Existing longitudinal data are limited to a single study of mid-adolescent youth that found that caffeine use predicted increases in aggressive behavior at a 1-year follow-up [16]. Moreover, potential mechanisms linking caffeine use to increases in conduct problems remain to be examined.

The present study investigates the role of daytime sleepiness as a mechanism linking caffeine use to conduct problems. Daytime sleepiness is a prevalent vulnerability mechanism with empirical links to caffeine and conduct problems [13]. During puberty, children experience significant changes to sleep patterns, including a tendency for later bedtimes and wake times, shorter overall sleep duration, daytime sleepiness, and a decrease in delta, nonrapid eye movement sleep [17]. These alterations have been linked to changing patterns and volumes of secretion in melatonin, reproductive hormones, and orexin. Pubertal development increases youths ability and interest in staying up late and may result in an “evening chronotype,” a pattern of delayed sleep and waking [17]. Combined with psychosocial factors such as early school start times and increased use of technology at night, many children experience short and mistimed sleep. To counteract the resulting daytime sleepiness, young adolescents often consume caffeine, which tends to exaggerate sleep problems by disturbing sleep and circadian timing [18]. Because children and adolescents tend to have less routine patterns of caffeine consumption than adults do [19], they may be at increased risk of experiencing withdrawal-induced daytime sleepiness [20]. Daytime sleepiness is a confirmed effect of caffeine withdrawal even after periods of abstinence as brief as several hours [4].

Empirical data implicate sleep-related problems in the etiology of conduct problems [18–20]. Daytime sleepiness, in particular, increases risk factors for conduct problems, including

learning difficulties, low school achievement, absenteeism, and decreased involvement in extracurricular activities [21–23]. Daytime sleepiness affects emotional regulation and cognitive control, which may exacerbate impulsivity and aggression [24]. Pathways linking caffeine use to conduct problems via effects on daytime sleepiness, however, remain to be investigated.

The present study

We examined the prospective influence of caffeine use on daytime sleepiness and conduct problems among a large sample of middle school students in West Virginia (WV). Beginning in the first semester of sixth grade, children provided 4 waves of data at 6-month intervals. We used growth curve modeling to test the hypothesis that baseline caffeine use will forecast growth in conduct problems and that growth in daytime sleepiness will act as a mechanism linking baseline caffeine use to growth in conduct problems. We considered as covariates in the model a number of potential confounders with links to caffeine use and conduct problems including sex/gender, family affluence, maternal education, and family management practices. We also included baseline pubertal status and the rate of increase in pubertal development to account for physical maturation in specifying the independent influence of caffeine.

Methods

Sample

Hypotheses were tested with data from the Young Mountaineer Health Study [25]. A single cohort of students enrolled in 20 geographically diverse public middle schools in 5 counties in WV participated in a school-based survey. Students provided data twice per year in grades 6 and 7. The baseline assessment (Time 1 [T1]) occurred during the height of the COVID pandemic, from October to December 2020. Students ($n = 1,671$) who attended school in face-to-face or hybrid (part in-person, part virtual) formats provided T1 data. This excludes participants who attended a state run virtual-only school format. Of the students attending some amount of in-person school at T1, 1,349 (80.7%) completed the study survey (mean age: 11.5 years). Three follow-up surveys were conducted at ~6-month intervals (T2, $N = 1,649$; T3, $N = 1,909$, T4, $N = 1,698$). The total sample for this analysis includes 2,633 unduplicated responses.

Procedures

Students' caregivers received an introductory letter/email prior to data collection and were given an opportunity to “opt out” their children from the study, which was sponsored by the participating school districts. Students responded to a confidential, computer-based survey during the school day using Qualtrics software. Students were accessed either inside schools or as part of designated classroom hours at home with their camera turned on, depending on accessibility during COVID-19 mitigation efforts. The first screen of the survey presented included a consent description and explained that continuing the survey indicated consent to participate. Data collection was supervised by research staff. Students provided their school ID number on their surveys. This ID was recorded by an honest broker within the school system. Only then was the deidentified

data provided to the University-based research team. The institutional review board of WV University approved all study protocols (#1903499093A001).

Measures

Conduct problems. At T1–T4, students completed the 6-item Oregon Adolescent Depression Project Conduct Disorder Screener [26]. Students reported their behavior in the past 6 months on a scale ranging from 0 (*never*) to 4 (*most of the time or all of the time*). Example items include, “I broke rules at home,” “I broke rules at school,” and “I got into fights.” Items were summed to indicate increased levels of symptoms. Cronbach’s alphas exceeded .73 across waves.

Caffeine consumption. Youth completed a widely used [15,27], 5-item caffeine measure. Respondents were asked, “How many cups/glasses/cans/or bottles do you usually drink every day of the following drinks?” followed by 4 items: coffee, tea, caffeinated soda (e.g., Cola drinks, Mountain Dew, Dr. Pepper), and energy drinks that contain caffeine (e.g., Red Bull, Monster, Rockstar, Bolt, etc.). Response options ranged from 1 (*none*) to 7 (*6 or more glasses/cups/cans/bottles*). Participants were also asked: “How many caffeine ‘shots’ (e.g., 5-Hour Energy) do you usually have each day?” and responded on the same scale as the previous items. Each type of caffeinated substance was then weighted by caffeine content (coffee [6x], tea [3x], soda/pop [1x], energy drinks [3x], and caffeine shots [12x]) to create an estimate of milligrams of intake per day. Per recommendations discussed above (7, 8) caffeine scores were coded to indicate no daily use (0 mg), moderate use (1–99 mg), or high use (≥ 100 mg). The high use threshold, 100+mg, represents the approximate caffeine content in an average cup of coffee.

Daytime sleepiness. Daytime sleepiness was assessed at each wave with a 4-item scale recommended by the Pediatric Sleep Disturbance and Sleep-Related Impairment [28] item bank. Students reported behavior for the past week on a scale ranging from 0 (*never*) to 4 (*almost always*). Example items included, “I had a hard time getting things done because I was sleepy” and “I had problems during the day because of poor sleep.” Items were summed to indicate increased daytime sleepiness. Cronbach’s alphas exceeded 0.84 across waves.

Covariates. Students completed the 4-item family management scale from the Communities that Care survey [29]. Students described their parents’ behavior on a scale ranging from 0 (*never*) to 3 (*always*). The items included: “My parents/caregivers ask if I’ve gotten my homework done;” “My parents/caregivers would know if I did not come home on time;” and “The rules in my family are clear.” Items were summed to indicate healthy family management practices. Cronbach’s alpha at T1 was .70. Participants completed the 5-item Pubertal Development Scale [30]. Items were summed to indicate more advanced pubertal development. Cronbach’s alphas exceeded .80 for boys and 0.76 for girls across waves.

Demographics. Students completed the 6-item family affluence scale developed for the Health Behaviour in School-aged Children study [31]. Response scales varied. Example items included, “Does your family own a car, van or truck?” and “Do you have your own bedroom for yourself?” Item responses were

standardized and summed to indicate increased affluence. Participant sex and/or gender was assessed with the following question: “How do you describe your gender?”: “Boy”, “Girl”, “Gender Nonconforming”, “Other (Please specify)”. Due to low numbers and puberty only being calculated on those who reported “Boy” or “Girl”, the latter 2 categories were excluded from analysis. A sex/gender variable was coded as 1 = “Male”, and 0 = “Female.” Based on responses to the question, “Who lives in your household?” family structure was dichotomized as “Lives with both biological parents” and “Different arrangements.” The vast majority of students reported being White/non-Hispanic. Race/ethnicity was dichotomized as “White/non-Hispanic” and “Other.” Maternal education was dichotomized as 1 = “College degree or higher”, and 0 = “Less than college degree”. For all demographics, the first available value was used.

Statistical analysis

Data were analyzed using Latent Growth Curve Modeling (LGCM) in Mplus 8.1. This method mean trajectories in an outcomes as a latent process (see Figure 1). In LGCM, the intercept represents the starting point with all factor loadings fixed to one, indicating that the intercept is constant across time. The slope represents the rate of change over time. Factor loadings are set to reflect the time structure in months. Given the strong psychometric properties of the manifest variables and model parsimony, we elected not to represent the variables as each wave as latent constructs. Prior to modeling growth curves, key analytical assumptions were checked (i.e., linear growth patterns, correlated error patterns, multicollinearity among predictors) and found to be satisfactory for the proposed analyses and study questions. The model was estimated with the full information maximum likelihood estimator, which utilizes all data; no cases are dropped due to attrition. To use all observations, mean estimation (for covariates) was used in conjunction with full information maximum likelihood (for outcomes) to recover missing information. A final model was selected based on parsimony and model fit criteria per the Root Mean Square Error of Approximation (RMSEA; closer to 0 and a 90% confidence interval less than 0.08 preferred), Comparative Fit Index (CFI; closer to 1 preferred), and Tucker-Lewis Index (TLI; closer to 1 preferred).

In the final model, we estimated LGCMs for pubertal development, daytime sleepiness, and conduct disorder. Study covariates (intercept pubertal development, gender, maternal education, family management, and family affluence) were allowed to correlate with each other and with caffeine use. Nonsignificant covariates were trimmed from the final model. Caffeine use was stable over time in the sample suggesting that regressing the mediator and outcome on baseline caffeine was

Specification for latent growth curve models

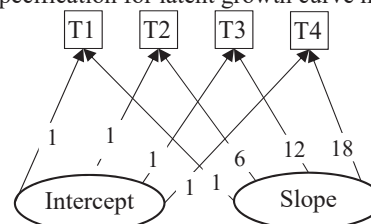


Figure 1. Specification for latent growth curve models.

appropriate. We present unstandardized estimates (labeled “Est.”) for regression parameters except for the growth estimates, which are presented as standardized estimates. Indirect effects were estimated via bootstrapping for paths from high and some caffeine to the slope of conduct disorder.

Results

Descriptive statistics

Table 1 presents descriptive statistics for study variables. Approximately, 50% of participants described their sex/gender as “boy.” Non-Hispanic White participants comprised 86% of the sample. Approximately 26% of participants reported their mother’s education as completing college education or higher. Average family affluence was 9.24 (range 0–13) and family management was 13.54 (range 4–16). Approximately 13% of participants reported consuming no caffeine, 25% reported using some caffeine (under 100 mg per day), and over 60% of the students reported using 100 mg or more of caffeine per day. Average daytime sleepiness increased over time ($M = 14.95$ at T1, $M = 15.92$ at T4), as did conduct disorder ($M = 8.45$ to $M = 9.18$) and pubertal development ($M = 2.13$ to $M = 2.65$).

Preliminary analyses

Unconditional linear growth curve models were specified for pubertal development, conduct problems, and daytime sleepiness. For pubertal development, model fit was $\chi^2(5) = 42.95$, $p < .001$; RMSEA = 0.65 (0.05, 0.08); CFI = .98, TLI = .98. Standardized estimates reflecting pubertal growth from T1 to T4 were .00, .20, .41, and .62. Model fit for conduct problems was $\chi^2(5) = 4.97$, $p = .42$; RMSEA = .00 (0.00, 0.03); CFI = 1.00, TLI = 1.00; standardized estimates for linear growth were .00, 0.25, .43, and .59. The fit for daytime sleepiness was $\chi^2(5) = 4.15$, $p = .53$; RMSEA = .00 (0.00, 0.03); CFI = 1.00, TLI = 1.00. Standardized estimates for linear growth were .00, .25, .47, and 0.67.

Final model

Figure 2 presents the test of study hypotheses. The model fit the data well: $\chi^2(133) = 519.67$, $p < .001$; RMSEA = .04 (.03, .04); CFI = .94, TLI = .92. After controlling for significant covariates (i.e., sex/gender, family affluence, family management, maternal education, pubertal development), ≥ 100 mg of caffeine versus no use was significantly associated with growth in daytime sleepiness (Est. = 0.38, $p = .001$) and growth in conduct problems (Est. = 0.22, $p = .015$). The indirect effect of 100 mg of caffeine relative to no caffeine use on growth in conduct problems via growth in daytime sleepiness was also significant (Est. = 0.22, $p = .002$). Using some (less than 100 mg) of caffeine was not associated with growth in daytime sleepiness or conduct problems relative to no caffeine use.

Covariates. The intercept of pubertal development (Est. = 0.17, $p = .012$), family affluence (Est. = -0.24 , $p < .0001$), family management (Est. = -0.34 , $p < .0001$), and sex/gender (Est. = -1.18 , $p < .0001$) were associated with the intercept of daytime sleepiness. The intercept of pubertal development (Est. = 0.11, $p = .015$), family affluence (Est. = -0.10 , $p < .0001$), family management (Est. = -0.27 , $p < .0001$), and sex/gender (Est. = .077, $p < .0001$) were associated with the intercept of conduct problems. Maternal education (Est. = -0.15 , $p = .21$) was marginally associated with the intercept of conduct problems. The intercept and slope of pubertal development was associated with growth in daytime sleepiness (Est. = 0.37, $p < .001$; Est. = 1.60, $p = .015$) but not growth in conduct problems.

We ran sensitivity analyses to determine if other definitions of caffeine intake, such as maximum daily caffeine and a decile variable, were similarly associated with the outcomes. We compared Akaike Information Criterion (AIC; smaller is better) values to determine whether other definitions of caffeine improved model fit. Fit was best for modeling caffeine as the previously reported three-level caffeine group (AIC: 106,075.81) compared to maximum daily caffeine (AIC: 135,047.34) or the decile variable (AIC: 112,271.60). Primary associations with caffeine use were similar across the models.

Table 1
Descriptive statistics

Variable	n	Mean or n	SD or valid percentage	Minimum	Maximum
Daytime sleepiness					
T1	1,263	14.95	4.81	6	30
T2	1,617	15.37	4.88	6	30
T3	1,859	15.63	5.10	6	30
T4	1,637	15.92	5.30	6	30
Conduct problems					
T1	1,293	8.45	2.93	6	30
T2	1,604	8.75	3.36	6	30
T3	1,815	8.88	3.80	6	30
T4	1,603	9.18	4.14	6	30
Family affluence					
T1	2,442	9.24	2.40	0	13
Family management					
T1	2,412	13.54	2.92	4	16
Caffeine (T1)	2,431				
None		306	12.59%		
Moderate (1–99 mg)		602	24.76%		
High (100+ mg)		1,523	62.65%		
Sex/gender	2,633				
Boy		1,193	50.02%		
Girl		1,192	49.98%		

SD = standard deviation.

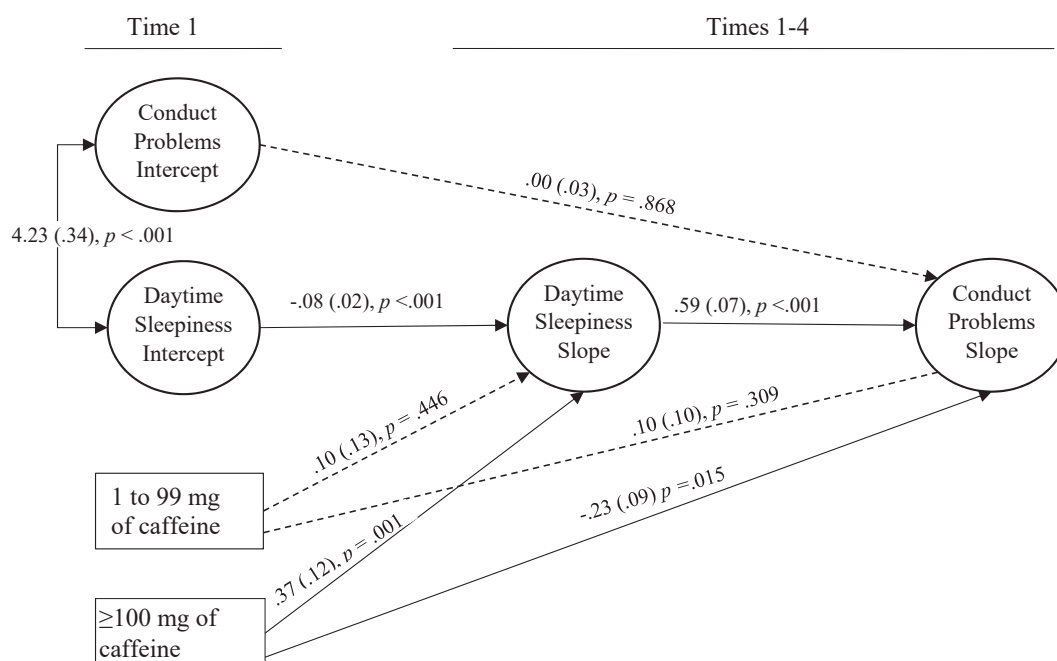


Figure 2. Structural equation model path analysis depicting indirect effects of caffeine use on conduct problems via daytime sleepiness among young adolescents. Shown is a path analysis from structural equation modeling. The independent variables are presented as rectangles representing 2 dummy variables. Ovals represent latent growth variables. The intercept (I) represents the average starting point for the cohort, and the slope (S) represents the average increase in the variable from T1 to T4. Nonsignificant paths are represented by a dotted line. Parameters shown are unstandardized beta coefficients with their standard error in parentheses.

Discussion

Accumulating evidence documents the independent negative effect of caffeine on adolescents' health and engagement in risk behavior. In the present study, we extended current findings in the following 2 key ways: (1) by focusing specifically on caffeine-related increases in conduct problems; and (2) testing daytime sleepiness as a mechanism explaining the effect. We analyzed 4 waves of survey data from a sample of middle-school students and implemented random coefficients models that specify caffeine effects on variability in individual trajectories of daytime sleepiness and conduct problems in our sample. Our analyses revealed a positive association between daily caffeine consumption of 100+ mg and growth in conduct problems from ages 11 to 13 years. Growth in daytime sleepiness mediated this link. These findings held despite controlling for covariates known to contribute to both caffeine use and conduct problems among youth. At baseline, pubertal development was associated with conduct problems and daytime sleepiness. More rapid pubertal development over time was linked to increases in daytime sleepiness but not increases in conduct problems.

Study findings are consistent with past, cross-sectional research [12,16]. The present study along with accumulating empirical findings regarding the effects of caffeine on adolescents' substance use onset and escalation [27,32,33] converge to underscore the public health threat posed by use of caffeine by young adolescents. Although the effects of caffeine use among adults are mixed [34], the predominance of negative effects for early adolescents appears increasingly clear. Young adolescents experience rapid development in multiple neurobiological and socioemotional systems and are particularly sensitive to the effects of substances [11]. Our findings support the

recommendation of the AAP and AACAP that children under 12 years of age should not consume caffeine [10].

The present study is the first, to our knowledge, to consider mechanisms of action through which caffeine use promotes conduct problems. Daily caffeine use of 100+ mg predicts increases in daytime sleepiness, which, in turn, predicted growth in conduct problems. Study findings are consistent with research on the importance of optimal sleep among adolescents and the need to increase public health efforts aimed at deterring caffeine use and improving sleep hygiene practices [35]. In addition to poor sleep at night, caffeine use can generate withdrawal symptoms, as children cannot effectively "medicate" their dependency on caffeine during school hours leading to fatigue, irritation and problems with concentration [36]. This may undermine academic engagement [37] which, in turn can promote affiliations with peers who encourage externalizing and substance use behaviors [38]. Notably, we considered pubertal development as a covariate in the final model. Consistent with past research, baseline levels and growth in pubertal development were linked to increased daytime sleepiness [39]. Independent of pubertal development, caffeine evinced a significant effect on daytime sleepiness and conduct problems.

Study findings highlight the importance of interventions aimed at discouraging caffeine consumption by children and adolescents. Interventions with adults to reduce or quit caffeine have yielded promising results, especially in relation to strategies that encourage gradual reduction ("caffeine fading") over time [40]. Unfortunately, to our knowledge, no evidence-based interventions for preventing adolescent caffeine use exist. Effective strategies to consider include increased messaging to youth and parents, restricting caffeine access in schools, and consideration of governmental controls.

This study is the first, to our knowledge, to model group means in growth trajectories of daytime sleepiness and conduct problems as consequences of caffeine use. This methodological rigor increases confidence in drawing conclusions regarding effects. A number of limitations, however, pertain. First, our sample is from WV and includes primarily White/non-Hispanic children. Findings may not generalize to other geographical regions or racial/ethnic groups. Second, our study depends on youth self-report and common method bias may influence the strength of findings. No data were available on when children first began using caffeine. Although the use of trajectory modeling is a strength, evidence for the direction of effects is correlational and requires confirmation in experimental contexts. Our assessment of sleep was limited to daytime sleepiness. Poor quality sleep and sleep duration may more directly inform caffeine use and daytime sleepiness as well as affect the extent to which caffeine addiction and withdrawal symptoms occur. Future research should consider multidimensional assessments of sleep. Other limitations pertain to a lack of data on consumption of sugar and the need for additional follow up into midadolescence. These concerns notwithstanding, we conclude that daily caffeine consumption among early adolescents is positively associated with conduct problems over time and daytime sleepiness is a partial mediator of this effect.

Role of Funder/Sponsor

The NIH had no role in the design and conduct of the study.

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