




## ORIGINAL ARTICLE OPEN ACCESS

# Relationship Between Sleep Apnea Symptoms and Metabolic Syndrome Among Racially and Ethnically Diverse Adolescents and Young Adults in the United States

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## ABSTRACT

**Background:** Metabolic Syndrome (MetSyn) and Sleep Apnea (SA) contribute to long-term cardiometabolic risks among adolescents and young adults (AYAs). Emerging research suggests that certain race and ethnic groups experience disproportionate burdens of MetSyn and SA. Therefore, this study investigated the association of SA symptoms and MetSyn among AYAs in the United States and reported on associated racial and ethnic disparities.

**Methods:** National Health and Nutrition Examination Survey data from 2015 to 2020 ( $N = 2539$ ) were analyzed. Sleep disorders, medical conditions, and anthropometric data were collected via interviews and physical examinations. MetSyn was defined based on the International Diabetes Federation criteria. Bivariate associations were assessed by univariate logistic regression models and age-adjusted associations by multivariable logistic regression models. Stratified analyses examined race/ethnic group differences in the associations.

**Results:** Nearly 50% of the sample (mean age 20.6 years, 48.9% female, 55.3% non-Hispanic White) reported SA symptoms and 4.6% had MetSyn. After adjustment, central obesity was a consistent predictor of overall sleep apnea symptoms [aOR = 1.58; 95% CI: 1.29, 1.94], snoring [aOR = 2.10; 95% CI: 1.70, 2.60], breath cessation [aOR = 2.59; 95% CI: 1.42, 4.73] and daytime sleepiness [aOR = 1.34; 95% CI: 1.06, 1.68]. Non-Hispanic Black individuals with MetSyn had significantly higher odds of sleep apnea symptoms [aOR = 4.19; 95% CI: 1.40, 12.51], snoring [aOR = 6.64; 95% CI: 2.10, 21.0], and breath cessation [aOR = 8.64; 95% CI: 3.12, 23.93] versus participants of other races and ethnicities without MetSyn.

**Conclusion:** This study highlights the significant relationships between parameters of MetSyn and SA symptoms, and the disproportionately higher odds of SA symptoms among certain race/ethnic groups with a heavy burden of metabolic disorders.

## 1 | Introduction

Sleep apnea (SA), a chronic sleep-related disorder, characterized by repeated interruptions in breathing during sleep, has

emerged as a highly prevalent and increasingly recognized health concern in the United States (U.S.) [1]. Over the past 2 decades, the number of individuals with SA has increased from 25 million to 30 million [2], affecting 26% of Americans

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aged 30–70 years [3]. SA exhibits distinct prevalence patterns, with a higher prevalence among males versus females, older versus younger individuals, and Hispanics, non-Hispanic blacks, and Asians versus non-Hispanic whites [4–6]. Moreover, increased susceptibility to sleep disorders has been observed in 60%–70% of cerebrovascular and cardiovascular patients [7], 78% of metabolic and bariatric surgery (MBS) candidates [8], and 40% of long COVID patients [9] in the United States [3, 10]. While traditionally regarded as a condition that primarily affects older adults, SA has been increasingly recognized as a significant contributor to health issues among adolescents and young adults between the ages of 13–25 (AYAs) [4]. Health consequences in this age group extend beyond the mere disruption of sleep, affecting both short-term cognitive function and long-term health outcomes [5].

When discussing SA as a significant driver of AYA health, it is important to first consider both healthy and unhealthy sleep patterns. The American Academy of Sleep Medicine recommends that children 13–18 years get 8–10 h of sleep per night, while those  $\geq 18$  years get at least 7 h [6]. Unfortunately, only a small proportion of AYAs meet these recommendations; insights from recent decades demonstrates that nearly 73% and 33% of high school adolescents (15–18 years) and U.S. adults, respectively, experience sleep insufficiency [7]. Furthermore, U.S. individuals in both these age groups also experience high prevalences of sleep disturbances and daytime tiredness which could be indicative of sleep health disorders [8]. There are multiple drivers of negative sleep patterns among AYAs that are either intrinsic or extrinsic [7]. Intrinsic factors encompass the internal biological timing system known as the circadian rhythm and the sleep/wake homeostasis system. Some external factors contributing to poor sleep patterns among adolescents include academic requirements, extracurricular activities, and socializing with peers [7]. These factors may contribute to adolescents getting inadequate sleep given that a hallmark of adolescence is increased academic and social obligations and involvement in extracurricular activities such as sports. Other extrinsic factors contributing to adolescent sleep patterns include the use of electronic devices such as computers, phones, and television [7]. These factors particularly may contribute to adolescents getting inadequate sleep in the absence of adult/parental supervision. The burden of sleep insufficiency, particularly among adolescents, as well as the multiple drivers of negative sleep patterns among AYAs call for a closer evaluation of specific sleep-related disorders such as SA within this group.

A related and equally concerning public health issue among the U.S. AYA population is the growing prevalence of metabolic syndrome (MetSyn)—a group of metabolic abnormalities that includes obesity, high blood pressure, dyslipidemia, and insulin resistance, among the U.S. AYA population [9–11]. A mounting body of evidence has suggested a bidirectional relationship between SA and MetSyn [12–14]. Indeed, current literature posits that the rapid acceleration of obesity in the United States may be a significant contributor to the simultaneous increase in SA [11]. Notably, 40% of individuals with obesity are afflicted by SA, while 70% of SA patients have obesity [15]. Additionally, adolescents (12–18 years) were 3.5 times more likely to have obstructive SA with every 1 standard deviation increase in BMI z-score, further substantiating the inter-connectedness between

metabolic health and sleep disorders among U.S. AYAs [16]. While current literature examines the relationship between SA and metabolically-associated anthropometric measures, including waist and neck circumference, body mass index (BMI), and body weight [15, 17, 18], there is a dearth of research examining the independent associations of other metabolic parameters, encompassed within MetSyn, and SA among both adolescents and young adults in the United States, especially from the perspective of racial and ethnic group disparities.

The prevalence of SA and MetSyn varies across race and ethnicity groups in the United States [19], and is most prevalent among African American, Hispanic, and Native American communities [20, 21]. The disproportionately high rates of metabolic disorders, especially obesity, among African American individuals significantly increases their risk of SA compared with other races and ethnicities [22]. Additionally, SA-related mortalities in the United States are more common among Black versus White populations, with annual mortalities consistently increasing by 2.7% among Black individuals from 1999 to 2019 [23]. These disparities are further amplified by differences in health insurance coverage, healthcare access and utilization, and diagnosis rates across race and ethnicity groups [24]. This is a significant public health concern given that SA is an independent risk factor for multiple cardiovascular disease ( $p < 0.05$ ) and mortality among minority race and ethnicity groups [24]. Despite this, very few studies have examined the relationships between SA and MetSyn among an ethnically diverse population of AYAs [25, 26]. This is a critical gap in adolescent sleep and metabolic health literature that warrants further investigation into the extent to which race and ethnicity influence the relationship between SA and MetSyn among AYAs.

Therefore, this study aimed to bridge this gap by examining the relationship between SA and MetSyn among U.S. AYAs, identifying key relationships between MetSyn parameters and SA symptoms, and assessing race/ethnicity group disparities in these relationships. We hypothesized that U.S. AYAs with an SA symptom will have increased odds of MetSyn compared with AYAs without an SA symptom. Additionally, we expected to see higher odds of MetSyn among AYAs identifying as non-Hispanic Black or Hispanic/Latino compared to those identifying as non-Hispanic White.

## 2 | Methods

This cross-sectional study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines [27].

### 2.1 | Study Data & Design

This study utilized cross-sectional data collected by the National Health and Nutrition Examination Survey (NHANES) between 2015 and 2020. As detailed in the NHANES survey methods and analytic guidelines [28], data from the 2019–2020 and 2017–2018 cycles were combined to create a nationally representative

sample with minimal impact from sampling disruptions caused by the COVID-19 pandemic in 2020 [29]. The sample quality was further strengthened by the complex 3-step weighting method created by NHANES, which ensured that the NHANES sample was representative of the larger non-institutional, civilian U.S. population [28]. For this study, MEC weights generated by NHANES were combined for the 2015–2016 and 2017–2020 data cycles, and applied to the study sample. In addition, the appropriate NHANES-generated variables for sample clusters and strata were included in all analyses. More details regarding the NHANES weights can be found in the NHANES Analytics Guidelines for the 2015–2016 data cycle [28].

## 2.2 | Participant Recruitment & Study Sample

AYAs between the ages of 16–25 years ( $N = 2539$ ) were included in this study. They were part of a larger sample of individuals ( $N = 25,531$ ) living in the United States or U.S. territories that were recruited by NHANES through a multistage, representative sampling approach [28]. This involved random selection of sampling units, followed by the recruitment of households and individuals to participate in comprehensive health examinations and interviews. Further details regarding this sampling technique are provided in the NHANES survey methods and analytic guidelines [28].

While the definition of adolescence often includes individuals younger than 16 years [30], this analysis was restricted to those 16 years or older due to the lack of sleep disorder data for younger age groups in the NHANES database. In addition, participants with missing data regarding MetSyn parameters (central obesity, high-density lipoprotein (HDL) cholesterol, triglycerides, blood pressure readings, and fasting glucose levels) and SA risk factors were assumed to be missing at random and were excluded from the analysis ( $N = 399$ ). Therefore, the final study sample included 2539 AYAs (Figure 1).

All participants above the age of 18 years provided written informed consent prior to enrolling in the NHANES study. Those below the age of 18 provided their assent along with parental permission to participate. All data were de-identified by NHANES and made publicly available. The review board of the University of Texas Health Science Center at Houston deemed this study to be exempt from review because of the use of de-identified, publicly available data.

## 2.3 | Assessment of Demographics & Body Measurements

NHANES collected a number of demographic details about participants enrolled in their study. These demographics were either self-reported on questionnaires or recorded during in-person interviews using computer-assisted personal interviewing (CAPI). Demographic data included participants' age (in years), gender (male or female), and race/ethnicity (non-Hispanic White, non-Hispanic Black, non-Hispanic Asian, Hispanic/Latino, Other or multiple races), which were included as covariates for this study. The use of CAPI reduced errors in data

entry and strengthened the integrity of the data. Additionally, participants' height, weight, waist circumference, body mass index (BMI), and blood pressure readings were measured during physical examinations in mobile examination centers (MEC). Biospecimen samples were also collected during these visits.

## 2.4 | Assessment of Outcome: Sleep Apnea Symptoms

SA symptoms were assessed using the NHANES Sleep Disorder Questionnaire (SLQ). Items on the SLQ included (1) frequency of snoring ("In the past 12 months, how often did you snore while you were sleeping?"), (2) snorting or gasping or breathing cessation ("In the past 12 months, how often did you snort, gasp, or stop breathing while sleeping?"), and (3) feeling excessively/overly sleepy during the day ("In the past month, how often did you feel excessively or overly sleepy during the day?") [31]. For this study, a composite SA variable was created where "never" or "rarely" (1–2 nights/week) snoring or snorting/gasping or breathing cessation were categorized as non-SA symptoms, while "occasionally" (3–4 nights/week) or "frequently" ( $\geq 5$  nights/week) were categorized as SA symptoms. Similarly, for daytime sleepiness, "never," "rarely" (1 time/month), and "sometimes" (2–4 times/month) were categorized as non-SA symptoms, while "often" (5–15 times/month) and "almost always" (16–30 times/month) were categorized as SA symptoms. Participants who did not provide an answer to any of the SLQ items were not included in the analysis ( $N = 129$ ).

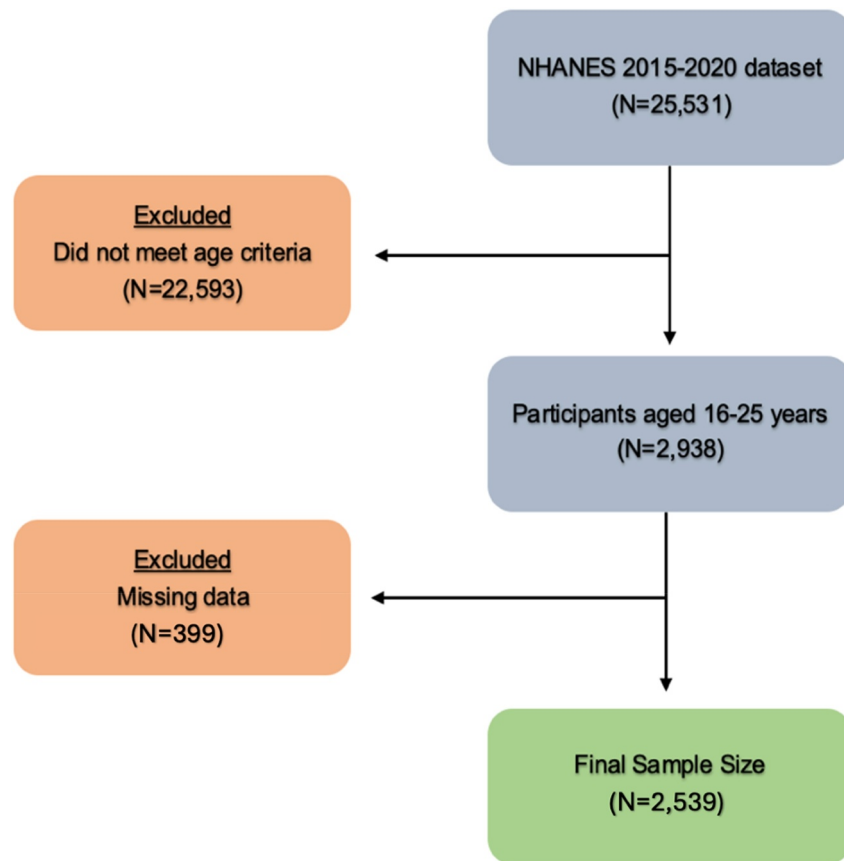
## 2.5 | Assessment of Exposure: Metabolic Syndrome

Based on prior literature [32–36], a dichotomous MetSyn outcome variable was created for this study. MetSyn was defined as having central obesity (i.e., waist circumference  $\geq 94$  cm in males and  $> 80$  cm in females) with two or more of the following metabolic risk factors:

(1) **Hypertension**: elevated systolic ( $\geq 130$  mmHg) or diastolic ( $\geq 85$  mmHg) blood pressure; (2) **Hyperglycemia**: an elevated fasting glucose level ( $\geq 5.6$  mmol/L); (3) **Low HDL cholesterol**:  $\leq 1.03$  mmol/L for males and  $\leq 1.29$  mmol/L females; (4) **Hypertriglyceridemia**: elevated triglyceride ( $\geq 1.7$  mmol/L). For this study, the average of 3 blood pressure readings was used (for both SBP and DBP) to account for within-person variations in measurements. Enzymatic and photometric methodology was used to measure serum triglyceride and fasting glucose levels, which is elaborated in the NHANES laboratory procedure manual [37].

## 2.6 | Statistical Analysis

Individual NHANES datasets were sorted and merged by participant ID to create a complete dataset for analysis. Participant demographics were expressed as frequencies and weighted percentages for categorical characteristics and mean and standard error (SE) for continuous characteristics. All bivariate



**FIGURE 1** | Selection of participants from the NHANES 2015–2020 study sample. Participants were chosen from the NHANES 2015–2016 and 2017–2020 data cycles ( $N = 25,531$ ). After excluding participants younger than 16 years or older than 25 years ( $N = 22,593$ ), we further excluded those with missing data on metabolic syndrome and sleep disorders ( $N = 399$ ). Therefore, our final unweighted sample included 2539 participants.

associations between MetSyn parameters, SA symptoms and participant demographics were assessed using weighted univariate logistic regression models. The age-adjusted associations between MetSyn parameters and SA symptoms were determined using weighted multivariable logistic regression models. A stratified analysis by race/ethnicity was performed to examine the age-adjusted association of SA symptoms with MetSyn by race/ethnicity groups. All regression models were appropriately weighted and accounted for the NHANES clustered and stratified sampling design. Odds ratios (OR) and 95% confidence intervals (CI) were reported. The type I error ( $\alpha$ ) level was set at 5%; a  $p$ -value  $< 0.05$  was considered statistically significant. All analyses were performed using SAS Studio version 9.4 (SAS Institute, Cary, NC).

### 3 | Results

This study included 2539 AYAs (mean age 20.6 years ( $SE = 0.1$ ); 48.9% female, 55.3% non-Hispanic White, 12.9% non-Hispanic Black, 8.5% Hispanic, 4.9% non-Hispanic Asian, 13.5% Mexican American, and 4.9% other or multi-race). Roughly 4.6% of participants had MetSyn, while 47.9% had central obesity, 7.3% had elevated blood pressure, 8.5% had elevated triglycerides, 30.9% had low HDL cholesterol, and 26.3% had elevated fasting glucose (Table 1).

Approximately 58.6% of participants reported SA symptoms: 22.6% reported snoring, 3.9% experienced breath cessation while asleep, and 32.1% experienced daytime sleepiness (Supporting Information Table S1 and S2). The prevalence of SA symptoms was highest among individuals who identified as non-Hispanic white (54.65%), followed by non-Hispanic Black (13.3%), Mexican American (13.2%), and Hispanic (9.1%) participants. The prevalence of MetSyn by individual SA symptoms is presented in Figure 2; there was a higher prevalence of MetSyn among those with versus without SA symptoms (4.7% vs. 4.5%;  $p = 0.84$ ). When examined by individual SA symptoms, participants who experienced breath cessation had a significantly higher prevalence of MetSyn (12.9% vs. 4.2%) compared with those who did not experience breath cessation ( $p = 0.01$ ). Overall, SA symptoms were significantly associated with age, central obesity, elevated blood pressure, and low HDL cholesterol (Table 1).

Bivariate analyses highlighted notable associations between each MetSyn parameter and individual SA symptoms (Table 2). After adjustment for age, results showed that individuals with MetSyn had 3.02 higher odds [95% CI: 1.17, 7.78] of experiencing breath cessation versus those without MetSyn. Elevated blood pressure significantly increased the odds of overall SA symptoms [aOR = 1.59; 95% CI: 1.10, 2.30], snoring [aOR = 2.30; 95% CI: 1.46, 3.60] and breath cessation [aOR = 4.63; 95% CI: 1.77, 12.05] (Table 2). Central obesity was consistently associated with overall SA symptoms [aOR = 1.58; 95% CI: 1.29, 1.94],

**TABLE 1** | Participant descriptive and metabolic syndrome (MetSyn) characteristics by prevalence of sleep apnea symptoms ( $n = 2539$ ), National Health and Nutrition Examination Survey, 2015–2020.

	Overall ( $n = 2539$ )	Sleep apnea symptoms		<i>p</i> -value
		No ( $n = 1376$ )	Yes ( $n = 1163$ )	
Age (in years) <sup>a</sup>	20.6 (0.1)	20.4 (0.1)	20.8 (0.1)	0.01*
Female <sup>b</sup> , $n$ (%)	1278 (48.9)	669 (47.2)	609 (50.8)	0.13
Race/Ethnicity <sup>b</sup> , $n$ (%)				0.47
Mexican American	441 (13.5)	245 (13.8)	196 (13.2)	
Hispanic	263 (8.5)	143 (7.9)	120 (9.1)	
Non-Hispanic white	772 (55.3)	410 (55.9)	362 (54.6)	
Non-Hispanic black	612 (12.9)	331 (12.5)	281 (13.3)	
Asian	274 (4.9)	160 (5.4)	114 (4.4)	
Other/multi-race	177 (4.9)	87 (4.4)	90 (5.5)	
Socioeconomic status <sup>b</sup> , $n$ (%)				0.26
At or below poverty line	947 (28.8)	536 (29.9)	411 (27.7)	
Above poverty line	1592 (71.2)	840 (70.1)	752 (72.3)	
MetSyn individual parameters <sup>b</sup> , MetSyn <sup>c</sup> , $n$ (%)				
Central obesity	1161 (47.9)	548 (57.7)	613 (54.4)	< 0.01*
Elevated blood pressure	156 (7.3)	66 (5.7)	90 (9.2)	< 0.01*
Elevated triglyceride	75 (8.5)	41 (9.3)	34 (7.5)	0.51
Low HDL cholesterol	744 (30.9)	345 (26.1)	399 (36.2)	< 0.01*
Elevated fasting glucose	278 (26.3)	133 (25.2)	145 (27.6)	0.58
MetSyn	110 (4.6)	54 (4.5)	56 (4.7)	0.84

<sup>a</sup>Mean (SD) reported.

<sup>b</sup> $N$  (%) reported.

<sup>c</sup>(> 2 parameters and central obesity).

\*statistically significant at  $\alpha = 0.05$ .

snoring [aOR = 2.10; 95% CI: 1.70, 2.60], breath cessation [aOR = 2.59; 95% CI: 1.42, 4.73] and daytime sleepiness [aOR = 1.34; 95% CI: 1.06, 1.68]. However, low HDL was the strongest predictor of overall SA symptoms [aOR = 1.62; 95% CI: 1.23, 2.14], and was associated with 62% and 34% higher odds of snoring [aOR = 1.62; 95% CI: 1.24, 2.12] and experiencing daytime sleepiness [aOR = 1.34; 95% CI: 1.01, 1.80], respectively. The forest plots in Figure 3 offer a visual perspective of these age-adjusted associations.

The stratified analysis by race/ethnicity demonstrated a consistently higher likelihood of SA symptoms, snoring, and breath cessation among non-Hispanic Black participants who had MetSyn (Table 3); these participants were 4.2 times more likely to experience SA symptoms [aOR = 4.19; 95% CI: 1.40, 12.51], 6.64 times more likely to snore [aOR = 6.64; 95% CI: 2.10, 20.99], and 8.64 times more likely to experience breath cessation [aOR = 8.64; 95% CI: 3.12, 23.93] while asleep. Non-Hispanic Asian participants with elevated blood pressure had the highest odds of experiencing overall SA symptoms [aOR = 3.98; 95% CI: 1.03, 15.31] while Non-Hispanic Black participants with elevated blood pressure had the higher odds of snoring [aOR = 3.18; 95% CI: 1.57, 6.42], compared to participants of other race and ethnicities (Table 3).

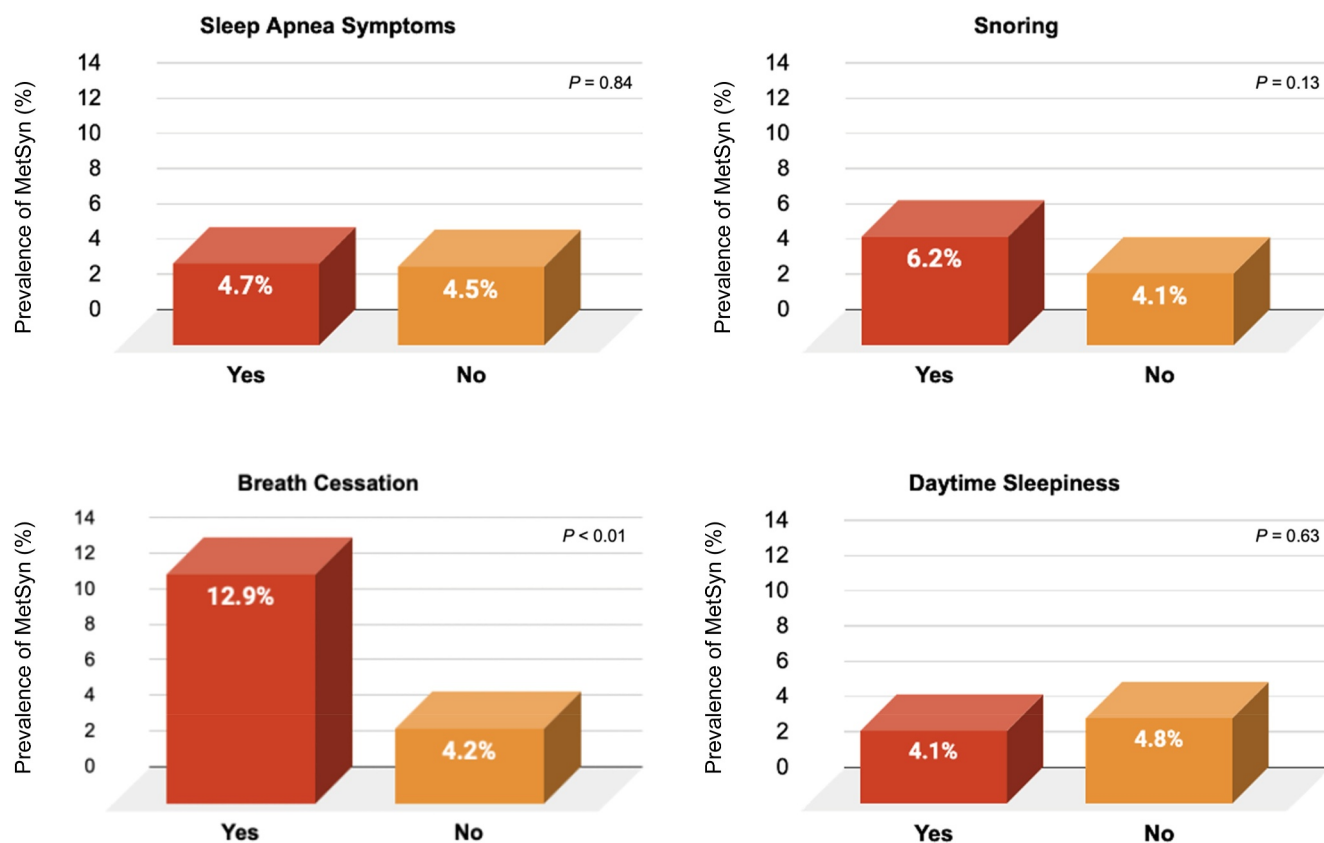
Furthermore, central obesity was the most significant predictor of overall SA symptoms, snoring, and breath cessation across most

racies/ethnicities. Non-Hispanic Asian participants with central obesity had the highest odds of overall SA symptoms [aOR = 2.34; 95% CI: 1.38, 3.96 and snoring [aOR = 3.11; 95% CI: 1.65, 5.85], and breath cessation [aOR = 5.80; 95% CI: 2.27, 14.86]. Similar associations were observed among participants with low HDL cholesterol; non-Hispanic Black participants with low HDL had the highest odds of overall SA symptoms [aOR = 1.849 95% CI: 1.24, 2.89] and snoring [aOR = 2.20; 95% CI: 1.33, 3.64], while Non-Hispanic Asian participants with low HDL were the only race and ethnicity group to have significantly higher odds of breath cessation [aOR = 3.55; 95% CI: 1.60, 7.88] (Table 3). Lastly, Non-Hispanic Black participants with elevated fasting glucose were the only group to demonstrate increased odds of overall SA symptoms [aOR = 2.91; 95% CI: 1.39, 6.11] and breath cessation [aOR = 2.96; 95% CI: 1.23, 7.15] (Table 3).

## 4 | Discussion

This study is one of the first to examine SA symptoms as an outcome of MetSyn exposure. Findings suggest that nearly half of the U.S. AYA population between 2015 and 2020 experienced SA symptoms including snoring, breath cessation, and daytime sleepiness, and almost 5% met the criteria for MetSyn. While MetSyn only had a statistically significant association with breath cessation, individual MetSyn parameters including





**FIGURE 2** | Prevalence of metabolic syndrome (MetSyn) by sleep apnea symptoms.

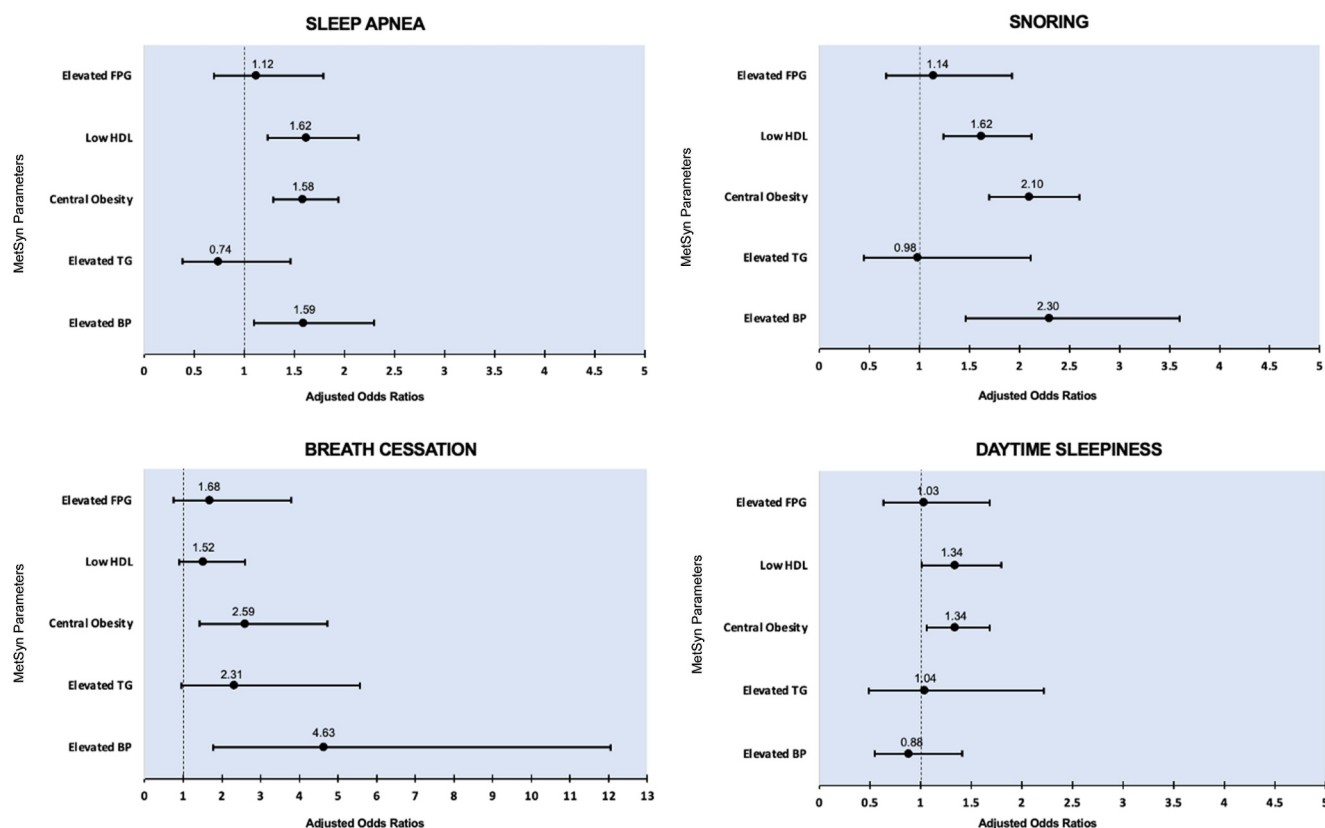
**TABLE 2** | The bivariate and age-adjusted associations between metabolic syndrome parameters and sleep apnea.

	Sleep apnea OR (95% CI)	Snoring OR (95% CI)	Breath cessation OR (95% CI)	Daytime sleepiness OR (95% CI)
MetSyn				
Unadjusted	1.05 (0.62, 1.78)	1.56 (0.86, 2.85)	3.34 (1.32, 8.47)*	0.86 (0.45, 1.63)
Adjusted	1.01 (0.60, 1.70)	1.45 (0.81, 2.62)	3.02 (1.17, 7.80)*	0.98 (0.85, 1.06)
Elevated blood pressure				
Unadjusted	1.68 (1.14, 2.47)*	2.53 (1.62, 3.94)*	5.36 (1.95, 14.69)*	0.90 (0.57, 1.43)
Adjusted	1.59 (1.10, 2.30)*	2.30 (1.46, 3.60)*	4.63 (1.77, 12.05)*	0.88 (0.55, 1.41)
Elevated triglyceride				
Unadjusted	0.80 (0.41, 1.57)	1.06 (0.49, 2.27)	2.50 (1.02, 6.12)*	1.09 (0.52, 2.28)
Adjusted	0.74 (0.38, 1.46)	0.98 (0.45, 2.11)	2.31 (0.96, 5.56)	1.04 (0.49, 2.22)
Central obesity				
Unadjusted	1.62 (1.34, 1.97)*	2.20 (1.78, 2.72)*	2.79 (1.54, 5.07)*	1.35 (1.08, 1.68)*
Adjusted	1.58 (1.29, 1.94)*	2.10 (1.70, 2.60)*	2.59 (1.42, 4.73)*	1.34 (1.06, 1.68)*
Low HDL cholesterol				
Unadjusted	1.60 (1.22, 2.11)*	1.58 (1.21, 2.07)*	1.46 (0.87, 2.46)	1.33 (0.99, 1.80)
Adjusted	1.62 (1.23, 2.14)*	1.62 (1.24, 2.12)*	1.52 (0.90, 2.59)	1.34 (1.01, 1.80)*
Elevated fasting glucose				
Unadjusted	1.13 (0.72, 1.79)	1.15 (0.69, 1.91)	1.71 (0.77, 3.81)	1.03 (0.63, 1.68)
Adjusted	1.12 (0.70, 1.79)	1.14 (0.67, 1.93)	1.68 (0.75, 3.79)	1.03 (0.63, 1.68)

Note: Reference group for each parameter included those who did not meet the criteria for that parameter.

Abbreviations: CI = confidence intervals; OR = odds ratios.

\*Significant at  $p < 0.05$ .



**FIGURE 3** | Age-adjusted associations between metabolic syndrome parameters and sleep apnea symptoms. BP = blood pressure; FPG = fasting plasma glucose; HDL = high-density lipoprotein; MetSyn = Metabolic Syndrome; TG = triglycerides.

central obesity, elevated blood pressure, and low HDL cholesterol significantly increased the odds of experiencing SA symptoms. The high prevalence of MetSyn features and SA symptoms among AYAs in the United States is concerning given the clinical implications of poor metabolic and sleep health on adolescent development and well-being. Sleep disorders, especially SA, impact elements of adolescent cognitive and emotional health [38], contributing to an increased vulnerability to depression and anxiety, impaired academic performance, and a heightened risk of accidents and injuries [5, 39]. An interesting aspect of the relationship between SA symptoms and individual MetSyn features is its bidirectionality.

Previous research has demonstrated significant relationships between metabolic irregularities and sleep disorders. As such, central obesity—measured by waist circumference—and deposition of ectopic fat in the pharynx region narrows the airways and reduces lung volume over time, making it difficult to breathe while asleep and increasing the frequency of snoring, experiencing shortness of breath, and collapse of airways [40, 41]. Findings from this study aligned with previous research, demonstrating 58% higher odds of SA and a greater than 2-fold increase in the likelihood of SA-related symptoms among those with versus those without central obesity. Conversely, studies have found that SA-related sleep deprivation and reduced sleep quality cause increased caloric intake, obesity, ectopic fat, insulin resistance and eventually MetSyn, further demonstrating the strong bidirectional relationship between central obesity and SA [42–44].

Independent of obesity, several studies have linked impaired glucose metabolism and insulin resistance to SA-related symptoms [45–48]. The American Diabetes Association (ADA) and American Association of Sleep Medicine (AASM) have recognized type 2 diabetes as an independent risk factor for SA, influencing sleep outcomes through the central nervous system [47, 49]. Several studies provide evidence of SA increasing the risk or likelihood of glucose dysfunction, but very few have examined this relationship in the opposite direction. This study is one of the first to demonstrate a 14%–66% increase in the likelihood of several SA symptoms with elevated fasting glucose levels among AYAs. In addition, hypertension is one of the many cardiovascular disorders that increases the risk of SA and its symptoms [50]. Literature posits that pathophysiological factors including hypoxemia, nocturnal fluid shift, increased sympathetic tone, impaired quality of sleep, and the renin-angiotensin-aldosterone system contribute to the interdependent relationship between hypertension and SA [51]. However, a majority of studies focus on the effect of SA on the risk of hypertension rather than vice versa. For instance, hypoxia—as a result of SA—is known to contribute significantly to the risk of developing hypertension and hyperglycemia [52, 53]. Other reviews and meta-analyses have also found an independent association between OSA and MetSyn among adolescents—specifically, OSA was associated with an increased risk of insulin resistance, hypertension, and dyslipidemia [54]. Although these studies focus on older individuals, they underscore the potential mechanisms that may be driving the relationship between SA symptoms and MetSyn among AYAs in the United States. This study presents

**TABLE 3** | Age-adjusted associations of sleep apnea symptoms and metabolic syndrome (MetSyn) by race and ethnicity.

	<b>Sleep apnea OR (95% CI)</b>	<b>Snoring OR (95% CI)</b>	<b>Breath cessation OR (95% CI)</b>	<b>Daytime sleepiness OR (95% CI)</b>
<b>MetSyn</b>				
Mexican American	0.96 (0.58, 1.60)	1.52 (0.79, 2.92)	6.26 (1.53, 25.59)*	0.40 (0.13, 1.26)
Hispanic	0.36 (0.08, 1.63)	0.53 (0.09, 3.04)	a	0.21 (0.02, 2.00)
Non-Hispanic white	1.30 (0.53, 3.17)	1.41 (0.49, 4.06)	2.42 (0.43, 13.48)	1.52 (0.65, 3.57)
Non-Hispanic black	4.19 (1.40, 12.51)*	6.64 (2.10, 20.99)*	8.64 (3.12, 23.93)*	1.08 (0.46, 2.52)
Asian	0.07 (0.01, 0.73)	a	a	0.20 (0.02, 1.97)
Other/Multi-race	0.40 (0.06, 2.73)	0.84 (0.15, 4.72)	a	0.33 (0.04, 2.61)
<b>Elevated blood pressure</b>				
Mexican American	1.70 (0.54, 5.33)	3.14 (1.05, 9.41)*	10.66 (2.16, 52.54)*	0.41 (0.08, 1.97)
Hispanic	0.72 (0.16, 3.21)	1.90 (0.35, 10.24)	1.72 (0.19, 15.66)	0.72 (0.09, 5.44)
Non-Hispanic white	1.57 (0.91, 2.72)	2.05 (0.97, 4.29)	6.27 (1.41, 27.80)*	1.01 (0.53, 1.91)
Non-Hispanic black	2.60 (1.48, 4.56)*	3.18 (1.57, 6.42)*	2.77 (1.06, 7.25)*	0.90 (0.47, 1.73)
Asian	3.98 (1.03, 15.31)*	3.45 (0.74, 16.20)	a	2.50 (0.52, 12.06)
Other/Multi-race	0.69 (0.19, 2.52)	1.58 (0.44, 5.73)	17.25 (3.30, 90.21)*	0.46 (0.10, 2.09)
<b>Elevated triglyceride</b>				
Mexican American	1.02 (0.51, 2.06)	1.39 (0.71, 2.74)	5.08 (1.59, 16.24)*	1.21 (0.34, 4.27)
Hispanic	0.24 (0.03, 2.27)	a	a	0.50 (0.04, 5.96)
Non-Hispanic white	0.85 (0.27, 2.70)	1.28 (0.38, 4.31)	1.11 (0.46, 2.69)	1.17 (0.37, 3.71)
Non-Hispanic black	2.61 (0.19, 36.33)	2.75 (1.05, 7.21)*	10.96 (3.91, 30.76)*	3.49 (0.51, 23.63)
Asian	0.17 (0.02, 1.60)	a	a	0.64 (0.08, 5.10)
Other/Multi-race	0.37 (0.08, 1.79)	0.23 (0.05, 1.19)	a	0.67 (0.13, 3.42)
<b>Central obesity</b>				
Mexican American	1.17 (0.76, 1.79)	1.19 (0.78, 1.82)	1.98 (0.66, 5.95)	1.25 (0.79, 1.99)
Hispanic	1.45 (0.86, 2.44)	1.64 (0.81, 3.31)	1.59 (0.59, 4.32)	1.11 (0.74, 1.67)
Non-Hispanic white	1.58 (1.14, 2.19)*	2.39 (1.65, 3.47)*	3.66 (1.44, 9.32)*	1.34 (0.93, 1.94)
Non-Hispanic black	1.80 (1.35, 2.40)*	2.31 (1.56, 3.41)*	1.59 (0.51, 4.98)	1.57 (1.12, 2.20)*
Asian	2.34 (1.38, 3.96)*	3.11 (1.65, 5.85)*	5.80 (2.27, 14.86)*	1.15 (0.56, 2.34)
Other/Multi-race	2.28 (1.13, 4.59)*	2.27 (0.95, 5.41)	a	2.13 (0.86, 5.24)
<b>Low HDL cholesterol</b>				
Mexican American	0.96 (0.60, 1.54)	1.10 (0.63, 1.91)	1.38 (0.62, 3.06)	0.69 (0.42, 1.13)
Hispanic	1.47 (0.89, 2.45)	1.60 (1.02, 2.50)*	2.12 (0.80, 5.61)	1.44 (0.84, 2.48)
Non-Hispanic white	1.87 (1.19, 2.94)*	1.78 (1.13, 2.80)*	1.41 (0.59, 3.34)	1.60 (0.99, 2.58)
Non-Hispanic black	1.89 (1.24, 2.89)*	2.20 (1.33, 3.64)*	1.64 (0.61, 4.35)	1.29 (0.87, 1.92)
Asian	1.87 (1.24, 2.83)*	1.84 (1.15, 3.00)*	3.55 (1.60, 7.88)*	1.00 (0.64, 1.54)
Other/Multi-race	1.64 (0.49, 5.49)	1.13 (0.33, 3.90)	0.44 (0.02, 8.48)	1.70 (0.54, 5.36)
<b>Elevated fasting glucose</b>				
Mexican American	1.37 (0.63, 2.98)	1.71 (0.61, 4.75)	1.82 (0.50, 6.64)	0.84 (0.46, 1.53)
Hispanic	1.05 (0.42, 2.62)	0.88 (0.34, 2.29)	0.41 (0.04, 4.29)	1.34 (0.46, 3.88)
Non-Hispanic white	0.90 (0.44, 1.82)	0.66 (0.27, 1.64)	1.62 (0.37, 7.08)	1.06 (0.51, 2.21)
Non-Hispanic black	2.91 (1.39, 6.11)*	2.28 (1.14, 4.58)*	2.96 (1.23, 7.15)*	0.82 (0.51, 1.33)
Asian	1.33 (0.53, 3.34)	0.70 (0.30, 1.62)	1.12 (0.66, 1.88)	3.20 (1.33, 7.73)*
Other/multi-race	1.51 (0.55, 4.20)	6.28 (1.50, 26.27)*	4.11 (0.34, 49.29)	0.99 (0.34, 2.89)

Abbreviations: aOR = adjusted odds ratios; CI = confidence intervals.

a sample size not sufficient to generate odds ratios | Reference group for each parameter included those who did not meet the criteria for that parameter.

\*Significant at  $p < 0.05$ .



evidence of a 59% increase in the likelihood of SA symptoms among AYAs with elevated blood pressure, and a 2- to 4-fold increase in snoring and breath cessation, respectively.

Another important point of consideration is the racial/ethnic disparities in the prevalence and risk of SA. Multiple studies have found that the prevalence of OSA is higher among non-Hispanic black versus non-Hispanic White children, and that women identifying as non-Hispanic black, Hispanic, or other/multiple races are more likely to have MetSyn compared to non-Hispanic white women [20, 55–57]. However, very few studies have examined the association of MetSyn and SA symptoms in a racially and ethnically diverse sample of AYAs. This study addresses this gap by highlighting the disproportionately higher odds of SA and its symptoms among non-Hispanic Black adolescents with MetSyn, elevated blood pressure, elevated triglycerides, central obesity, low HDL cholesterol, and/or elevated fasting glucose. Additionally, Mexican American adolescents with elevated blood pressure and elevated triglycerides were likely to snore and experience breath cessation, respectively. By demonstrating the heightened risk of SA symptoms among non-Hispanic Black adolescents and Mexican American adolescents with MetSyn-related conditions in the United States [55, 58, 59], this study underscores the urgent need for extensive sleep-disorder screening of AYAs who present with metabolic abnormalities. Integrating considerations of metabolic health into sleep health interventions is paramount, especially in addressing sleep disorders among adolescents.

This study is one of the first in the United States to present detailed insights on the association between MetSyn parameters and SA symptoms in a racially and ethnically diverse sample of AYAs. The sample weights applied to NHANES data ensured nationally representative and unbiased measures of associations, allowing for the generalizability of this study's findings to AYAs aged 16–25 years across the United States. Additionally, the analytical sample was considerably large, comprising 2539 AYAs, with a relatively balanced number of individuals across SA groups. The use of such a large sample size allowed for the generation of a precise estimate of the association between MetSyn and SA symptoms within this study population. The use of nationally representative data provided us an opportunity to better understand the specific public health needs of an important segment of the U.S. population. Collectively, these findings suggest the need to increase screening for MetSyn parameters within AYA populations, given their significant associations with SA and SA symptoms.

It is important to consider the limitations of this study while interpreting its results. Due to the limited SA symptom-related information collected by NHANES, this study was unable to analyze other SA symptoms such as fatigue, sleep duration, and sleep fragmentation. Supporting sleep studies to verify the true prevalence of SA and its related symptoms were not conducted in this sample, given the focus on available data on SA symptoms. As such, the approximation of sleep apnea using associated symptoms may encompass some amount of misclassification. Adolescents may report daytime sleepiness for several reasons unrelated to SA, including staying up late at night, unusual sleep schedule, or heavy school/college workload, which could not be accounted for in this study. Other behavioral factors such as

alcohol use and cigarette smoking could not be examined as covariates in this study, given that this data was only available for participants above the age of 18 years. Thus, the influence of residual confounding cannot be excluded. Because of the study's cross-sectional design, it was not possible to establish temporality or causation between SA symptoms and MetSyn or its components. The use of a longitudinal design, such as a prospective cohort study or a randomized controlled trial, may be more conducive to elucidating a temporal relationship between exposure and outcome. However, there are important factors related to both time and cost that should be considered when implementing a longitudinal study design.

## 5 | Conclusion

This study underscores the significant relationship between SA symptoms and MetSyn among U.S. adolescents and young adults, with central obesity and elevated blood pressure emerging as key contributors. While there was no significant modification by race and ethnicity, findings highlighted notable disparities in the likelihood of SA symptoms across race and ethnicity groups. Given the increasing prevalence of both SA and MetSyn in younger populations, addressing these interconnected health concerns through early screening, lifestyle interventions, and equitable healthcare access is imperative. Future research should further investigate causal pathways and explore tailored strategies to mitigate these disparities and improve overall health outcomes in at-risk populations.

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## Author Contributions

D.K.E. and S.E.M. were responsible for the study design, analysis, interpretation, writing, critical review, and final approval of the final manuscript. A.C., L.X., and J.P.A. supported with the writing, critical review, and approval of the final manuscript.

## Conflicts of Interest

The authors declare no conflicts of interest.

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## Supporting Information

Additional supporting information can be found online in the Supporting Information section.