Sleep Duration and Risk of Type 2 Diabetes

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abstract

BACKGROUND: Associations between sleep duration and type 2 diabetes (T2D) risk markers in childhood have been little studied. We examined associations between self-reported sleep duration and T2D risk markers in children.

METHODS: Cross-sectional study of 4525 multiethnic UK children aged 9 to 10 years. Sleep time was calculated from self-reported usual time of going to bed and getting up on a school day, validated in a subset using accelerometers. Fasting blood samples provided levels of serum lipids and insulin, plasma glucose, and HbA1c. Physical measures included height, weight, bioimpedance, and blood pressure. Multilevel linear regression models of anthropometric, T2D, and cardiovascular risk markers with sleep duration were adjusted for sex, age, month, ethnicity, socioeconomic position, observer (physical measures only), and random effect of school.

RESULTS: On average, children slept 10.5 hours per night (95% range 8.0–12.0 hours). There were strong inverse graded relationships between sleep duration, adiposity, and diabetes risk markers. In adjusted models, a 1-hour-longer sleep duration was associated with 0.19 lower BMI (95% confidence interval [CI] 0.09 to 0.28), 0.03 kg/m^5 lower fat mass index (95% CI 0.00 to 0.05 kg/m^5), 2.9% lower homeostasis model assessment insulin resistance (95% CI 1.2% to 4.4%), and 0.24% lower fasting glucose (95% CI 0.03% to 0.44%); there was no association with HbA1c or cardiovascular risk. Associations with insulin and glucose remained after an additional adjustment for adiposity markers.

CONCLUSIONS: The finding of an inverse association between sleep duration and T2D risk markers in childhood is novel. Intervention studies are needed to establish the causality of these associations, which could provide a simple strategy for early T2D prevention.

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Dr Rudnicka conducted the initial analyses and drafted the initial manuscript; and all authors conceptualized and designed the study, coordinated and supervised data collection, interpreted the data, critically reviewed and revised the manuscript, approved the final manuscript as submitted, and agree to be accountable for all aspects of the work.

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WHAT'S KNOWN ON THIS SUBJECT: Shorter sleep duration has been associated with type 2 diabetes (T2D) in adults and with obesity in both adults and children. However, associations between sleep duration and T2D risk markers in childhood have been little studied.

WHAT THIS STUDY ADDS: This study demonstrates a novel graded association between short sleep duration and elevated T2D risk markers in a large, multiethnic population of 9- to 10-year-old children. The report confirms the association between short sleep duration and body fatness.

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The prevalence of type 2 diabetes (T2D), overweight, and obesity has been rising in the United Kingdom and in many other countries,[1,2] not only in adults but also in adolescents and children.[3] Understanding the early determinants of adiposity and T2D risk in young people could be particularly important for reducing the risks of T2D and obesity across the life course.

There has been particular interest in the importance of sleep for T2D and adiposity risks. Sleep duration has complex prospective relations with adiposity and T2D in adults, and both short and long sleep durations are associated with higher risk.[4–6] In contrast, studies in childhood have shown graded inverse associations between sleep duration and levels of adiposity, with increased sleep duration associated with lower levels of obesity; there has been little evidence that longer sleep duration is associated with increased adiposity.[5,7] However, little is known about the effects of sleep on other T2D risk markers in childhood, particularly glycemic blood markers and insulin resistance. Such associations could be of public health importance; whereas sleep durations have appeared relatively stable in adulthood,[8] recent evidence suggests that average sleep duration has declined in children and adolescents over time (particularly over the last 15 years, during which actual sleep duration has declined by 0.73 minutes per year).[9] The ramifications of this reduced sleep duration on future health are yet to be established. However, if shorter sleep duration is related to emerging T2D risk in childhood, evidence on optimal sleep duration for protecting against T2D risk could underpin efforts to prevent T2D risk at an early stage.[10] Moreover, early ethnic differences in T2D precursors (particularly in South Asian children, who have a worse metabolic profile compared with children of white European ancestry[11]) might be partially explained by differences in sleep duration. If so, this may offer an additional strategy to reduce ethnic differences in T2D risk in early life.

We therefore examined the associations between self-reported sleep duration, T2D, and cardiovascular risk factors in a large-scale, multiethnic population-based study of 9- to 10-year-old children.

METHODS

The Child Heart and Health Study in England (CHASE) was a cross-sectional investigation of the cardiovascular and T2D risk profiles of UK primary school children aged 9 to 10 years of white European, South Asian, and black African-Caribbean origins. Ethical approval was obtained from the relevant multicenter research ethics committee. Full details of study methods have been published.[11] The study was based in 200 state primary schools in London, Birmingham, and Leicester, half with a high prevalence of UK South Asian children (stratified by Indian, Pakistani, and Bangladeshi origin) and half with a high prevalence of UK black African-Caribbean children (stratified by black African and black Caribbean origin). Informed parental consent and child assent were obtained.

Measurements of Body Composition

A single survey team of 3 trained research nurses made all the measurements between October 2004 and February 2007; to limit ethnic differences in observer bias, each observer measured approximately one-third of the children in each ethnic group. Height and weight were measured, and BMI was calculated as kilograms per meter squared. To provide an objective measure of adiposity, fat mass was determined from arm-to-leg bioelectrical impedance and measured on the right side using the Bodystat 1500 bioelectrical impedance monitor (Bodystat Ltd, Douglas, Isle of Man, UK). Fat mass was derived by using equations derived specifically for UK children of this age group, which were specific to sex and ethnic group.[12] Fat mass was height standardized (fat mass index = fat mass [kilograms]/height [in meters]^5). Skinfold thicknesses were also measured to provide a subcutaneous measure of adiposity at the biceps, triceps, and subcapsular and suprailiac locations. Skinfold thickness provides a better predictor of body fatness compared with weight-for-height measures. Seated blood pressure (BP) was measured twice in the right arm after a 5-minute rest using an Omron HEM-907 (Omron Electronics Ltd, Milton Keynes, UK) with the appropriate cuff size; the mean of the 2 values was used in analysis after an adjustment for cuff size.[13]

Blood Measurements

Blood samples were obtained after an overnight fast; children were asked not to eat on the morning of the examination, and those who reported having eaten breakfast were excluded from analysis. Serum for insulin assay was separated and frozen on dry ice immediately after collection. All other samples were shipped to a central laboratory within 48 hours. Insulin, glucose, hemoglobin A1c (HbA1c), and blood lipids were measured as described previously. Homeostasis model assessment equations were used to provide an estimate of insulin resistance.[14] Serum urate was assayed by using an enzymatic method.[15]

Questionnaire Data

Children were asked the following 2 questions about sleeping habits on a school day: "What time do you usually go to bed on schooldays?" and "What time do you usually get up in the morning on schooldays?" The difference between these 2 times was used to define sleep

duration. The ethnic origin of a child was based on self-defined parental ethnicity (which was coded using a classification similar to the 2001 UK census), if this was not available, parental report of the ethnic origin of the child (if self-defined parental ethnicity was not available), parental and grandparental places of birth as previously described.[11] In the present analyses, "white European" includes children whose ethnic origin was defined as "white British," "white Irish," "white European," or a combination of these and excludes "white other." "South Asian" includes "Indian," "Pakistani," "Bangladeshi," "Sri Lankan," or a combination of these. Remaining Asian children were classified as "Asian other" and included Asian mixed ethnicities, Chinese, and Middle Eastern ethnic groups. "Black African-Caribbean" includes "black African," "black Caribbean," "black British," "black other," or combination of these. Children of other ethnic groups and mixed ethnic origins (except Asian) were allocated to a separate "other" group. Parental socioeconomic position was based on self-reported parental occupation and coded by using UK National Statistics Socioeconomic Classification for the parent with the highest grade.[16] The 3-class version was used in all analyses (professional and managerial, intermediate, routine and manual) as previously described.[17] Self-reported pubertal status was measured in girls only by using the Tanner development score.[18]

Physical Activity Assessment

In a subset of children recruited from 79 schools in the latter phase of the study (January 2006–February 2007), objective assessment of physical activity was conducted by using a waist-worn accelerometer (ActiGraph GT1M; ActiGraph, LLC, Pensacola, FL). Details of the physical activity assessment have been published previously.[19] In brief, children were asked to wear the monitors (worn at the waist above the left hip using an elasticated belt) during waking hours for 7 whole days. On return of the instruments, a dedicated software program was used to determine activity outcomes (including steps by hour of the day), allowing activities before and after self-reported bedtimes to be examined. It also allowed a comparison of monitor nonwear time with reported sleep duration.

Statistical Analysis

Statistical analyses were conducted by using Stata/SE software (Stata 13 for Windows; Stata Corp LP, College Station, TX). All diabetes and cardiovascular risk markers and body composition variables were examined for normality and log transformed when necessary. Multilevel linear regression models adjusted for age in quartiles, sex, month, ethnicity, social position, and random effect for school were used to calculate adjusted means for measures of body composition and diabetes and/or cardiovascular risk markers by 5 categories of sleep duration (<9, 9–9.9, 10–10.9, 11–11.9, and ≥12 hours). Continuous linear associations with sleep duration in hours were determined by using the same adjustments; for outcomes that were log transformed, associations were quantified as the percentage difference per extra hour of sleep. Associations with sleep duration were also examined in boys and girls separately and by ethnicity. Effects of adjustment for pubertal status (girls only) were also explored. Linear associations between sleep duration, T2D, and cardiovascular risk markers that were statistically significant were further adjusted for measures of adiposity, including fat mass index and fat-free mass index. The contribution of sleep duration to previously reported ethnic differences in diabetes and cardiovascular risk markers[11,20] was also examined.

RESULTS

Of 8641 children invited to participate in CHASE, 5887 (68%) took part. Among 5681 singleton children, 4525 (80%) provided a fasting blood sample, had complete data for measures of body composition, and self-reported data for bedtime and getting up time on a school day. On average, children were 10.0 years old (SD 0.4 years, reference range 9.2–10.7 years). Sleep duration was on average 10.5 hours per night on a school day (95% central range 8.0–12.0 hours; Fig 1). In a subset of 1766 children who wore an accelerometer during waking hours, a mean of 600 steps per hour were recorded during the hours of 8 AM to 7 PM on a school day but only 54 steps in the hour after reported bedtime, compared with 234 steps in the hour before bedtime. One hour before wake-up time, the number of steps recorded was 0 compared with an average of 435 steps 1 hour after wake time. In the subset that wore an accelerometer, daily nonwear time was 10.2 hours, and reported sleep duration was 10.3 hours (equivalent to a mean difference of 7 minutes, 95% confidence interval [CI] 4 to 10 minutes).

Table 1 shows demographic characteristics of the children in relation to sleep duration on a school day. Children who had longer sleep durations were on average slightly younger and more likely to be girls (Table 1). Sleep duration differed marginally by ethnicity; white European children had the longest mean sleep duration, and black African-Caribbean children had the shortest. There was no clear evidence of any trend in sleep duration by parental social position.

There were strong inverse linear relationships between sleep

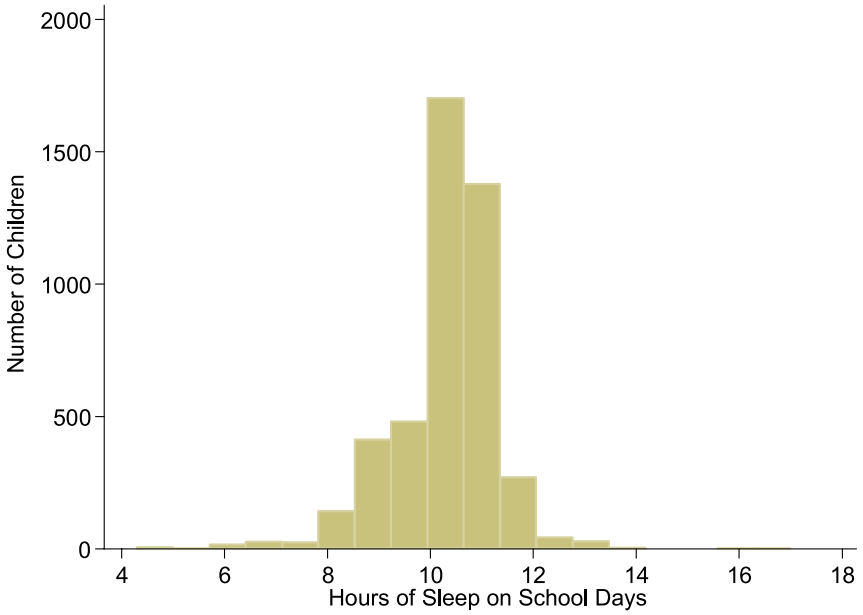

FIGURE 1
Distribution of hours of sleep on school days.

duration and all measures of body size and fatness (Fig 2, Table 2, and Supplemental Fig 3). Children who slept longer were on average shorter and had lower body weight, fat-free mass, levels of fat mass index, and skinfold thickness; effect sizes for adiposity measures (including BMI, sum of skinfolds, and leptin) were statistically significant and between 1% and 3% per extra hour of sleep. Sleep duration was also inversely related to insulin, insulin resistance, and glucose. Per extra hour of sleep, insulin levels were lower by nearly 3% (95% CI 1.2% to 4.5% reduction), and a similar association was seen for insulin resistance (Table 2). The size of the association with glucose was smaller, and there was no clear evidence of an association with HbA1c (Table 2). There was no evidence of associations with lipids or BP. Similar strengths of associations were found in analyses stratified by ethnicity and sex (Supplemental Table 4). There was no consistent evidence of effect modification by sex (likelihood ratio test for interaction with sex had a *P* value >.1 for all measures of body size and cardiometabolic risk markers) or ethnicity (likelihood ratio test had a *P* value >.2 for interaction in all instances).

In addition to the factors adjusted for in Table 2, Table 3 shows that associations with insulin and insulin resistance were slightly attenuated, with further adjustment for height (Table 3; model 1). Additional adjustments for fat mass index (model 2), fat-free mass index (model 3), or both (model 4) attenuated the associations further for insulin and insulin resistance and were no longer statistically significant after an adjustment for fat-free mass index. In the fully adjusted model 4, coefficients were approximately halved when compared with coefficients in Table 2

TABLE 1 Demographic Characteristics for 4525 Children by Duration of Sleep With Adjusted Average Sleep Durations

	Hours of Self-reported Sleep on a School Night[a]						
	<9 h	9–9.9 h	10–10.9 h	11–11.9 h	≥12 h	Adjusted Mean Sleep Hours (95 % CI)	*P*[b]
N	279 (6.2%)	841 (18.6%)	2014 (44.5%)	1166 (25.8%)	225 (5%)		
Average age, y (SD)	10.0 (0.4)	10.0 (0.4)	10.0 (0.4)	9.0 (0.4)	9.8 (0.4)	10.3 (10.2 to 10.3)	.05
Boys	180 (8.2)	431 (19.7)	963 (44.1)	501 (22.9)	110 (5.0)	10.2 (10.1 to 10.2)	—
Girls	99 (4.2)	410 (17.5)	1051 (44.9)	665 (28.4)	115 (4.9)	10.4 (10.3 to 10.4)	<.0001
Ethnic group							
White European	43 (3.9)	179 (16.4)	493 (45.3)	316 (29.0)	58 (5.3)	10.4 (10.3 to 10.5)	<.0001
Black African-Caribbean	87 (7.6)	245 (21.4)	508 (44.4)	252 (22.0)	51 (4.5)	10.1 (10.1 to 10.2)	—
South Asian	89 (7.2)	217 (17.5)	551 (44.4)	335 (27.0)	50 (4.0)	10.2 (10.2 to 10.3)	—
Asian other	17 (6.1)	56 (20.1)	121 (43.5)	71 (25.5)	13 (4.7)	10.2 (10.1 to 10.3)	—
Other	43 (5.6)	144 (18.6)	341 (44.1)	192 (24.8)	53 (6.9)	10.3 (10.2 to 10.4)	—
Parental socioeconomic position							
Managerial, professional	55 (4.3)	220 (17.4)	629 (49.6)	316 (24.9)	47 (3.7)	10.3 (10.2 to 10.4)	.04
Intermediate	75 (6.5)	239 (20.6)	511 (44.1)	290 (25.0)	45 (3.9)	10.2 (10.1 to 10.3)	—
Routine, manual	96 (6.9)	254 (18.3)	586 (42.3)	361 (26.1)	88 (6.4)	10.3 (10.2 to 10.3)	—
Economically inactive	33 (7.2)	77 (16.9)	171 (37.5)	143 (31.4)	32 (7.0)	10.3 (10.2 to 10.4)	—
Unclassified or missing	20 (7.8)	51 (19.8)	117 (45.5)	56 (21.8)	13 (5.1)	10.2 (10.1 to 10.3)	—

[a] Values are mean and SD for age; for all categorical variables, values are *N* (row %).
[b] *P* value from likelihood ratio test for heterogeneity is from the same multilevel model.

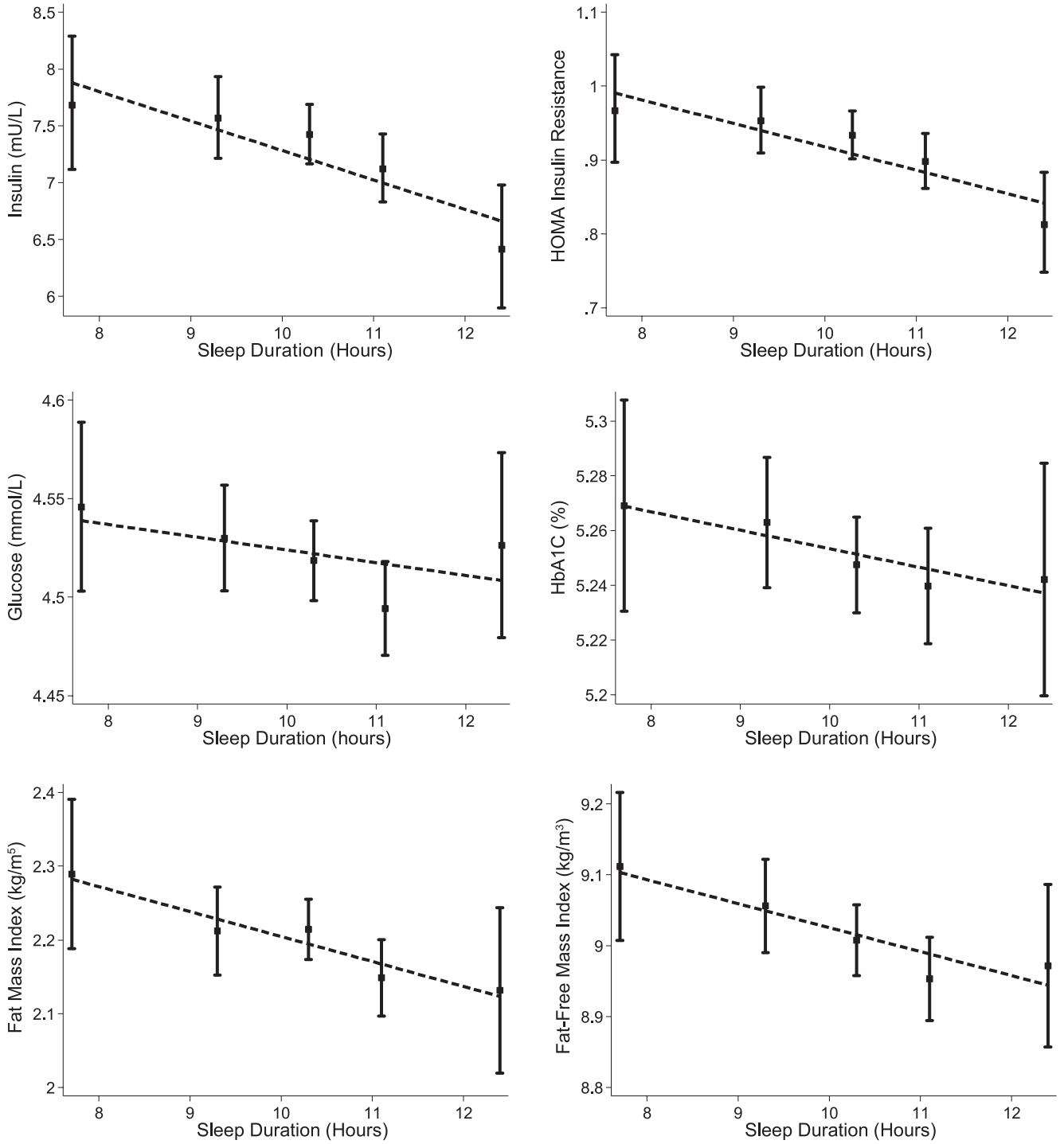

FIGURE 2

Adjusted means for T2D risk markers and body fatness indices by sleep duration. Means are adjusted for sex, age quartile, month, ethnicity, social position, observer (physical measures only), and random effect for school. HOMA, homeostasis model assessment.

that did not adjust for height or adiposity. Associations between sleep duration and glucose were little affected by adjustment for height or body fatness. Adjustment for pubertal status (recorded in girls only) also made no difference to the findings, as did the exclusion of girls who had entered puberty. Physical activity was recorded objectively over a week in a subset of children (n = 1492) by using Actigraph monitors. Adjustment for the average level of physical activity on a school day did not alter the results, but there was a loss in power because of the analyses being based on reduced numbers.

TABLE 2 Adjusted Mean Values of T2D and Cardiovascular Risk Markers and Difference in Risk Markers Per Hour Increase in Sleep

	Adjusted Means (95% CI)						
	Hours of Self-reported Sleep on a School Night						
	<9 h	9–9.9 h	10–10.9 h	11–11.9 h	≥12 h	Difference per h of Sleep (95% CI)[a]	*P*[b]
Body size	279	841	2014	1166	225		
Height (cm)	140.1 (139.3 to 140.9)	141.0 (140.6 to 141.5)	140.2 (139.9 to 140.5)	140.0 (139.6 to 140.4)	139.0 (138.2 to 139.9)	−0.26 (−0.44 to −0.08)	.005
Weight (kg)	37.8 (36.7 to 38.9)	38.2 (37.6 to 38.8)	37.2 (36.8 to 37.6)	36.5 (36.0 to 37.0)	35.4 (34.2 to 36.6)	−0.51 (−0.76 to −0.26)	<.0001
BMI	19.1 (18.7 to 19.5)	19.0 (18.8 to 19.3)	18.7 (18.6 to 18.9)	18.4 (18.2 to 18.6)	18.2 (17.7 to 18.6)	−0.19 (−0.28 to −0.09)	<.0001
Sum skinfolds (mm)[c]	42.0 (39.6 to 44.5)	42.8 (41.3 to 44.2)	41.2 (40.3 to 42.1)	39.9 (38.8 to 41.1)	38.8 (36.4 to 41.3)	−1.79 (−3.08 to −0.49)	.007
Fat mass (kg)	12.6 (12.0 to 13.3)	12.7 (12.3 to 13.1)	12.3 (12.0 to 12.5)	11.8 (11.5 to 12.2)	11.2 (10.5 to 12.0)	−0.27 (−0.42 to −0.11)	.001
Fat-free mass (kg)	25.2 (24.7 to 25.7)	25.5 (25.2 to 25.8)	25.0 (24.8 to 25.2)	24.7 (24.5 to 24.9)	24.2 (23.7 to 24.8)	−0.24 (−0.35 to −0.13)	<.0001
Fat mass index (kg/m^5)	2.29 (2.19 to 2.39)	2.21 (2.15 to 2.27)	2.21 (2.17 to 2.26)	2.15 (2.10 to 2.20)	2.13 (2.02 to 2.24)	−0.03 (−0.05 to 0.00)	.03
Fat-free mass index (kg/m^3)	9.11 (9.01 to 9.22)	9.06 (8.99 to 9.12)	9.01 (8.96 to 9.06)	8.95 (8.89 to 9.01)	8.97 (8.86 to 9.09)	−0.04 (−0.06 to −0.01)	.002
Cardiometabolic risk markers							
Insulin (mU/L)[c,d]	7.68 (7.12 to 8.29)	7.57 (7.22 to 7.93)	7.42 (7.17 to 7.69)	7.12 (6.83 to 7.43)	6.42 (5.90 to 6.98)	−2.88 (−4.50 to −1.23)	.001
Insulin resistance[c,d]	0.97 (0.90 to 1.04)	0.95 (0.91 to 1.00)	0.93 (0.90 to 0.97)	0.90 (0.86 to 0.94)	0.81 (0.75 to 0.88)	−2.81 (−4.41 to −1.17)	.001
Glucose (mmol/L)[c]	4.55 (4.50 to 4.59)	4.53 (4.50 to 4.56)	4.52 (4.50 to 4.54)	4.49 (4.47 to 4.52)	4.53 (4.48 to 4.57)	−0.24 (−0.44 to −0.03)	.03
HbA1c (%)	5.27 (5.23 to 5.31)	5.26 (5.24 to 5.29)	5.25 (5.23 to 5.26)	5.24 (5.22 to 5.26)	5.24 (5.20 to 5.28)	−0.01 (−0.02 to 0.00)	.11
HbA1c (mmol/mol)	34 (34 to 35)	34 (34 to 34)	34 (34 to 34)	34 (34 to 34)	34 (34 to 34)		.11
Leptin (ng/mL)[c,d]	9.2 (8.2 to 10.3)	9.7 (9.1 to 10.4)	9.5 (9.1 to 9.9)	8.6 (8.1 to 9.1)	8.0 (7.1 to 9.0)	−3.03 (−5.42 to −0.59)	.02
Total cholesterol (mmol/L)	4.6 (4.5 to 4.7)	4.6 (4.5 to 4.6)	4.6 (4.5 to 4.6)	4.6 (4.5 to 4.6)	4.5 (4.4 to 4.6)	−0.01 (−0.03 to 0.01)	.32
LDL cholesterol (mmol/L)	2.7 (2.6 to 2.8)	2.7 (2.6 to 2.7)	2.7 (2.6 to 2.7)	2.7 (2.7 to 2.7)	2.7 (2.6 to 2.8)	−0.01 (−0.02 to 0.01)	.57
HDL cholesterol (mmol/L)	1.5 (1.5 to 1.5)	1.5 (1.5 to 1.5)	1.5 (1.5 to 1.5)	1.5 (1.5 to 1.5)	1.5 (1.5 to 1.5)	0.00 (−0.01 to 0.01)	.70
Triglyceride (mmol/L)[c]	0.8 (0.8 to 0.8)	0.8 (0.8 to 0.8)	0.8 (0.8 to 0.8)	0.8 (0.8 to 0.8)	0.8 (0.7 to 0.8)	−0.35 (−1.37 to 0.67)	.50
Urate (mmol/L)[c]	0.2 (0.2 to 0.2)	0.2 (0.2 to 0.2)	0.2 (0.2 to 0.2)	0.2 (0.2 to 0.2)	0.2 (0.2 to 0.2)	0.20 (−0.48 to 0.89)	.56
Systolic BP (mm Hg)	104.1 (102.9 to 105.4)	105.1 (104.3 to 105.8)	104.9 (104.4 to 105.4)	104.5 (103.8 to 105.1)	104.3 (102.9 to 105.7)	0.03 (−0.25 to 0.31)	.84
Diastolic BP (mm Hg)	62.9 (61.8 to 64.1)	63.5 (62.8 to 64.2)	63.0 (62.4 to 63.5)	62.8 (62.2 to 63.4)	62.4 (61.2 to 63.6)	0.00 (−0.25 to 0.25)	.97

Means of outcome variables by sleep category from multilevel model are adjusted for sex, age quartile, month, ethnicity, social position, observer (physical measures only), and random effect for school.

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

[a] Regression coefficients from the multilevel model for continuous association with hours of sleep are adjusted for sex, age quartile, month, ethnicity, social position, observer (physical measures only), and random effect for school. For log-transformed outcomes, these represent percent differences.

[b] *P* values for regression coefficients from the multilevel model assume a linear continuous association with sleep duration.

[c] Geometric means for log-transformed variables.

[d] Insulin and insulin resistance data are missing for 76 children; leptin data are missing for 97 children.

We examined ethnic differences in T2D precursors before and after adjusting for sleep duration. Compared with white Europeans, black African-Caribbean children had 8.5% higher levels of insulin (95% CI 3.2% to 14.0%) after adjustment for age quartiles, sex, height, month, and social position; this difference reduced to 7.6% higher (95% CI 2.3% to 13.3%) with an additional adjustment for sleep duration. In South Asian children, the difference in insulin levels compared with white European children was 30.3% (95% CI 23.9% to 36.9%) before adjustment for sleep duration and 29.5% (95% CI 23.3% to 36.3%) after adjustment for sleep duration.

TABLE 3 Adjusted Difference in T2D Risk Markers Per Hour Increase in Sleep

	% Difference in Metabolic Risk Marker per 1-h Increase in Sleep (95% CI)							
Model	Insulin	*P*	Insulin Resistance	*P*	Glucose	*P*	HbA1c	*P*
1	−2.22 (−3.77 to −0.65)	.006	−2.16 (−3.70 to −0.60)	.007	−0.21 (−0.42 to −0.01)	.04	−0.01 (−0.01 to 0.00)	.14
2	−1.49 (−2.89 to −0.07)	.04	−2.16 (−3.70 to −0.60)	.007	−0.22 (−0.42 to −0.01)	.04	0.00 (0.00 to 0.00)	.31
3	−1.09 (−2.52 to 0.37)	.14	−1.04 (−2.47 to 0.41)	.16	−0.19 (−0.39 to 0.02)	.08	−0.01 (−0.01 to 0.00)	.20
4	−1.15 (−2.54 to 0.26)	.11	−1.04 (−2.47 to 0.41)	.16	−0.20 (−0.41 to 0.00)	.05	−0.01 (−0.01 to 0.00)	.14

Model 1 includes adjustments for sex, age quartile, month, ethnicity, social position, anthropometric observer, height, and random effect for school. Model 2 is the same as model 1 plus fat mass index. Model 3 is the same as model 1 plus fat-free mass index. Model 4 is the same as model 1 plus fat mass index and fat-free mass index. Insulin and insulin resistance data are missing for 76 children.

DISCUSSION

Main Findings

The current study is novel in showing inverse associations between reported sleep duration and T2D risk factors in early life, which are independent of adiposity and observed in different ethnicities. It also shows strong inverse associations between reported sleep duration and adiposity (including detailed measures of body fatness), confirming findings from earlier studies. Given the rising prevalence of diabetes worldwide and especially in low- to middle-income countries, we believe our findings will help motivate further simple, pragmatic trials in this area.

Relation to Earlier Studies

Both short and long sleep durations have been linked to adiposity and T2D in adulthood.[4–6] Previous cross-sectional observations in childhood have shown an inverse association between sleep duration, levels of BMI, and obesity (with 0.75 lower BMI for every additional hour of sleep[21]), and pooled estimates suggest short durations of sleep are associated with a near doubling of obesity prevalence compared with long durations.[5,22] These associations have also been confirmed in an analysis of prospective studies in childhood, which have shown slightly smaller associations, with 0.5 lower BMI per hour of sleep.[23] These observations concur with adult observations, in which pooled findings suggest inverse associations between sleep duration, BMI, and obesity.[5] The current study confirms the inverse association with BMI and extends these observations by demonstrating strong graded inverse associations with more detailed measures of adiposity, including sum of skinfolds and bioelectrical impedance-derived measures of fat mass. Inverse associations between height and sleep duration may well be explained by maturation, with more mature children tending to have later weekday bedtimes. The inverse association with leptin is of particular interest because this may allude to a biological mechanism of effect by which increased sleep may alter appetite by downregulation of leptin production,[24] although we accept that circulating leptin levels generally reflect fat mass in stable-weight individuals. This agrees with earlier work suggesting that short sleep durations in childhood are associated with a higher intake of energy-dense and sugary foods.[25]

The current study is also novel in demonstrating inverse associations between reported sleep duration and T2D risk markers, including insulin, insulin resistance, and blood glucose (although associations with HbA1c were marginal). These associations appear to be partially independent of the detailed adiposity measures. In one previous, small study (with a sample size of 245 participants), Matthews et al[26] showed an inverse association between sleep duration and homeostasis model assessment measures of insulin resistance in adolescence. However, we are not aware of any other population-based studies that have reported similar associations earlier in childhood except among high-risk obese children.[27] Sleep duration did not appear to explain early ethnic differences in T2D risk that were previously reported, particularly the higher levels of insulin resistance and glycemia among South Asian children.[11]

The current study also provided the opportunity to examine sleep durations and cardiovascular risk factors (including blood lipids and BP) when there was no consistent evidence of an association. These null findings are in agreement with a limited number of earlier observations in childhood,[28,29] suggesting that sleep duration does not alter other cardiovascular risks in early life other than by increasing obesity and metabolic risks, which, if sustained or accentuated, take time to accelerate cardiovascular risks.

Strengths and Limitations

The current study has a number of strengths and limitations that require further consideration. The study was large and included a multiethnic child population. Although ethnic differences in sleep duration were observed (white European children slept the longest and black African children the shortest), associations with adiposity measures and T2D risk were consistent across ethnic groups. Moreover, associations were

not materially altered by adjustments for socioeconomic position and pubertal status (in girls only), and they remained after adjustments for other potential confounders (ie, sleep-T2D associations remained after an adjustment for measures of adiposity). The study was cross sectional, which should not be judged as a disadvantage given the plausible short-term effects of sleep duration on cardiometabolic risk. The possibility of reverse causality, in which metabolic dysregulation alters sleep patterns, seems unlikely given the child population.[30] Reassuringly, findings were consistent with observations from longitudinal studies as outlined above. One potential weakness was that sleep duration was derived from weekday self-reported bedtimes and not weekend reported bedtimes, which may underestimate total weekly sleep durations.[31] However, the reported mean and distribution of sleep time were in keeping with other studies of similarly aged children (ie, mean 10.3, SD 1.1 hours versus mean 10.5, SD 0.7 hours in a large cohort of children aged 9 years).[32] Accelerometer assessment in a subset[19] provided further validation of sleep time by showing similar monitor nonwear time and reported sleep duration and that, on average, children recorded few steps 1 hour before compared with 1 hour after reported wake time and far fewer steps 1 hour after compared with 1 before reported bedtime. However, a reduction or absence of steps may not fully reflect sleep, only rest, because other sedentary activities could be taking place. Moreover, waking time accelerometry (as opposed to sleep-time accelerometry) does not allow sleep quality to be assessed, which could plausibly exert metabolic effects.[33]

Biological mechanisms by which sleep may alter T2D risk have been proposed, including the dysregulation of neuroendocrine control of appetite.[24,34] This lends weight to a potentially causal association. If the association is indeed causal, it would be important to establish evidence-based sleep-time recommendations, which would be particularly relevant given trends toward decreasing sleep time in contemporary children.[9] However, robust experimental evidence (ie, trials) of the association between sleep duration and T2D risk is needed before causality can be inferred. Unfortunately, evidence from a small number of experimental studies in childhood and adulthood examining the effects of changing sleep duration on adiposity has so far been inconclusive largely because the effects of interventions on sleep duration have been modest.[35,36] Hence, interventions that are more effective in altering sleep duration are needed. Establishing causality is a priority because increasing sleep duration could offer a simple, cost-effective approach to reducing adiposity and T2D risk from early life.

CONCLUSIONS

In the current study, increasing the mean weekday sleep duration (10.5 hours) by half an hour could be associated with 0.1 lower BMI and a 0.5% reduction in insulin resistance. These differences should be considered in relation to the following, which is the largest observed ethnic difference in BMI and insulin resistance within this study population: children of South Asian origin had 30% higher insulin resistance[11] and 0.4 lower BMI compared with children of white European ancestry.[37] If experimental evidence were to corroborate the associations observed between sleep duration and T2D precursors (allowing a causal association to be inferred), these effects could plausibly persist into later life. Levels of insulin resistance in childhood have been shown to impact T2D risk over a 10-year period and may magnify with increasing age.[38] Hence, reducing levels even by modest amounts in childhood may have longer-term implications for reduced T2D in later life.[39] Furthermore, greater weight gain trajectories in childhood are associated with greater risks for adolescent nonalcoholic fatty liver disease, which is a well-accepted precursor to diabetes risks.[40]

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ABBREVIATIONS

BP: blood pressure
CHASE: the Child Heart and Health Study in England
CI: confidence interval
HbA1c: hemoglobin A1c
T2D: type 2 diabetes

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