1. **Title:** Cross-sectional and prospective associations of sleep duration and bedtimes with adiposity and obesity risk in 15,810 youth from 11 international cohorts
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**Abstract**

**Objectives:** To investigate associations of bedtimes and sleep durations with adiposity levels in children and adolescents. **Methods:** Individual data were pooled for 12,247 children (5819 with follow-up adiposity at 2.3 ± 1.4 years post-baseline) and 3563 adolescents from 11 international studies. We investigated associations between questionnaire-based sleep durations, bedtimes, and four groups of combined bedtimes and sleep lengths (later-shorter (reference) / earlier-shorter / later-longer / earlier-longer) with measured adiposity (body mass index (BMI) and waist circumference *z*-scores) and weight status. **Results:** In children, longer sleep durations were consistently associated with lower adiposity markers and earlier bedtimes were related to lower BMI *z*-score. Compared to sleeping <10h, longer baseline sleep favourably predicted Δwaist *z*-score in girls (≥10h & <11h (β-coefficient (95% confidence interval (CI))): -0.06 (-0.12 to -0.01)) and boys (≥11h: -0.10 (-0.18 to -0.01)). Combined groups that were defined by longer sleep (later-longer and earlier-longer sleep patterns) were associated with lower adiposity in children, and later-longer sleep favourably predicted Δwaist *z*-score in girls (-0.09 (-0.15 to -0.02)). In adolescents, longer sleep durations and earlier bedtimes were associated with lower BMI *z*-score, and also with lower waist *z*-score in boys. Combined groups characterised by earlier bedtimes were associated with the same outcomes, for instance, earlier-shorter (-0.22 (-0.43 to -0.01) and earlier-longer (-0.16 (-0.25 to -0.06) sleep were both associated with lower BMI *z*-score. **Conclusions:** If the associations are causal, longer sleep duration and earlier bedtimes should be targeted for obesity prevention, emphasising longer sleep for children and earlier bedtimes for adolescents.

**Introduction**

Cross-sectional studies, and meta-analyses of prospective studies, report that longer sleep duration is associated with lower adiposity and obesity risk in youth.1–3 There is considerable heterogeneity in the results of individual prospective studies, however, and investigations rarely include adiposity markers other than weight-for-height indices, even though centrally stored adiposity is metabolically more harmful than total adiposity.4 In addition, most investigations continue to solely focus on sleep duration. Sleep is a multidimensional construct of partly overlapping dimensions, including sleep duration, timing, quality, and variability. Investigation of diverse sleep parameters and their links with health are needed.5

There is some inconsistency in the literature, but an increasing number of cross-sectional studies6,7 and a small but emerging prospective evidence-base8–11, have recently reported that later bedtimes are associated with higher adiposity and increased overweight and obesity in youth. Some studies even suggest that the associations are distinct from sleep duration.12–14 This information could be pivotal in terms of refining obesity prevention efforts. It may also have important implications for sleep recommendations, which have historically focussed on duration as opposed to any other sleep parameter.15 More studies are required to investigate sleep dimensions simultaneously, but it is challenging to investigate the independent or interactive effects of bedtimes and sleep durations, because they are often highly correlated. This is particularly true in school-aged youth, the majority of whom will wake-up within a narrow set of times on weekday mornings to attend school, thus bedtimes largely dictate variation in sleep duration. Reassuringly, this issue can be overcome in large cohorts of children, if the number of individuals with atypical behaviours is sufficient to allow robust estimation of closely related exposures with outcomes.16 As an example, Olds and colleagues conducted a cross-sectional analysis of 2200 9- to 16-year-olds from Australia who were grouped into four sleep-wake groups. Relative to a late-bed/late-rise sleep pattern, an early-bed/early-rise pattern (which was characterised by an approximately equal sleep duration but 86 minutes earlier bedtime) was associated with lower BMI *z*-score and lower likelihood of overweight and obesity.12 Similar studies are warranted, as are prospective investigations of sleep durations and bedtimes to quantify the influence of both dimensions on youth adiposity over time.6

It is important to shed light on the possible pathways by which sleep parameters may be related to adiposity. The best available evidence remains of low quality, but later bedtimes and short sleep are hypothesised to create more opportunities for sedentary time, including screen-time such as TV viewing, and fatigue-induced reductions in daily physical activity.17 Previous studies have either failed to consider the role of awake-time movement behaviours, or have typically estimated activity metrics using data from questionnaires1–3, which are prone to bias and random error.18 Providing additional evidence to support one or more of the hypothesised pathways could help to ameliorate the consequences of poor sleep by identifying modifiable intermediate behaviours that can be targeted for mitigation.

The International Children´s Accelerometry Database (ICAD) provides a unique opportunity to analyse sleep-adiposity associations across a large and heterogeneous international dataset of children and adolescents. The dataset also benefits from device-measured estimates of habitual sedentary time and physical activity.19 We examined the cross-sectional and prospective associations of sleep duration and bedtimes with total adiposity, central adiposity, and overweight and obesity in ICAD. We also investigated combinations of behaviours and scrutinised associations when adjusted for device-measured sedentary time and physical activity, and reported TV viewing.

***Methods***

*Study design and participants*

This study is based on the second release of ICAD, a repository of harmonised data from 21 studies that assessed sedentary time and physical activity by waist-worn accelerometers in children aged 3 to 18 years.19,20 Eleven contributing studies, from 6 European countries (UK, Switzerland, Denmark, Estonia, Norway, Portugal) and Australia, also collected information about sleep duration and/or bedtimes and were included in this study.21–28 With the exception of two cluster randomised controlled trials24,25 (from which only control participant data, or pre-intervention data from intervention-arm participants, were used) all studies were observational. Extensive descriptions of all source data and the harmonisation procedures for study variables can be accessed via the ICAD website (<http://www.mrc-epid.cam.ac.uk/research/studies/icad/data-harmonisation/>).

*Sleep duration and timing*

Five studies21,24,25,27,28 used proxy-reports by parents to capture sleep parameters (including all studies that involved young children - who would have struggled to provide accurate self-reported information), otherwise sleep data were self-reported.22,23,26 It has previously been shown that self- and parent-reported sleep durations are equally valid in 9-17y olds, and that data from both sources agree substantially with polysomnography, which is the gold-standard for sleep assessment.29 Seven studies collected information about the time that children and adolescents usually went to bed and got out of bed22,24–26 and two studies asked when participants usually went to sleep and woke up.21,23 Four studies collected free-text (hour and minute) responses21,23–25and five studies offered a choice of categories.22,26 Two studies collected free-text responses to the question ‘How many hours per night does your child *usually* sleep?’.27,28 The current analyses are based on weekday data only, which was collected by all studies (only five collected information about weekends21–25), and is considered to be more indicative of habitual sleep behaviours in school-aged youth because of set school day routines.30 Due to the broad age range in ICAD, and because there appear to be age-related differences in the associations of sleep with adiposity1–3, study participants were categorised as children (<12 years) or adolescents (≥12 years). Data from all studies were harmonised to create categorical variables for bedtime (after 22:00 / between 21:00 to 22:00 / before 21:00) and age-group specific sleep durations (children: <10h / ≥10h & <11h / ≥11h; adolescents: <9h / ≥9 & <10h / ≥10h). Bedtime and sleep duration categories were further collapsed to age-specific categories signifying earlier (children: before 21:00; adolescents: before 22:00) and later bedtimes (children: 21:00 or later; adolescents: 22:00 or later), and shorter (children: <10h; adolescents: <9h) and longer sleep durations (children: ≥10h; adolescents: ≥9h). These data were combined to create four age-group specific combinations of bedtimes and sleep duration (later-shorter / earlier-shorter / later-longer / earlier-longer).

*Adiposity markers*

In each study, trained personnel measured height and weight using standardised techniques and calibrated equipment21–28 and the resulting data were used to calculate body mass index (BMI, kg/m2), which was converted to *z*-scores and weight status categories.31 In all European studies (*n*=9), waist circumferences were measured at the end of gentle expiration, with an anthropometric tape placed at the midpoint between the lowest rib margin and the iliac crest.21–26 Waist circumferences were also converted to *z*-scores.32 Eight studies collected follow-up data which were used to calculate changes in adiposity (ΔBMI and ΔWaist *z*-scores) by subtracting follow-up from baseline values.21–27

*Covariables*

All studies collected information about putative confounders or mediators including participant age, sex, parental education, sedentary time, and physical activity. Parental education was based on maternal education (or paternal qualifications given missing data) and was harmonised to three categories (School / College / University). Sedentary time and physical activity were assessed with uniaxial, waist-mounted Actigraph (Pensacola, USA) accelerometers that were deployed for 4-7 consecutive days. Raw accelerometer files were collated and reprocessed centrally using commercially available software (KineSoft v3.3.80, Loughborough, UK).19 Files were initially reintegrated to 60s epochs and non-wear periods were identified and excluded by scanning the data array for periods of consecutive zeros ≥60 minutes, allowing for 2 minutes of non-zero interruptions. To account for children and adolescents who wore monitors overnight whilst asleep, all registered data between midnight and participant-specific getting out of bed times, were discarded. If participant-specific getting out of bed times were missing, then group-level times which represented the average time of getting out of bed for a given study, age and sex, were used. Validated cut-points were incorporated to estimate the average daily time that children spent sedentary, in light intensity physical activity and moderate-to-vigorous physical activity (MVPA) on weekdays.33 All days with ≥500 minutes of monitor wear time were considered valid, and participants were required to provide ≥1 valid weekday of data. Seven studies gathered information about child ethnicity. Parents either reported their own ethnicity which was used a proxy21 or their child’s ethnicity23,26, and in one study ethnicity was determined by visual inspection by the research team.22 The data were harmonised to two categories (white / other). Maternal height and weight were self-reported in all but one study22 and the data were used to calculate maternal BMI (kg/m2). Sexual maturity was collected in eight studies, via Tanner staging by researchers26, or by parent- and/or self-assessment.21,22,25,27 For this investigation, pubertal status was based on breast development for girls and pubic hair for boys, and children aged ≤6 years with missing data were assigned a Tanner score of 1. Tanner stages were collapsed to three groups (pre-puberty (Tanner score 1) / in puberty (scores 2 and 3) / completing puberty (scores 4 and 5).34 Eight studies collected parent- or self- reported information about whether participants had a TV set in their bedroom and the amount of daily time spent watching TV (including DVDs and videos).22,23,26–28

*Statistics*

Multilevel linear and logistic regression models, with random intercepts to accommodate study-level clustering of data, were used to investigate associations of sleep durations (reference: children: <10h; adolescents: <9h), bedtimes (reference: after 22:00), and combinations of behaviours (reference: later-shorter) with adiposity markers and weight status. Models were initially adjusted for age and sex (model A) and subsequently also for sedentary time and MVPA (model B). Likelihood ratio tests (LRT) were used to identify improved fit (*p*<0.05) if models were specified with random study-level slopes or sex-by-exposure interactions. If model fit was improved by including a sex-by-exposure interaction term, the model was stratified by sex. Models were also stratified by sex if for any level of an exposure category there was some evidence for a sex-by-exposure interaction (Wald test *p*<0.1). Additional adjustments were made in turn for parental education, ethnicity, pubertal status, maternal BMI, and TV viewing duration and having a bedroom TV. These extra models were framed as sensitivity analyses as they involved smaller analytical samples due to missing data. The analysis was also replicated to include adjustment for total active minutes (the sum of light and MVPA minutes) rather than solely MVPA. All analyses were *a priori* stratified by children and adolescents. Prospective associations between baseline sleep duration and bedtimes with subsequent changes in adiposity (ΔBMI and Δwaist *z*-scores from baseline to follow-up) were quantifiable only in children due to limited longitudinal data for adolescents. Longitudinal models were specified as described above but were consistently adjusted for baseline age, follow-up time, and the baseline value of the outcome variable. All analyses were performed with Stata/SE 17.1 software (StataCorp, College Station, TX). No corrections were made for multiple comparisons but exact *p*-values are reported. Readers are encouraged to focus on patterns of results, and on the range of plausible values of associations, as indicated by confidence intervals.35

**Results**

*Descriptive characteristics*

The characteristics of included participants are shown in **Table 1**. The sample was predominantly white and more than one-quarter of participants were overweight or obese. The prevalent bedtimes were between 21:00 to 22:00 and after 22:00 in children (age range: 3.8 to 11.9 years) and adolescents (12.0 to 18.4 years), respectively. The average sleep durations across combined bedtime and sleep length categories were: later-shorter (children: 9.2h; adolescents: 8.3h), earlier-shorter (children: 9.7h; adolescents: 8.7h), later-longer (children: 10.2h; adolescents: 9.3h), and earlier-longer (children: 10.8h; adolescents: 9.9h). Tables S1-S2 provide individual cohort characteristics. In total 5819 children, who are described in **Table 2**, contributed to prospective analyses. Children were followed-up over a mean of 2.3 (range: 0.5 to 8.0) years.

*Cross-sectional associations with adiposity and weight status in children and adolescents*

Models A (available on request) and B consistently yielded comparable results, highlighting that adjusting for sedentary time and MVPA did not influence any associations. With regard to total adiposity, **Table 3** shows that longer sleep durations and earlier bedtimes were dose-dependently associated with lower BMI *z*-score in both age groups. **Figure 1** illustrates that longer sleep durations were associated with lower odds of overweight and obesity in children (≥10h & <11h: 0.87 (0.80 to 0.95); *p*=0.003; ≥11h: 0.72 (0.63 to 0.83); *p*<0.001) and adolescents (≥10h: 0.76 (0.61 to 0.95); *p*=0.018). Earlier bedtimes were also dose-dependently associated with lower odds of overweight and obesity in children (21:00 to 22:00: 0.88 (0.77 to 0.99); *p*=0.047; before 21:00: 0.68 (0.59 to 0.80); *p*<0.001). With regard to waist *z*-score, Table 3 further shows that longer sleep durations were dose-dependently associated with lower values in children, and there was some evidence that bedtimes before 21:00 were associated with lower waist *z*-score in boys. An association of earlier bedtimes with lower waist *z*-score in girls was entirely attenuated when adjusted for pubertal status (see Table S3). In adolescents, longer sleep durations and earlier bedtimes were associated with lower waist *z*-score in boys, but not girls.

Results of the combined analyses are shown in **Table 4**. In children, relative to a later-shorter sleep pattern, later-longer sleep was associated with lower BMI *z*-score, and with lower waist *z*-score in boys. The results were most consistent and strongest for earlier-longer sleep, which was associated with lower values of all adiposity markers in boys and girls, and, as illustrated in Figure 1, also with lower odds of overweight and obesity (0.72 (0.64 to 0.82; *p*<0.001). In adolescents, earlier-shorter and earlier-longer sleep were both associated with lower BMI *z*-score, and also with lower waist *z*-score in boys.

There were no differences in any associations when they were adjusted for total active minutes instead of MVPA, and there was no evidence for confounding by parental education, ethnicity, or maternal BMI (results available on request). Associations in children were generally weaker when adjusted for pubertal status (Tables S3-S6), TV viewing and having a TV in the bedroom (Tables S7-S10). Full results of the logistic regression analyses are shown in Tables S11-12.

*Prospective associations with changes in adiposity from baseline to follow-up in children*

Tables S13-S14 provide study-level characteristics for participants who contributed to prospective analyses. Without exception, models A (results available on request) and B produced comparable results, indicating that adjusting for baseline sedentary time and MVPA did not influence any associations. **Table 5** shows that compared to a baseline sleep duration of <10h, sleeping between ≥10h and <11h in girls, and sleeping for ≥11h in boys, favourably predicted Δwaist *z*-score over follow-up. Relative to a bedtime after 22:00, a baseline bedtime between 21:00 to 22:00 favourably predicted Δwaist *z*-score and ΔBMI *z*-score. Results of the combined analyses revealed that a later-longer sleep pattern at baseline favourably predicted Δwaist *z*-score in girls. There were no differences in any associations when adjusted for total active minutes at baseline instead of MVPA, and there was no evidence for confounding by parental education, ethnicity, maternal BMI, pubertal status, or baseline TV viewing and having a TV in the bedroom (results available on request).

**Discussion**

In this pooled analysis of individual data from 11 international studies we replicated the consistent cross-sectional finding that longer sleep durations are associated with lower adiposity and weight status in children and adolescents.1–3 We further discovered that earlier bedtimes were associated with lower total adiposity, lower odds of overweight and obesity in both sexes, and with lower central adiposity in boys. It was also possible in this large-scale analysis to determine that whilst earlier-longer and later-longer sleep patterns were cross-sectionally associated with lower adiposity in children, earlier-shorter and earlier-longer patterns were seemingly optimal for adolescents. The reported associations were independent of covariates including device-measured sedentary time and physical activity. However, there was some indication that TV viewing and having a TV in the bedroom, exerted a partial confounding or mediating effect upon the cross-sectional results reported in children. In prospective analyses (conducted in children only), a longer baseline sleep duration and a bedtime between 21:00 to 22:00, both favourably predicted changes in adiposity over follow-up. A later-longer sleep pattern was favourably associated with Δwaist *z*-score in girls.

There is considerable evidence for associations between short sleep duration with higher expressions of BMI and obesity in youth.1–3 In this study, relative to the shortest sleep durations (children: <10h; adolescents: <9h), the longest sleep lengths (children: ≥11h; adolescents: ≥10h) were associated with 28% and 24% lower odds of overweight and obesity in children and adolescents, respectively. These estimates are on the lower side of the summary odds and risk ratios that have been produced by various meta-analyses to date2,3, which might be explained by the narrow range of sleep durations investigated in this study. There is evidence that short sleep may be associated with higher central adiposity in boys only.36 In adolescents, we similarly found that longer sleep was associated with lower central adiposity in boys, but not girls. Sex divergence of the associations between sleep duration with central adiposity may relate to the physiological changes that occur during puberty. A recent meta-analysis of prospective studies conducted in youth reported that every one hour longer baseline sleep was favourably associated with ΔBMI *z*-score over follow-up (β-coefficient (95% CI): -0.06 (-0.09 to -0.04)).3 We found no significant associations with ΔBMI *z*-score, but longer baseline sleep duration favourably predicted Δwaist *z*-score in children, and the association was very similar in magnitude to the meta-analysed estimate.

There is some inconsistency in the literature, but an increasing number of studies report that later bedtimes are associated with higher adiposity and weight status in youth.6–14 In cross-sectional analyses, we found that earlier bedtimes were dose-dependently associated with lower BMI *z*-score in both age groups, and with lower likelihood of overweight and obesity in children. Bedtimes between 21:00 to 22:00 and before 21:00 were associated with 23% and 32% lower odds of child overweight and obesity, respectively. Following adjustment for breast development as an indicator of pubertal status, an association of earlier bedtimes with lower waist *z*-score in girls was entirely attenuated. This highlights the importance of controlling for maturation in future studies of sleep and adiposity in youth. Independent of covariates, earlier bedtimes were associated with lower waist *z*-score in boys, particularly adolescent boys for whom associations were dose-dependent. Crucially, compared to a later bedtime, prospective analyses showed that a baseline bedtime between 21:00 to 22:00 favourably predicted Δwaist *z*-score in children, and there was some evidence, albeit weaker, that the same bedtime predicted ΔBMI *z*-score. These are important observations, especially considering that centrally stored adiposity is more metabolically harmful than total adiposity4, and because many children move to an evening circadian preference and later sleep timing as they age.37 Our results contribute to a small prospective evidence-base which shows that child bedtimes appear to be inversely associated with central adiposity and obesity development in youth.8–11

We attempted to disentangle the associations of longer sleep durations with lower adiposity, from that of earlier bedtimes with lower adiposity, by quantifying the effects of four combinations of behaviours. Each combination was characterised by a different average sleep length (later-shorter (reference) < earlier-shorter < later-longer < earlier-longer). We anticipated that a later-shorter pattern of sleep would be least desirable and that as sleep durations increased across categories so too would associations with lower adiposity. Results deviating from this pattern could point toward the importance of earlier bedtimes over and above longer sleep duration. The cross-sectional results for children showed that, relative to a later-shorter sleep pattern (mean sleep duration: 9.2h), later-longer sleep (10.2h) was associated with lower total adiposity and with lower central adiposity in boys, but, consistent with our hypothesis, associations were largest and most consistent for earlier-longer sleep (10.8h). Earlier-longer sleep was associated with lower total and central adiposity in both sexes and with 28% lower odds of overweight and obesity. Prospectively, a later-longer sleep pattern was favourably associated with Δwaist *z*-score in girls. In adolescents, compared to a later-shorter sleep pattern (8.3h), earlier-longer sleep (9.9h) was associated with lower total adiposity and with lower central adiposity in boys, but magnitudes of association were strongest for earlier-shorter sleep (8.7h), and there were no associations for later-longer sleep (9.3h). Our results appear to indicate that longer sleep durations are important for adiposity levels in children whereas earlier bedtimes may be key for adolescents. This finding is reminiscent of the results of a study of Australian youth (of mean age 13.4y). Olds and colleagues reported that relative to a late-bed/late-rise pattern of equivalent sleep duration (there was a difference of only 7 min/d between groups), an early-bed/early-rise pattern was associated with lower BMI *z*-score and lower likelihood of overweight and obesity.12 This may be related to adolescents with earlier bedtimes having an overall healthier lifestyle.12,13,38

With regard to possible mechanisms of action, this study provides new evidence that associations between sleep parameters with youth adiposity are independent of device-measured total sedentary time and physical activity. However, we did find that controlling for TV viewing and having a bedroom TV diminished the cross-sectional associations that were observed in children. It appears that TV viewing behaviours either partially confound or mediate the associations of longer sleep duration and earlier bedtimes with lower childhood adiposity. Importantly, another study found that an association of short sleep duration with higher adiposity in children was partially mediated by more TV viewing, which was measured at an intermediate time point.39 Watching TV, particularly late on in the evening, is associated with increased snacking of calorie-dense foods, overconsumption of foodstuffs due to missed satiety cues, exposure to food advertising, product placement of sugar-sweetened drinks40, and reduced sleep quality due to blue wavelength light emissions.41 Our results indicate that TV viewing should be limited in children who have later bedtimes or who are short sleepers. There was no evidence that TV parameters accounted for any of the reported associations in adolescents. This agrees with the finding of another large study, which found that associations of sleep parameters with adolescent adiposity and weight status were independent of myriad screen-based behaviours (including TV viewing duration, electronic gaming, and social media use).7 It is unfortunate that we were unable to investigate other potentially important covariates, such as sleep quality markers, depressive symptoms, eating patterns, and dietary factors. There is increasing evidence in the paediatric population that diet mediates a causal pathway between sleep duration with overweight and obesity.42

This pooled study investigated associations of bedtimes and sleep durations, separately and in combination, with adiposity and weight status in an international sample of youth. Rigorous procedures were used to harmonise data from 11 studies to enable a large, pooled analysis of individual-level data. It is unfortunate that not all covariables were available in the full sample and that time-variant factors were not assessed repeatedly over time. Even so, in various sub-selections we were able to adjust for a wider array of putative covariables than most studies have previously accounted for, including parental education, ethnicity, pubertal status, maternal BMI, and aspects of time-use, including device-measured sedentary time and intensity-specific physical activity. To mitigate selection biases and maximise sample size, and because variability in awake-time movement behaviours is smaller across weekdays than weekends43, participants were only required to provide ≥1 valid weekday of accelerometer data. This may have meant that habitual behaviours were not precisely captured for all participants, but 4 valid weekdays were provided on average, and more than four-fifths (82.5%) of the entire sample registered ≥3 valid weekdays of data. It is a limitation that not all participating studies were nationally representative and the vast majority of participants were white. There is evidence for effect modification of sleep-health associations by ethnicity and hence caution is warranted before generalising the results to non-white youth.44,45 All studies ascertained information for sleep by subjective reporting and most studies asked about the time that participants usually went to bed and got out of bed. The data for sleep duration may therefore be overestimated due to factors including screen-time engagement whilst in bed, sleep onset latency, overnight waking, and wakefulness on mornings prior to getting up (the latter three behaviours are nonetheless components of a normal sleep-wake cycle46). It is at least encouraging that most studies asked participants to recall sleep habits over a specific and recent time period and utilised parent-reports for young children, practices that are known to enhance the validity of subjectively reported sleep durations.30 The focus of this study was on weekday behaviours, but weekday-to-weekend sleep differences are common in youth, and it is a weakness that they were not considered. It has been suggested that regular bedtimes and weekend sleep compensation could protect against overweight and obesity, the latter by ameliorating the detrimental effects of short sleep on weekdays.47 Future studies should conceptualise sleep as a multidimensional construct and should examine all sleep parameters, including the duration, timing, quality, and variability of sleep, with respect to health.5

In conclusion, longer sleep duration and earlier bedtimes appear to be modifiable determinants of youth adiposity and weight status. If the associations are causal, both behaviours should be targeted for obesity prevention, potentially emphasising longer sleep for children and earlier bedtimes for adolescents.

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