The Journal of Child Psychology and Psychiatry

Journal of Child Psychology and Psychiatry **:* (2024), pp **–** doi:10.1111/jcpp.14004

Shorter night-time sleep duration and later sleep timing from infancy to adolescence

Ifigeneia Manitsa,[1,2] Alice M. Gregory,[3] Matthew R. Broome,[1,2,4,5] Andrew P. Bagshaw,[2,4] Steven Marwaha,[1,2,6] and Isabel Morales-Muñoz[1,2]

[1]Institute for Mental Health, University of Birmingham, Edgbaston, Birmingham, UK; [2]School of Psychology, University of Birmingham, Edgbaston, Birmingham, UK; [3]Department of Psychology, Goldsmiths, University of London, London, UK; [4]Centre for Human Brain Health, University of Birmingham, Edgbaston, Birmingham, UK; [5]Early Intervention Service, Birmingham Women's and Children's NHS Trust, Birmingham, UK; [6]Specialist Mood Disorders Clinic, Birmingham and Solihull Mental Health Trust, Birmingham, UK

Background: Here, we (a) examined the trajectories of night-time sleep duration, bedtime and midpoint of night-time sleep (MPS) from infancy to adolescence, and (b) explored perinatal risk factors for persistent poor sleep health. **Methods:** This study used data from 12,962 participants in the Avon Longitudinal Study of Parents and Children (ALSPAC). Parent or self-reported night-time sleep duration, bedtime and wake-up time were collected from questionnaires at 6, 18 and 30 months, and at 3.5, 4–5, 5–6, 6–7, 9, 11 and 15–16 years. Child's sex, birth weight, gestational age, health and temperament, together with mother's family adversity index (FAI), age at birth, prenatal socioeconomic status and postnatal anxiety and depression, were included as risk factors for persistent poor sleep health. Latent class growth analyses were applied first to detect trajectories of night-time sleep duration, bedtime and MPS, and we then applied logistic regressions for the longitudinal associations between risk factors and persistent poor sleep health domains. **Results:** We obtained four trajectories for each of the three sleep domains. In particular, we identified a trajectory characterized by persistent shorter sleep, a trajectory of persistent later bedtime and a trajectory of persistent later MPS. Two risk factors were associated with the three poor sleep health domains: higher FAI with increased risk of persistent shorter sleep (OR = 1.20, 95% CI = 1.11–1.30, $p < .001$), persistent later bedtime (OR = 1.28, 95% CI = 1.19–1.39, $p < .001$) and persistent later MPS (OR = 1.30, 95% CI = 1.22–1.38, $p < .001$); and higher maternal socioeconomic status with reduced risk of persistent shorter sleep (OR = 0.99, 95% CI = 0.98–1.00, $p = .048$), persistent later bedtime (OR = 0.98, 95% CI = 0.97–0.99, $p < .001$) and persistent later MPS (OR = 0.99, 95% CI = 0.98–0.99, $p < .001$). **Conclusions:** We detected trajectories of persistent poor sleep health (i.e. shorter sleep duration, later bedtime and later MPS) from infancy to adolescence, and specific perinatal risk factors linked to persistent poor sleep health domains. **Keywords:** Sleep duration; chronotype; trajectories; perinatal risk factors; ALSPAC.

Introduction

Sleep is crucial for health and well-being, and poor sleep can place children at risk for poor health. Short sleep in children (i.e. from infancy to school-aged children) has been associated with poor educational attainment (Blunden et al., 2018), impaired problem-solving skills (Fallone, Owens, & Deane, 2002), socioemotional problems (Morales-Muñoz et al., 2020), attention deficit and hyperactivity disorder (ADHD) (Huhdanpää et al., 2019; Morales-Muñoz & Gregory, 2023), poorer psychosocial and physical health outcomes (Quach, Price, Bittman, & Hiscock, 2016) and obesity (Hart et al., 2013; Tuohino et al., 2019) in childhood and adolescence, among other adverse outcomes.

Studies examining the developmental course of sleep duration at a population level indicate an overall decline in sleep duration from childhood to pre-adolescence (Price et al., 2014) and adolescence (Iglowstein, Jenni, Molinari, & Largo, 2003). Regarding sleep duration across childhood, daytime naps are a sleep aspect that differs between infants and pre-schoolers versus children and adolescents (Galland, Taylor, Elder, & Herbison, 2012). More specifically, in newborns, sleep is distributed throughout the day and night (Davis, Parker, & Montgomery, 2004), and at around 10–12 weeks of age, infants' sleep becomes increasingly nocturnal (Iglowstein et al., 2003; Paavonen et al., 2020). Between the ages of 1 and 4 years, children continue to sleep during the day, with an average of 2–3 h needed at 1 year old (Sadeh, Mindell, Luedtke, & Wiegand, 2009) to 1–2 h at 4 years old (Iglowstein et al., 2003). It is by age 5 when daytime napping ceases and overnight sleep duration gradually declines throughout childhood (Staton et al., 2020). Concerning sleep timing, there are also differences between childhood and adolescence, with earlier bedtimes being more appropriate in younger children and later bedtimes more frequent for adolescents (Mindell & Meltzer, 2008) due to pubertal changes in the homeostatic and circadian regulation of sleep (Hagenauer, Perryman, Lee, & Carskadon, 2009).

However, there are considerable inter- and intraindividual differences which are not well understood.

Conflict of interest statement: No conflicts declared.

Trajectory studies allow us to detect specific groups of children, based on their sleep and how it changes with age. Existing research investigating the trajectories of sleep duration has identified short, medium and long sleep duration groups across childhood (e.g. from 1.5 to 9 years old) (Blunden et al., 2018; Plancoulaine et al., 2018; Touchette et al., 2009). Among groups, it is important to highlight the trajectory characterized by persistent shorter sleep, which potentially constitutes the group at highest risk of developing adverse outcomes (Blunden et al., 2018; Medic, Wille, & Hemels, 2017). Long sleep duration has been also associated with adverse health outcomes, particularly in adults (Jike, Itani, Watanabe, Buysse, & Kaneita, 2018), but less is known about the effects of long sleep in children. Therefore, our focus will be on persistent shorter sleep duration, as the evidence in this area across childhood is more conclusive.

Other relevant sleep features in childhood, such as sleep timing, have received less attention (Magee & Blunden, 2020). Examples of sleep timing are bedtime and midpoint of sleep (MPS; i.e. defined as the middle time point between bedtime and wakeup timing), which are both indirect measures of chronotype (Urbanek et al., 2018). Chronotype is defined as the individual preference in sleep timing and activity (Taylor & Hasler, 2018), and late chronotype is associated with adverse health consequences (Fabbian et al., 2016). In childhood, later chronotype is associated with reduced school performance at 10 years old (Zerbini & Merrow, 2017), and increased mental health problems at 6–12 years old (Eid et al., 2020) and sleep problems at 4–5 years old (Jafar et al., 2017). As with sleep duration trajectories, sleep timing trajectories allow us to detect children with a persistent sleep problem over time (later chronotype), which would constitute the group at highest risk for future adverse outcomes. Finally, among the scarce studies investigating the adverse outcomes associated with sleep trajectories in childhood, recent research suggests that longer sleep duration trajectories are associated with better cognitive functioning in childhood (Cai et al., 2023), and that higher sleep–wake problems during late childhood lead to higher levels of mental health problems in adolescence (Shimizu, Zeringue, Erath, Hinnant, & El-Sheikh, 2021).

Sleep duration and sleep timing can be affected by a wide range of factors in childhood. Screen time exposure (Fadzil, 2021), ethnicity (Galland et al., 2012), age (Paavonen et al., 2020; Sadeh et al., 2009), socioeconomic and family risks (e.g. distress and marital hostility) (Williamson, Mindell, Hiscock, & Quach, 2019), cumulative adverse childhood experiences (Sullivan, Rochani, Huang, Donley, & Zhang, 2019) and parents' behaviour (Bruni et al., 2014; Iglowstein et al., 2003) are all associated with shorter sleep duration in children (e.g. from 0 to 12 years old) and beyond (e.g. adulthood). Other risk factors associated with late chronotype in childhood (i.e. from birth until school-aged children) include biological (e.g. puberty), environmental (e.g. noise and light) and interpersonal factors (e.g. co-sleeping) (Liu, Ji, Rovit, Pitt, & Lipman, 2022). Considering that the perinatal period (i.e. from pregnancy until first year of child's life) is recognized as a critical period of increased plasticity in the early development of humans (Barba-Müller, Craddock, Carmona, & Hoekzema, 2019), risk factors occurring at this very early stages of life might be crucial in the development of poor sleep health in children. Despite little research on the topic, the existing study indicates that perinatal maternal mental health has an impact on the sleep development of the child (Morales-Munoz et al., 2018). Therefore, further research is needed to understand the role of perinatal risk factors in the development of poor sleep health in children.

The main aim of this study was to detect specific groups of young people (from infancy to adolescence) with persistent poor sleep health (i.e. persistent night-time short sleep, persistent late bedtime and persistent late MPS from infancy to adolescence), in addition to describing the night-time sleep duration and sleep timing trajectories in a large population-based cohort study. Finally, we examined a range of perinatal risk factors occurring before age of 6 months which might be longitudinally associated with persistent poor sleep health. We hypothesized that we would detect a group with persistent shorter night-time sleep duration (Touchette et al., 2009) and also a group with later bedtime and later MPS (Magee & Blunden, 2020). Furthermore, we hypothesized that a range of early-life adversities, including family adversities, would be related to persistent poor sleep health (Plancoulaine et al., 2018).

Methods

Participants

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a birth cohort study, set in the United Kingdom, examining the determinants of development, health and disease during childhood and beyond (Boyd et al., 2013; Fraser et al., 2013). The ALSPAC study website contains details of all the data available through a fully searchable data dictionary and variable search tool (http://www.bristol.ac.uk/alspac/researchers/our-data/). Further details of the ALSPAC are provided in Appendix S1. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time.

Measures

Sleep. Parent-reported sleep was collected at the following available time points: 6, 18 and 30 months, and at 3.5, 4–5, 5–

6, 6–7, 9 and 11 years. Furthermore, self-reported sleep was collected at 15–16 years. More details regarding the sleep data collected appear in Appendix S2. Here, we focused on night-time sleep duration, bedtime and MPS, at each of these time points. Night-time sleep duration was calculated from questions asking what time the child/adolescent 'normally' went to sleep in the evening (bedtime) and woke up in the morning (waking time). MPS, which refers to the clock time between sleep-onset and wake-up time, was obtained by calculating the time halfway between bedtime and waking time. MPS highly correlates with dim-light melatonin onset in toddlers (Simpkin et al., 2014) and adults (Kantermann, Sung, & Burgess, 2015), and is a more reliable marker of chronotype than bedtime alone in adults (Kantermann et al., 2015), children (Werner, Lebourgeois, Geiger, & Jenni, 2009) and toddlers (Simpkin et al., 2014). Therefore, here, we included both bedtime and MPS as indirect measures of chronotype. Our approach to using midpoint of sleep as early as 6 months of age is justified by existing evidence, suggesting that the circadian rhythm of cortisol in infants appears as early as 2 weeks up to 9 months of age (Ivars et al., 2015), and that by the age of 2 months, there are clear signs of circadian rhythmicity regarding both the sleep–wake cycle and hormone secretion (Rivkees, 2003).

Perinatal risk factors. Family adversities during pregnancy were assessed using the Family Adversity Index (FAI) (Steer & Wolke, 2004), comprising 18 items on early parenthood, housing conditions, maternal education, financial difficulties, parents' relationship, family size, family major problems, maternal psychopathology, parents' substance abuse, crime records, partner support and social network. If adversity is present, an item is rated as 1 and scores are summed to create a total score. Previous literature highlights the effect of family adversities and cumulative adverse childhood experiences on sleep duration (Fadzil, 2021; Sullivan et al., 2019). Importantly, exposure to multiple risk factors predicts more severe, adverse developmental consequences compared to singular risk factor exposure (Evans, Li, & Whipple, 2013), which supports the hypothesis that cumulative exposure to adversity as measured with the FAI during pregnancy would lead to the development of sleep problems in childhood. Further details about the FAI are provided in Appendix S3.

Maternal postnatal depression at 8 weeks was measured with the Edinburgh Postnatal Depression Scale (Cox, Holden, & Sagovsky, 1987). The total score was used for this study. Higher scores indicate higher levels of depression. Depression during pregnancy is linked to changes in sleep behaviour (Plancoulaine et al., 2018).

Maternal postnatal anxiety at 8 weeks was measured using the Crown–Crisp Index (Joukamaa, 1992). Response options for all items are presented on a 3-point scale with higher scores indicating worse symptoms. We only used the total score from the anxiety subscale. Maternal anxiety is linked to shorter sleep duration in childhood (Schultz et al., 2020).

Prenatal maternal socioeconomic status was measured using the Cambridge Social Interaction and Stratification Scale, which provides a total score (Prandy, 1990), with higher scores indicating higher socioeconomic status. Socioeconomic status is associated with short sleep in children (Liu et al., 2022).

Child temperament was measured at 6 months using the Carey Temperament Scales (Carey & McDevitt, 1978). Response options range from 1 (almost never) to 6 (almost always) and higher scores indicate greater difficulty. We selected the 'Mood' and 'Activity' subscales. 'Mood' is designed to measure the general tone of affect, and 'Activity' is the motor component. These are the temperament factors in young children that are associated with sleep problems (Morales-Muñoz et al., 2020).

Child's sex, birth weight, health at 4 weeks (good vs. bad), maternal gestational age and age when baby was born were also included.

The factors above have been selected as potential risk factors, for the following reasons: (a) all these factors are perinatal risk factors occurring during pregnancy or before the child's age of 1 year; (b) the available data were collected before or at the age of 6 months, which is the first time point of our main outcome – this enables us to treat these factors as potential precursors; (c) as stated above, there is robust evidence supporting the association between each of these factors with sleep development in childhood; and (d) all these factors refer to relevant environmental and intrinsic perinatal factors in children's sleep development (Camerota, Propper, & Teti, 2019; Kocevska et al., 2023; Lund & Ystrom, 2022; Punamäki et al., 2022). All factors were reported by the mother. Further details of the questionnaires used above are provided in Table S1.

Although some studies on childhood sleep problem trajectories have organized early-life risk factors into multiple cumulative risk index domains according to socio-ecological theory [e.g. cumulative socio-ecological risks (Williamson et al., 2019)], in this study we were mainly interested in separate perinatal risk factors (rather than in cumulative risk) to identify those relevant (and specific) perinatal risk factors for persistent poor sleep health in childhood. This could help to design future targeted interventions to improve poor sleep health. FAI, which could be considered a cumulative risk index during pregnancy, has been included here as an independent risk factor, based on extensive studies within the ALSPAC that support it as an independent risk factor for family adversity (Adjei et al., 2022; Bowen, Heron, Waylen, & Wolke, 2005; Steer & Wolke, 2004).

Statistical analyses

A multi-staged analysis plan was developed. The first stage was conducted in SPSS-v27 to provide descriptive statistics for the normative patterns of night-time sleep duration, bedtime and MPS from infancy to adolescence and the various risk factors.

Second, latent class growth analyses (LCGA) were conducted using Mplus-v8 to investigate differing trajectories of night-time sleep duration, bedtime and MPS, separately, from infancy to adolescence. Several models were fitted by increasing the number of classes (i.e. 2 to 6 classes) (Jung & Wickrama, 2008). In LCGA, the cases are assigned to each class using a probabilistic allocation in terms of how closely each case's trajectory matches the group-based trajectories identified. The best-fitting classification model was initially chosen according to fit indices [i.e. Bayesian information criteria (BIC) and Vuong–Lo–Mendell–Rubin (VLMR) test] and entropy. Lower BIC values suggest better model fit. A significant VLMR value suggests that a K-class model fits the data better than a (K-1) class model. Entropy values approaching 1 indicate a clear delineation of classes. Finally, to decide upon the optimal class solution, an emphasis was placed on group sizes (i.e. >2%) and clinically relevant and informative interpretation, according to previous literature (Jung & Wickrama, 2008). Missing values due to attrition were handled by the full-information maximum-likelihood estimation method (Enders & Bandalos, 2001).

Third, Spearman correlation coefficients between the three poor sleep health domains identified from LCGA above (persistent shorter sleep, persistent later bedtime and persistent later MPS) were applied, to ascertain whether each of them could be treated as independent sleep domains for further analyses. Additionally, cross-tabulation analyses were conducted to look at the interrelationship between class memberships for each of the three poor sleep health domains. Finally, Pearson correlations between the different time points, for each

sleep variable, were also applied, to further understand the potential changes of the sleep variables over time.

Fourth, and as a primary analysis, logistic regression analyses using SPSS-v27 were applied to define perinatal risk factors longitudinally associated with persistent poor sleep health from infancy to adolescence. The explanatory variables were: (a) maternal perinatal risk factors (age at birth of child in ALSPAC), gestational age, postnatal anxiety, postnatal depression, FAI and socioeconomic status; and (b) child's early-life risk factors (sex, birth weight, health at 4 weeks, temperament–mood at 6 months and temperament–activity at 6 months). For the outcome, we created a new dichotomous variable, based on the best model fit classes obtained from LCGA. This was done for each of the three sleep variables separately: the classes representing persistent shorter sleep, persistent later bedtime and persistent later MPS were recoded as 1, while the other classes were recoded as 0. We decided to compare the class representing persistent poor sleep health with the other three classes together (i.e. Class 1 vs. the other three classes), as we mainly focused on the chronicity of these sleep conditions. Separate logistic regression analyses were applied for each sleep domain. To deal with missing data due to follow-up, we conducted logistic regressions to identify significant factors associated with attrition (see Tables S2a and S2b). Using the variables associated with selective dropout as predictors, we fitted a logistic regression model to determine weights for each individual using the inverse probability of response, following previous research (Hogan, Roy, & Korkontzelou, 2004; Kinner, Alati, Najman, & Williams, 2007). We used this weighting variable in the logistic regression analyses.

Finally, as secondary analyses, we conducted multinomial regression analyses. These included the same perinatal risk factors as in the logistic regression analyses above as predictors, and all the classes from the model with best model fit as the outcome. We used as reference the class with the largest sample size. This was done for each of the three sleep variables.

As sensitivity analyses, we applied the same analyses above (i.e. descriptive analyses, LCGA and logistic regressions), but excluded all the time points in childhood where napping still occurs (Iglowstein et al., 2003) (6, 18 and 30 months, and 3.5 and 4–5 years), and the latest time point (15–16 years), to explore whether the results remained similar or were heavily dependent on specific aspects associated with these time points. From 6 months to 4–5 years, daytime sleep still occurs, and thus this would impact night-time sleep and total sleep duration. At 15–16 years old, sleep variables were self- (rather than parent-) reported and we wanted to test whether there was bias associated with the source of information. Furthermore, to explore what per cent of cases were allocated to different classes after conducting sensitivity analyses as compared to when running the primary analyses, we conducted cross-tabulation analyses using SPSSv27, for each sleep variable separately.

Results

Sleep development from infancy to adolescence

In relation to the development of night-time sleep duration from infancy to adolescence, we observed an increased duration from 6 months to 4–5 years, with a subsequent decrease from 5–6 to 15–16 years. In relation to bedtime, except for the earliest time point of 6 months, where the average bedtime was around 20:00, we observed a trend towards later bedtime over time. Finally, in relation to MPS, we observed that the average MPS initially decreased over time, from 6 months to 3.5 years, while from 4 to 5 years onwards, there was an increased lateness. See Table 1 and Figure S1 for the descriptive patterns of the three sleep variables at all time points.

Sleep trajectories

Table 2 shows VLMR, BIC and entropy for all models assessed (2–6 classes) (Jung & Wickrama, 2008) for each of the sleep measures tested. Furthermore, the descriptive values regarding the three sleep variables, at each time point, are provided in Table S3. BIC decreased with the addition of each class indicating a better model fit for more classes. This pattern is typically found in large samples (Wiggins, Mitchell, Hyde, & Monk, 2015). However, decreases in BIC became considerably smaller in four classes compared to three classes (for each of the sleep domains). Furthermore, VLMR showed a statistically significant difference for the two classes, three classes, four classes and five classes models for night-time sleep duration and bedtime, while VLMR was statistically significant for two classes, three classes and four classes for MPS. Finally, the entropy values for the four-class model for each of the three sleep domains were the highest, compared to the other classes, indicating the highest classification accuracy for the four-class model. Overall, the three main-fit indices tested (i.e. VLMR, BIC and entropy) suggested that for each of the sleep domains, the four-class model offered the best model fit. To confirm this optimal four-class solution, we checked that there were large enough group sizes (>2% of the sample) in each of the classes, which was the case; and that the trajectories detected were physiologically plausible (Carno, Hoffman, Carcillo, & Sanders, 2003). The four-derived classes for each of the three sleep patterns tested appear in Figure 1.

The four-class model of night-time sleep duration included: Class 1 (N = 3,331, 25.5%) characterized by persistent average shorter sleep duration, Class 2 (N = 7,865, 60.7%) characterized by persistent average longer sleep duration, Class 3 (N = 386, 3.0%) characterized by persistent shorter sleep duration and Class 4 (N = 1,400, 10.8%) characterized by persistent longer sleep duration.

The four-class model of bedtime included: Class 1 (N = 365, 2.8%) characterized by persistent later bedtime, Class 2 (N = 2044, 15.8%) characterized by persistent average later bedtime, Class 3 (N = 7,524, 58.0%) characterized by persistent average earlier bedtime and Class 4 (N = 3,040, 23.4%) characterized by persistent earlier bedtime.

The four-class model of MPS included: Class 1 (N = 644, 5.0%) characterized by persistent later MPS, Class 2 (N = 3,079, 24.0%) characterized by persistent average later MPS, Class 3 (N = 7,027, 54.8%) characterized by persistent average earlier

Table 1 Descriptive analyses of night-time sleep duration and sleep timing, across each time point, from 6 months to 15–16 years old

	6 months Mean (*SD*)	18 months Mean (*SD*)	30 months Mean (*SD*)	3.5 years Mean (*SD*)	4–5 years Mean (*SD*)	5–6 years Mean (*SD*)	6–7 years Mean (*SD*)	9 years Mean (*SD*)	11 years Mean (*SD*)	15–16 years Mean (*SD*)
Night sleep duration (hr)	10.80 (1.36)	11.33 (1.08)	11.24 (1.01)	11.27 (0.90)	11.39 (0.72)	11.28 (0.72)	10.99 (0.64)	10.43 (0.65)	10.05 (0.66)	8.84 (1.03)
Bedtime (hh:mm)	20:08 (1:12)	19:46 (0:46)	19:49 (0:57)	19:45 (0:51)	19:44 (0:43)	19:52 (0:43)	20:23 (0:45)	21:10 (0:49)	21:40 (1:06)	21:38 (3:33)
Midpoint of sleep[a] (hh:mm)	1:32 (0:52)	1:28 (0:51)	1:26 (0:41)	1:23 (0:37)	1:26 (0:32)	1:31 (0:30)	1:54 (0:32)	2:24 (0:29)	2:45 (0:29)	3:58 (0:47)

[a]Midpoint of sleep represents a proxy for circadian rhythm with a lower number representing an earlier (more lark-like) rhythm, and a higher number representing a later (more owl-like) rhythm.

Table 2 Bayesian information criterion, Vuong–Lo–Mendell–Rubin likelihood test *p* values and entropy for classes 2–6, for night-time sleep duration, bedtime and midpoint of sleep, from 6 months to 15–16 years old

	BIC	VLMR-*p*	Entropy
Night-time sleep duration			
2 classes	233852.802	<.001	0.610
3 classes	232070.863	<.001	0.640
4 classes	**230129.693**	**.0007**	**0.703**
5 classes	229373.743	.0105	0.641
6 classes	228995.005	.4669	0.612
Bedtime			
2 classes	245618.438	<.001	0.692
3 classes	243045.338	<.001	0.662
4 classes	**240781.640**	**<.001**	**0.745**
5 classes	240040.377	.0003	0.661
6 classes	239493.038	.5642	0.632
Midpoint of sleep			
2 classes	175645.818	<.001	0.690
3 classes	173326.047	<.001	0.677
4 classes	**169851.748**	**<.001**	**0.701**
5 classes	168982.451	.0055	0.645
6 classes	168341.134	.0119	0.636

BIC, Bayesian information criterion; VLMR-*p*, Vuong–Lo–Mendell–Rubin likelihood ratio test. Statistical significance at $p < 0.05$. Bold values here indicate the n-classes selected based on best model fit.

MPS and Class 4 ($N = 2073$, 16.2%) characterized by persistent earlier MPS.

Finally, we tested the correlations between the three poor sleep health domains (persistent shorter sleep, persistent later bedtime and persistent later MPS), and we found that although these groups were related to each other (all $p < .001$), the strength of the associations was low to moderate (from $r_s = .18$ to $r_s = .59$) (Dancey & Reidy, 2004), indicating that they can be treated as independent sleep domains (see Table S4). Furthermore, the cross-tabulation analyses showed that less than half of the children from one poor sleep health group were also part of another poor sleep health group, suggesting low interrelationship between classes (see Table S5). In relation to the correlations of the different time points, for each sleep variable separately (see Table S6), we observed that the strongest associations appeared between the closest time points, while the weakest were between the time points that were more distant from each other.

Perinatal risk factors

A description of the perinatal and early-life risk factors occurring before age 6 months appears in Table 3. Several risk factors were differentially connected to the poor sleep health domains (primary analyses – see Table 4). Among those, two risk factors were longitudinally associated with all three of the poor sleep health domains tested. Specifically, higher FAI was significantly associated with increased risk of persistent shorter sleep

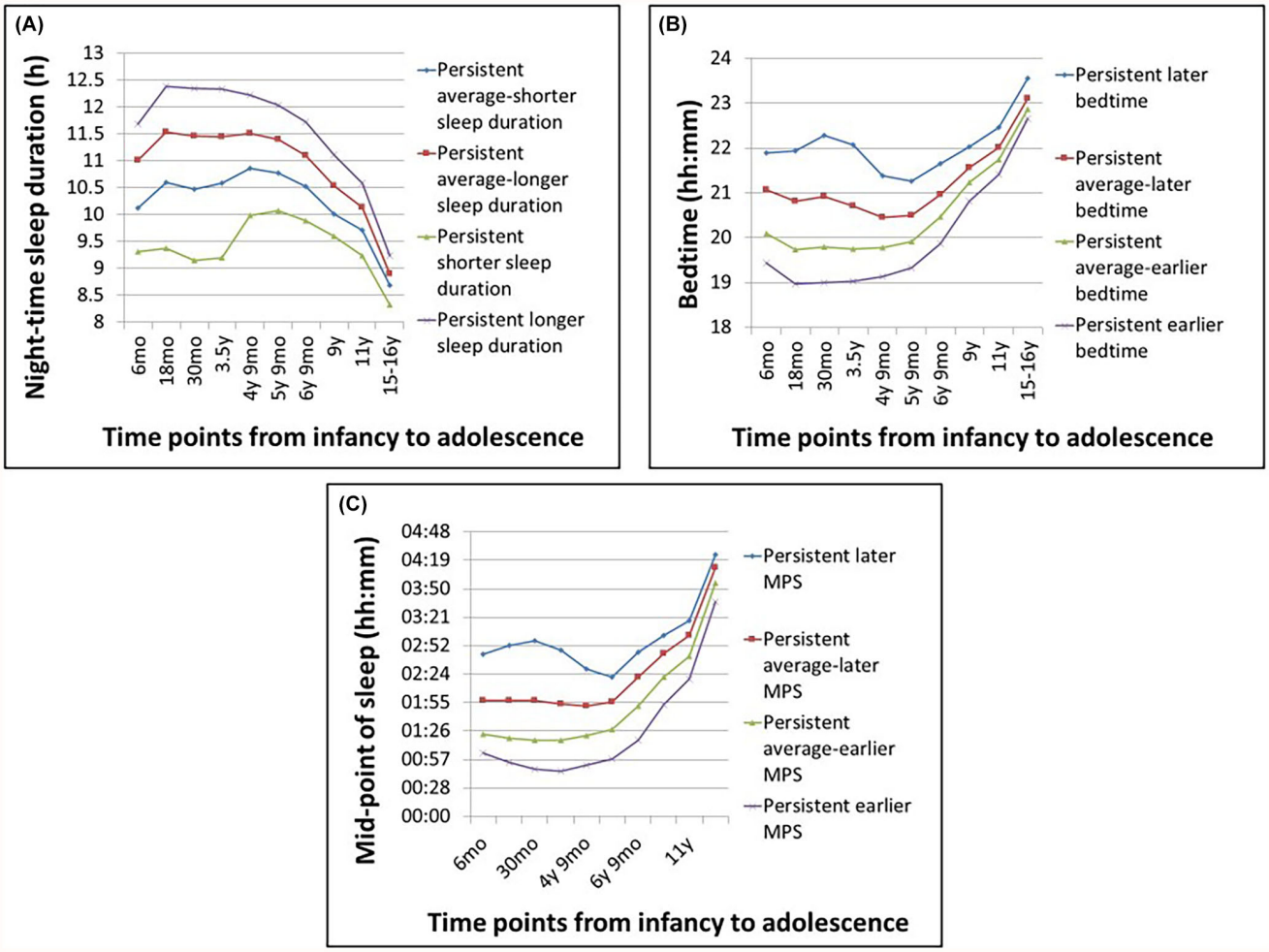

Figure 1 Growth trajectories of night-time sleep duration, bedtime and midpoint of sleep (MPS) across infancy, childhood and adolescence (from 6 months to 15–16 years). The LCGA detected the best model fit for four classes for all sleep patterns. X axis represents the 10 time points from infancy to adolescence, and the Y axis represents the mean number of hours (for A) and the mean clock time (for B and C). Graph A reflects the trajectories for night-time sleep duration. Class 1 represents persistent average shorter sleep duration; Class 2, persistent average longer sleep duration; Class 3, persistent shorter sleep duration; and Class 4, persistent longer sleep duration. Graph B shows the trajectories for bedtime. These trajectories show a Class 1 characterized by persistent later bedtime; Class 2, by persistent average-later bedtime; Class 3, by persistent average-earlier bedtime; and Class 4, by persistent earlier bedtime. Graph C shows the trajectories for MPS. These trajectories show a Class 1 characterized by persistent later MPS; Class 2, by persistent average-later MPS; Class 3, by persistent average-earlier MPS; and Class 4, by persistent earlier MPS

(OR = 1.20, 95% CI = 1.11–1.30, $p < .001$), persistent later bedtime (OR = 1.28, 95% CI = 1.19–1.39, $p < .001$) and persistent later MPS (OR = 1.30, 95% CI = 1.22–1.38, $p < .001$). Similarly, higher maternal socioeconomic status was significantly associated with reduced risk of persistent shorter sleep (OR = 0.99, 95% CI = 0.98–1.00, $p = .048$), persistent later bedtime (OR = 0.98, 95% CI = 0.97–0.99, $p < .001$) and persistent later MPS (OR = 0.99, 95% CI = 0.98–0.99, $p < .001$). Finally, it is important to highlight that although FAI was included as an item related to financial difficulties during pregnancy (i.e. Yes = 1 point), this differs from the maternal socioeconomic status measure included in this study (i.e. CAMSIS). The latter relies on patterns of social interaction to determine social structure and the respondents' position in it (Prandy, 1990). Therefore, CAMSIS constitutes a more robust measure of socioeconomic status. This supports our inclusion of FAI and socioeconomic status during pregnancy as separate variables.

As many of our predictors were treated as raw scores and as many of these variables were unlikely normally distributed due to the nature of the variables (e.g. anxiety), we conducted additional logistic regression analyses, where the continuous independent variables were Z-transformed. As can be seen in Table S7, very similar results remained after including Z-transformed variables.

Finally, and in relation to the secondary analyses using multinomial regression analyses, we observed similar results for the persistent poor sleep health class. In addition, the associations between predictors and outcomes differed slightly depending on the trajectory explored (see Table S8). For clarity, we will focus here on reporting those associations related to the persistent poor sleep health groups, as this was the main focus of our work. However, it is

Table 3 Descriptive values of perinatal and early-life risk factors

	N	%
Sex (male/female)	7,691/7,348	51.1/48.9
Baby's health at 4 weeks (poor/good)	40/12,216	0.3/99.7

	Mean	*SD*	Min	Max
Birth weight, kg	3.38	0.58	1.20	5.64
FAI during pregnancy, total score	4.38	4.31	0	18
Maternal age when birth, years	27.99	4.96	15	44
Maternal postnatal depression, total	5.86	4.61	0	29
Maternal postnatal anxiety, total	3.54	3.27	0	16
Maternal socioeconomic status, total	54.59	13.47	18.90	94.42
Gestational age, weeks	38.36	5.51	4	47
Temperament – mood score at 6 months	18.07	5.68	1	48
Temperament – activity score at 6 months	23.15	4.54	3	36

FAI, Family Adversity Index.

Table 4 Logistic regression analyses between perinatal and early-life risk factors and persistent sleep duration and sleep timing problems across childhood and adolescence (from 6 months to 15–16 years)

	Persistent short sleep[a]			Persistent late bedtime[a]			Persistent late midpoint sleep[a]		
Risk factors	OR	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
Child's sex (M vs. F)[b]	**0.723**	**0.550–0.95**	**.020**	1.250	0.935–1.67	.133	1.226	0.988–1.52	.064
Child's birth weight, kg	0.991	0.963–1.02	.567	1.009	0.977–1.04	.599	0.992	0.968–1.02	.488
Baby health (Poor vs. good) at 4 months	2.933	0.366–23.48	.311	2.917	0.352–22.56	.329	1.517	0.191–12.06	.694
FAI total score during pregnancy[c]	**1.203**	**1.114–1.30**	**<.001**	**1.284**	**1.188–1.39**	**<.001**	**1.300**	**1.224–1.38**	**<.001**
Maternal age when birth	**1.077**	**1.047–1.11**	**<.001**	**1.055**	**1.024–1.09**	**<.001**	1.003	0.980–1.03	.788
Maternal postnatal depression[c]	0.990	0.948–1.03	.639	1.018	0.972–1.07	.445	0.973	0.939–1.01	.145
Maternal postnatal anxiety[c]	1.034	0.974–1.11	.260	0.977	0.915–1.04	.487	1.007	0.958–1.06	.789
Maternal socioeconomic status[c]	**0.990**	**0.980–0.998**	**.048**	**0.978**	**0.967–0.989**	**<.001**	**0.985**	**0.977–0.993**	**<.001**
Gestational age	1.011	0.923–1.11	.820	0.904	0.824–0.99	.035	1.009	0.939–1.10	.806
Temperament–mood score at 6 months[c]	**1.035**	**1.011–1.06**	**.004**	1.025	0.998–1.05	.065	1.005	0.985–1.03	.645
Temperament–activity score at 6 months[c]	**1.052**	**1.020–1.08**	**.001**	1.003	0.971–1.04	.846	0.983	0.960–1.01	.171

FAI, Family adversity index.
[a]Three separate logistic regression analyses; one per main outcome.
[b]M = male (1); F = female (0); OR <1 means that being female is associated with persistent night-time short sleep duration and late midpoint of sleep.
[c]These risk factors were all treated as continuous variables, with higher scores indicating worse functioning. Statistical significance at $p < 0.05$. Bold values here indicate all statistically significant results from the logistic regression analyses.

important to highlight that a range of perinatal risk factors are also related to other sleep trajectories, which should be further considered in future research.

Sensitivity analyses

Regarding the developmental patterns and trajectories for night-time sleep duration, bedtime and MPS, we observed similar results to the main analyses when removing the early and late time points (see Tables S9 and S10, and Figure S2). Concerning the logistic regression analyses, we similarly observed that higher FAI was significantly associated with the three main outcomes (see Table S11). Overall, these sensitivity analyses showed similar results to the main analyses. In relation to the cross-tabulation analyses to compare the potential change of allocation cases per class between the primary and the sensitivity analyses, we observed that for each class, the percentage of cases that remained in the same class after sensitivity analyses ranged from 61.6% to 89.6% (see Table S12), suggesting that the potential impact of napping varied based on the outcome.

Discussion

To our knowledge, this is the first study to provide information on trajectories of both sleep duration and sleep timing from infancy to adolescence, using a large population-based cohort study. First, we detected different trajectories of night-time sleep duration, bedtime and MPS from 6 months to 15–16 years. For each of the three sleep domains, we found a group characterized by poor sleep health

(persistent shorter sleep, persistent later bedtime and persistent later MPS). Second, several perinatal risk factors were specifically associated with the three persistent poor sleep health domains tested, with family adversity and lower maternal socioeconomic status during pregnancy being the two factors associated with all three poor sleep health domains.

Sleep trajectories from infancy to adolescence

We found different trajectories of night-time sleep duration and sleep timing from 6 months to 15–16 years. More specifically, we detected four different trajectories for each of the sleep domains investigated. Concerning night-time sleep duration, the four trajectories included persistent shorter sleep, persistent average shorter sleep, persistent average longer sleep and persistent longer sleep. Previous research on night-time sleep duration in childhood has reported three (Tham et al., 2021; Zheng et al., 2021), four (Touchette et al., 2009) or five (Plancoulaine et al., 2018) sleep trajectories over time. Similar to the trajectories identified in previous research (Price et al., 2014; Zheng et al., 2021), the trajectories emerging from our study showed that night-time sleep duration decreased as participants' age increased. However, the discrepancies in the number of trajectories detected likely reflect differences between studies in terms of methodologies, time points and/or sample sizes. In our case, we provided trajectories from infancy to adolescence and hence covered a large part of the young person's development, while the existing studies on trajectories were limited to shorter periods of time, or at different ages during childhood. Furthermore, we included a population-based sample (n > 12,900), which is larger than those used in previous studies.

The existing evidence in relation to sleep timing trajectories is much scarcer, and to our knowledge, this is the first study reporting trajectories on bedtime and MPS from infancy to adolescence. Similar to night-sleep duration trajectories, we found four sleep timing trajectories (i.e. persistent earlier, persistent earlier average, persistent later average and persistent later). So far, only one study has reported individual differences in sleep timing longitudinally in 3,552 adolescents aged 12–13 years (Magee & Blunden, 2020), and found six sleep timing profiles (early larks, larks, intermediate, owls, variable owls and late owls). Future studies should provide further evidence on how different trajectories of sleep timing develop over time, to better understand how chronotype evolves and varies over time from birth until later stages of life.

Importantly, for the three sleep variables explored, we were able to identify a specific group of young people with persistent poor sleep health (i.e. persistent shorter night-time sleep and persistent later chronotype), which constitute the groups at highest risk for adverse outcomes. Future longitudinal studies should focus on investigating the impact that these specific trajectories have in a range of areas, including mental and physical health.

For future research, it will be essential to explore the development of these trajectories after adolescence. From our results, it seems that these trajectories tend to converge over time, with lower differences between trajectories when they reach adolescence. However, it will be crucial to see whether greater discrepancies appear when they reach specific milestones, such as young adulthood, which is a crucial stage for the onset of a range of mental health problems (Wood et al., 2018), and may subsequently impact the sleep patterns of young people. Therefore, further studies to investigate the development of sleep trajectories over prolonged periods of time are required.

Perinatal risk factors

We identified a range of perinatal risk factors longitudinally associating with the persistent poor sleep health domains tested – among these, family adversity and maternal socioeconomic status during pregnancy were the only factors associated with all three poor sleep health domains. Extending previous literature on social factors that influence sleep (Liu et al., 2022; Williamson et al., 2019), our findings showed that higher maternal socioeconomic status was associated with lower risk for persistent shorter sleep, later bedtime and later MPS. Previous research has also highlighted the association between family adversity and sleep duration and quality (Fadzil, 2021), which supports our findings. In contrast to previous research, the novelty of our study is that we investigated perinatal risk factors that occur before the age of 6 months, and which are associated with persistent poor sleep health from infancy to adolescence. Therefore, a better characterization of these perinatal factors will be essential to better understand the development of poor sleep health from very early stages of life, and potentially identify those at highest risk. Importantly, adverse life experiences (e.g. FAI) during pregnancy can also disrupt mothers' sleep in pregnancy (Touchette, Servot, Lemieux, & Berthelot, 2020), which may also have a negative impact on the child's sleep (Morales-Munoz, Saarenpaa-Heikkila, et al., 2018). This suggests that addressing mothers' sleep during pregnancy will also be crucial to understanding the development of poor sleep health across childhood, in addition to the perinatal risk factors identified in our study. Finally, and considering that adverse life experiences (e.g. FAI) are often intergenerational and chronic (Schickedanz, Escarce, Halfon, Sastry, & Chung, 2021), future studies should explore FAI as mediator between poor sleep health across early childhood and later child mental health outcomes.

Implications

Our findings could have implications for the future screening practices of clinicians and/or social workers to better work with families and effectively address sleep problems by targeting specific risk factors during the perinatal period. For example, clinicians and/or other practitioners working with children (e.g. social workers) should consider routinely screening for adverse childhood experiences, in addition to the inclusion of robust measures of socioeconomic status within their daily practices. This could allow early detection of children at risk for poor sleep health.

In addition, the fact that family adversity and maternal socioeconomic status during pregnancy were associated with poor sleep health suggests that policy-, housing- and/or neighbourhood-level interventions during pregnancy could be helpful in addressing the environmental variables that are less conducive to sleep. This could include addressing increased light or noise in lower-income neighbourhoods or poor-quality housing for families of lower SES. Furthermore, our findings align with the well-known inequalities in the health of infants in the United Kingdom, with the socioeconomic circumstances in which the infant is born and other early-life adversities having a major impact on their lives (Weightman et al., 2012). Therefore, our results add further support to the recently published recommendations by the Royal College of Paediatrics and Child Health (RCPCH) to reduce health inequalities as a result of child poverty and adverse childhood experiences (RCPCH, 2022).

Limitations

First, this study largely focused on parent or self-reported sleep, which could be different from objective sleep (Mazza, Bastuji, & Rey, 2020). During childhood, bedtime, wake time and therefore sleep time are under parental control and may not reflect or indirectly measure the child's individual chronotype and preferences. Considering this limitation, our findings should be interpreted with caution. Second, daytime sleep duration was not available for most of our time points (i.e. after 6 months old), and thus we were not able to provide trajectories on daytime or total sleep duration over time. Daytime naps have been linked with both positive (Leong et al., 2023; Souabni et al., 2021) and negative (Li et al., 2022) outcomes, can impact night-time sleep duration (Nakagawa et al., 2016) and the effects vary by age (Staton et al., 2020). While we could only focus on night-time sleep duration, future research should also consider daytime sleep. To address the possible impact of naps on our results, we conducted sensitivity analyses where we included only children over 5 years old [the age at which typically naps cease (Staton et al., 2020)] and the results remained similar. For example, a high percentage of cases remained in the same classes after running the sensitivity analyses, and family adversity and maternal socioeconomic status remained the most consistent risk factors for persistent shorter sleep, persistent later bedtime and persistent later MPS. Third, night-time sleep duration in this study was defined as time in bed, as it was calculated from bedtime and wake-up time. Thus, further research should include more accurate measures for sleep duration, such as actigraphy. Fourth, we included perinatal and early-life risk factors potentially associated with poor sleep health in childhood based on previous literature (and the available information in our existing data). However, other parental risk factors for poor sleep health such as genetic factors (Morales-Muñoz et al., 2021), maternal ADHD (Morales-Munoz, Saarenpaa-Heikkila, et al., 2018), maternal chronotype (Morales-Munoz et al., 2018), mother's and father's distress and/or marital/relational hostility (Williamson et al., 2019), parenting style (Tyler, Donovan, Scupham, Shiels, & Weaver, 2019) and screen time (Fadzil, 2021) were left unexplored as these were not available in our dataset. In addition, FAI and maternal socioeconomic status were only measured during pregnancy, therefore these factors might have changed over time and had a different impact on sleep problems. Fifth, due to the limitations of the data included in ALSPAC, we were not able to identify behavioural sleep problems as we did not have information on whether these were considered problematic, and this is why we refer here to the term 'poor sleep health'. However, where present, these behavioural sleep problems could have an impact on our identified trajectories of sleep (Magee, Gordon, & Caputi, 2014) and there could also be a bi-directional relationship between sleep and behavioural health problems across childhood (Shen et al., 2022). Future studies will need to investigate these issues in more detail. Sixth, we only included risk factors that occurred before or at 6 months of age, as the first time point of our outcome was at 6 months. However, we were not able to account for how changes in some of those variables such as maternal anxiety, maternal depression or temperament might impact changes in sleep. Seventh, some of our results could be partially explained by later contextual factors such as wake time and bedtime varying due to school start time and daily routines; however, the investigation of such contextual factors was out of scope in this study due to these data not being available. Eight, the sample of this study was predominantly of White ethnicity (i.e. around 97%), which limits the generalizability of our findings to other ethnic groups and ethnic comparisons. Individuals of ethnic minoritized backgrounds may be more likely to experience family adversity due to racism and discrimination and consequently be at higher risk for developing sleep problems (Grandner, Williams, Knutson, Roberts, & Jean-Louis, 2016). Therefore, future studies should specifically

investigate potential ethnicity differences in children's sleep development. Finally, the family adversity measure (i.e. the FAI) included in this study could be conceptualized as a cumulative risk factor for adversity during pregnancy and thus could not strictly align with the conceptualization of the rest of the variables, which were treated as separate risk factors. However, we included FAI as an independent variable of family adversity following a large number of studies using the ALSPAC (Adjei et al., 2022; Bowen et al., 2005; Steer & Wolke, 2004), which have used a similar approach. Furthermore, in this study, we were primarily interested in separate and individual risk factors for poor sleep health to inform the design of future targeted interventions. However, future studies should also explore the impact of cumulative risk factors during pregnancy and postnatally in the development of poor sleep health in the child to gain a broader understanding.

In summary, we provide evidence for different trajectories of night-time sleep duration and sleep timing from infancy to adolescence in a large population-based study, including trajectories of persistent poor sleep health over time. We also found that family adversity and maternal socioeconomic status during pregnancy link to the development of persistent shorter sleep and persistent later timing. Our research highlights the importance of measuring sleep development during the perinatal period and provides a basis to identify those children with persistent poor sleep health who may be at risk of a range of negative outcomes. Future research should investigate the long-term impact associated with persistent poor sleep health.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Further details of the ALSPAC cohort.
Appendix S2. Further details on sleep data.
Appendix S3. Further details on the Family Adversity Index (FAI).
Table S1. Psychometric properties about perinatal and early-life factors measures.
Table S2a. Differences in sociodemographic variables between non-participating and participating subjects in the study.
Table S2b. Descriptive values for sociodemographic variables at the beginning of the ALSPAC cohort, and at all the time points for this study.
Table S3. Mean and standard deviation for each class, at each time point, in relation to night-time sleep duration, bedtime and MPS.
Table S4. Spearman correlations between the three trajectories of sleep problems.
Table S5. Cross-tabulation analyses between the three poor sleep health domains to look at the interrelationship between class memberships.
Table S6. Intercorrelations between time points, for each sleep variable separately.
Table S7. Logistic regression analyses between perinatal risk factors and persistent sleep duration and sleep timing problems across childhood and adolescence (from 6 months to 15–16 years), where continuous predictors are Z-transformed.
Table S8. Multinomial regression analyses between perinatal risk factors and each of the LCGA classes, for each of the sleep variables.
Table S9. Descriptive analyses of night-time sleep duration and sleep timing, across each time point, from 5–6 to 11 years old; sensitivity analyses.
Table S10. Bayesian information criterion, Vuong–Lo–Mendell–Rubin likelihood test *p* values and entropy for classes 2–6, for night-time sleep duration, bedtime and midpoint of sleep, from 5–6 to 11 years old; sensitivity analyses.
Table S11. Logistic regression analyses between perinatal and early-life risk factors and persistent sleep duration and sleep timing problems across childhood and adolescence (from 5 to 11 years); sensitivity analyses.
Table S12. Cross-tabulation analyses for classes between the primary and the sensitivity analyses, for each sleep variable separately.
Figure S1. Descriptive patterns of sleep duration, bedtime and midpoint of sleep from infancy to adolescence.
Figure S2. Growth trajectories of night-time sleep duration, bedtime and midpoint of sleep (MPS) across childhood (from 5–6 to 11 years); sensitivity analyses.

Acknowledgements

This work was supported by The UK Medical Research Council and Wellcome (Grant: 217065/Z/19/Z) and the University of Bristol. I.M.M. and A.M.G. designed the study. I.M.M. and I.M. were primarily responsible for data analysis. I.M. drafted the initial manuscript. I.M.M, M.R.B., A.P.B., S.M. and A.M.G. reviewed and contributed critically to the writing of the article. The authors have declared that they have no competing or potential conflicts of interest.

Data availability statement

Access to ALSPAC data is through a system of managed open access (http://www.bristol.ac.uk/alspac/researchers/access/).

Correspondence

Isabel Morales-Muñoz, Institute for Mental Health, School of Psychology, University of Birmingham, 52 Pritchatts Rd, Birmingham B15 2SA, UK; Email: i.morales-munoz@bham.ac.uk

Key points

- Despite the well-known associations between sleep problems and both mental and physical health problems in young people, patterns of development and underlying factors explaining potential sleep problems including short sleep and late chronotype are under-researched in children and adolescents.
- We identified a cohort of young people characterized by persistent poor sleep health from infancy until adolescence in the form of persistent shorter sleep duration and persistent later chronotype.
- Maternal socioeconomic status and family adversities during pregnancy were the most relevant early-life risk factors.
- These results highlight the importance of measuring sleep development from early in life and provide a basis to identify those children with persistent poor sleep health who may be at risk of a range of negative outcomes.

References

Adjei, N.K., Schlüter, D.K., Straatmann, V.S., Melis, G., Fleming, K.M., McGovern, R., … & Taylor-Robinson, D.C. (2022). Impact of poverty and family adversity on adolescent health: A multi-trajectory analysis using the UK millennium cohort study. *The Lancet Regional Health. Europe*, *13*, 100279.

Barba-Müller, E., Craddock, S., Carmona, S., & Hoekzema, E. (2019). Brain plasticity in pregnancy and the postpartum period: Links to maternal caregiving and mental health. *Archives of Women's Mental Health*, *22*, 289–299.

Blunden, S., Magee, C., Attard, K., Clarkson, L., Caputi, P., & Skinner, T. (2018). Sleep schedules and school performance in indigenous Australian children. *Sleep Health*, *4*, 135–140.

Bowen, E., Heron, J., Waylen, A., & Wolke, D. (2005). Domestic violence risk during and after pregnancy: Findings from a British longitudinal study. *BJOG: An International Journal of Obstetrics and Gynaecology*, *112*, 1083–1089.

Boyd, A., Golding, J., Macleod, J., Lawlor, D.A., Fraser, A., Henderson, J., … & Davey Smith, G. (2013). Cohort profile: The 'children of the 90s'—The index offspring of the Avon longitudinal study of parents and children. *International Journal of Epidemiology*, *42*, 111–127.

Bruni, O., Baumgartner, E., Sette, S., Ancona, M., Caso, G., Di Cosimo, M.E., … & Ferri, R. (2014). Longitudinal study of sleep behavior in normal infants during the first year of life. *Journal of Clinical Sleep Medicine*, *10*, 1119–1127.

Cai, S., Tham, E.K.H., Xu, H.-Y., Fu, X., Goh, R.S.M., Gluckman, P.D., … & Broekman, B.F.P. (2023). Trajectories of reported sleep duration associate with early childhood cognitive development. *Sleep*, *46*, zsac264.

Camerota, M., Propper, C.B., & Teti, D.M. (2019). Intrinsic and extrinsic factors predicting infant sleep: Moving beyond main effects. *Developmental Review*, *53*, 100871.

Carey, W.B., & McDevitt, S.C. (1978). Revision of the infant temperament questionnaire. *Pediatrics*, *61*, 735–739.

Carno, M.-A., Hoffman, L.A., Carcillo, J.A., & Sanders, M.H. (2003). Developmental stages of sleep from birth to adolescence, common childhood sleep disorders: Overview and nursing implications. *Journal of Pediatric Nursing*, *18*, 274–283.

Cox, J.L., Holden, J.M., & Sagovsky, R. (1987). Detection of postnatal depression. Development of the 10-item Edinburgh postnatal depression scale. *The British Journal of Psychiatry: the Journal of Mental Science*, *150*, 782–786.

Dancey, C., & Reidy, J. (2004). *Statistics without maths for psychology: Using SPSS for Windows*. Upper Saddle River, NJ: Prentice Hall.

Davis, K.F., Parker, K.P., & Montgomery, G.L. (2004). Sleep in infants and young children: Part one: Normal sleep. *Journal of Pediatric Health Care*, *18*, 65–71.

Eid, B., Bou Saleh, M., Melki, I., Torbey, P.-H., Najem, J., Saber, M., … & Rabbaa Khabbaz, L. (2020). Evaluation of chronotype among children and associations with BMI, sleep, anxiety, and depression. *Frontiers in Neurology*, *11*, 416.

Enders, C.K., & Bandalos, D.L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, *8*, 430–457.

Evans, G.W., Li, D., & Whipple, S.S. (2013). Cumulative risk and child development. *Psychological Bulletin*, *139*, 1342–1396.

Fabbian, F., Zucchi, B., De Giorgi, A., Tiseo, R., Boari, B., Salmi, R., … & Manfredini, R. (2016). Chronotype, gender and general health. *Chronobiology International*, *33*, 863–882.

Fadzil, A. (2021). Factors affecting the quality of sleep in children. *Children (Basel, Switzerland)*, *8*(2), 122.

Fallone, G., Owens, J.A., & Deane, J. (2002). Sleepiness in children and adolescents: Clinical implications. *Sleep Medicine Reviews*, *6*, 287–306.

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Davey Smith, G., … & Lawlor, D.A. (2013). Cohort profile: The Avon longitudinal study of parents and children: ALSPAC mothers cohort. *International Journal of Epidemiology*, *42*, 97–110.

Galland, B.C., Taylor, B.J., Elder, D.E., & Herbison, P. (2012). Normal sleep patterns in infants and children: A systematic review of observational studies. *Sleep Medicine Reviews*, *16*, 213–222.

Grandner, M.A., Williams, N.J., Knutson, K.L., Roberts, D., & Jean-Louis, G. (2016). Sleep disparity, race/ethnicity, and socioeconomic position. *Sleep Medicine*, *18*, 7–18.

Hagenauer, M.H., Perryman, J.I., Lee, T.M., & Carskadon, M.A. (2009). Adolescent changes in the homeostatic and circadian regulation of sleep. *Developmental Neuroscience*, *31*, 276–284.

Hart, C.N., Carskadon, M.A., Considine, R.V., Fava, J.L., Lawton, J., Raynor, H.A., … & Wing, R. (2013). Changes in children's sleep duration on food intake, weight, and leptin. *Pediatrics*, *132*, e1473–e1480.

Hogan, J.W., Roy, J., & Korkontzelou, C. (2004). Handling drop-out in longitudinal studies. *Statistics in Medicine*, *23*, 1455–1497.

Huhdanpää, H., Morales-Muñoz, I., Aronen, E.T., Pölkki, P., Saarenpää-Heikkilä, O., Paunio, T., … & Juulia Paavonen, E. (2019). Sleep difficulties in infancy are associated with

symptoms of inattention and hyperactivity at the age of 5 years: A longitudinal study. *Journal of Developmental and Behavioral Pediatrics*, *40*, 432–440.
Iglowstein, I., Jenni, O.G., Molinari, L., & Largo, R.H. (2003). Sleep duration from infancy to adolescence: Reference values and generational trends. *Pediatrics*, *111*, 302–307.
Ivars, K., Nelson, N., Theodorsson, A., Theodorsson, E., Ström, J.O., & Mörelius, E. (2015). Development of salivary cortisol circadian rhythm and reference intervals in full-term infants. *PLoS One*, *10*, e0129502.
Jafar, N.K., Tham, E.K.H., Eng, D.Z.H., Goh, D.Y.T., Teoh, O.-H., Lee, Y.S., … & Broekman, B.F.P. (2017). The association between chronotype and sleep problems in preschool children. *Sleep Medicine*, *30*, 240–244.
Jike, M., Itani, O., Watanabe, N., Buysse, D.J., & Kaneita, Y. (2018). Long sleep duration and health outcomes: A systematic review, meta-analysis and meta-regression. *Sleep Medicine Reviews*, *39*, 25–36.
Joukamaa, M. (1992). Crown-crisp experiential index, a useful tool for measuring neurotic psychopathology. *Nordic Journal of Psychiatry*, *46*, 49–53.
Jung, T., & Wickrama, K.A.S. (2008). An introduction to latent class growth analysis and growth mixture modeling. *Social and Personality Psychology Compass*, *2*, 302–317.
Kantermann, T., Sung, H., & Burgess, H.J. (2015). Comparing the Morningness-Eveningness questionnaire and Munich ChronoType questionnaire to the dim light melatonin onset. *Journal of Biological Rhythms*, *30*, 449–453.
Kinner, S.A., Alati, R., Najman, J.M., & Williams, G.M. (2007). Do paternal arrest and imprisonment lead to child behaviour problems and substance use? A longitudinal analysis. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *48*, 1148–1156.
Kocevska, D., Schuurmans, I.K., Cecil, C.A.M., Jansen, P.W., van Someren, E.J.W., & Luik, A.I. (2023). A longitudinal study of stress during pregnancy, Children's sleep and polygenic risk for poor sleep in the general pediatric population. *Research on Child and Adolescent Psychopathology*, *51*, 1909–1918.
Leong, R.L.F., Lau, T., Dicom, A.R., Teo, T.B., Ong, J.L., & Chee, M.W.L. (2023). Influence of mid-afternoon nap duration and sleep parameters on memory encoding, mood, processing speed, and vigilance. *Sleep*, *46*(4), zsad025. https://doi.org/10.1093/sleep/zsad025
Li, L., Zhang, Q., Zhu, L., Zeng, G., Huang, H., Zhuge, J., … & Wu, C. (2022). Daytime naps and depression risk: A meta-analysis of observational studies. *Frontiers in Psychology*, *13*, 1051128.
Liu, J., Ji, X., Rovit, E., Pitt, S., & Lipman, T. (2022). Childhood sleep: Assessments, risk factors, and potential mechanisms. *World Journal of Pediatrics*, *20*, 105–121.
Lund, I.O., & Ystrom, E. (2022). Prenatal alcohol exposure and child sleep problems: A family-based quasi-experimental study. *JCPP Advances*, *2*, e12111.
Magee, C.A., & Blunden, S. (2020). Sleep timing during adolescence: A latent transition analysis approach. *Behavioral Sleep Medicine*, *18*, 131–146.
Magee, C.A., Gordon, R., & Caputi, P. (2014). Distinct developmental trends in sleep duration during early childhood. *Pediatrics*, *133*, e1561–e1567.
Mazza, S., Bastuji, H., & Rey, A.E. (2020). Objective and subjective assessments of sleep in children: Comparison of actigraphy, sleep diary completed by children and parents' estimation. *Frontiers in Psychiatry*, *11*, 495.
Medic, G., Wille, M., & Hemels, M.E. (2017). Short- and long-term health consequences of sleep disruption. *Nature and Science of Sleep*, *9*, 151–161.
Mindell, J.A., & Meltzer, L.J. (2008). Behavioural sleep disorders in children and adolescents. *Annals of the Academy of Medicine, Singapore*, *37*, 722–728.
Morales-Muñoz, I., & Gregory, A.M. (2023). Sleep and mental health problems in children and adolescents. *Sleep Medicine Clinics*, *18*, 245–254.
Morales-Muñoz, I., Kantojärvi, K., Uhre, V.-M., Saarenpää-Heikkilä, O., Kylliäinen, A., Pölkki, P., … & Paunio, T. (2021). The effects of genetic background for diurnal preference on sleep development in early childhood. *Nature and Science of Sleep*, *13*, 219–228.
Morales-Muñoz, I., Lemola, S., Saarenpää-Heikkilä, O., Kylliäinen, A., Pölkki, P., Paunio, T., … & Paavonen, E.J. (2020). Parent-reported early sleep problems and internalising, externalising and dysregulation symptoms in toddlers. *BMJ Paediatrics Open*, *4*, e000622.
Morales-Muñoz, I., Nolvi, S., Virta, M., Karlsson, H., Paavonen, E.J., & Karlsson, L. (2020). The longitudinal associations between temperament and sleep during the first year of life. *Infant Behavior & Development*, *61*, 101485.
Morales-Munoz, I., Partonen, T., Saarenpaa-Heikkila, O., Kylliainen, A., Polkki, P., Porkka-Heiskanen, T., … & Paavonen, E.J. (2018). The role of parental circadian preference in the onset of sleep difficulties in early childhood. *Sleep Medicine*, *54*, 223–230.
Morales-Munoz, I., Saarenpaa-Heikkila, O., Kylliainen, A., Polkki, P., Porkka-Heiskanen, T., Paunio, T., & Paavonen, E.J. (2018). The effects of maternal risk factors during pregnancy on the onset of sleep difficulties in infants at 3 months old. *Journal of Sleep Research*, *27*, e12696.
Nakagawa, M., Ohta, H., Nagaoki, Y., Shimabukuro, R., Asaka, Y., Takahashi, N., … & Yoda, H. (2016). Daytime nap controls toddlers' night time sleep. *Scientific Reports*, *6*, 27246.
Paavonen, J., Saarenpää-Heikkilä, O., MoralesMunoz, I., Virta, M., Häkälä, N., Pölkki, P., … & Karlsson, L. (2020). Normal sleep development in infants: Findings from two large birth cohorts. *Sleep Medicine*, *69*, 145–154.
Plancoulaine, S., Reynaud, E., Forhan, A., Lioret, S., Heude, B., & Charles, M.-A. (2018). Night sleep duration trajectories and associated factors among preschool children from the EDEN cohort. *Sleep Medicine*, *48*, 194–201.
Prandy, K. (1990). The revised Cambridge scale of occupations. *Sociology*, *24*, 629–655.
Price, A.M.H., Brown, J.E., Bittman, M., Wake, M., Quach, J., & Hiscock, H. (2014). Children's sleep patterns from 0 to 9 years: Australian population longitudinal study. *Archives of Disease in Childhood*, *99*, 119–125.
Punamäki, R.-L., Tammilehto, J., Flykt, M., Vänskä, M., Tiitinen, A., Poikkeus, P., & Lindblom, J. (2022). Determinants of adolescent sleep: Early family environment, obstetric factors, and emotion regulation. *Journal of Applied Developmental Psychology*, *80*, 101420.
Quach, J., Price, A.M.H., Bittman, M., & Hiscock, H. (2016). Sleep timing and child and parent outcomes in Australian 4-9-year-olds: A cross-sectional and longitudinal study. *Sleep Medicine*, *22*, 39–46.
RCPCH. (2022). Child health inequalities driven by child poverty in the UK – Position statement. Available from: https://www.rcpch.ac.uk/resources/child-health-inequalities-position-statement#rcpch-recommendations-to-reduce-health-inequalities-as-a-result-of-child-poverty. [last accessed 5 July 2023].
Rivkees, S.A. (2003). Developing circadian rhythmicity in infants. *Pediatric Endocrinology Reviews*, *1*, 38–45.
Sadeh, A., Mindell, J.A., Luedtke, K., & Wiegand, B. (2009). Sleep and sleep ecology in the first 3 years: A web-based study. *Journal of Sleep Research*, *18*, 60–73.
Schickedanz, A., Escarce, J.J., Halfon, N., Sastry, N., & Chung, P.J. (2021). Intergenerational associations between parents' and children's adverse childhood experience scores. *Children (Basel, Switzerland)*, *8*(9), 747.
Schultz, L.F., Kroll, C., Constantino, B., Trombelli, M.C.M.C., El Rafihi-Ferreira, R., & Mastroeni, M.F. (2020). Association

of Maternal Depression and Anxiety Symptoms with sleep duration in children at preschool age. *Maternal and Child Health Journal*, *24*, 62–72.
Shen, C., Mireku, M.O., Di Simplicio, M., Dumontheil, I., Thomas, M.S.C., Röösli, M., … & Toledano, M.B. (2022). Bidirectional associations between sleep problems and behavioural difficulties and health-related quality of life in adolescents: Evidence from the SCAMP longitudinal cohort study. *JCPP Advances*, *2*, e12098.
Shimizu, M., Zeringue, M.M., Erath, S.A., Hinnant, J.B., & El-Sheikh, M. (2021). Trajectories of sleep problems in childhood: Associations with mental health in adolescence. *Sleep*, *44*(3), zsaa190. https://doi.org/10.1093/sleep/zsaa190
Simpkin, C.T., Jenni, O.G., Carskadon, M.A., Wright, K.P.J., Akacem, L.D., Garlo, K.G., & LeBourgeois, M.K. (2014). Chronotype is associated with the timing of the circadian clock and sleep in toddlers. *Journal of Sleep Research*, *23*, 397–405.
Souabni, M., Hammouda, O., Romdhani, M., Trabelsi, K., Ammar, A., & Driss, T. (2021). Benefits of daytime napping opportunity on physical and cognitive performances in physically active participants: A systematic review. *Sports medicine (Auckland, N.Z.)*, *51*, 2115–2146.
Staton, S., Rankin, P.S., Harding, M., Smith, S.S., Westwood, E., LeBourgeois, M.K., & Thorpe, K.J. (2020). Many naps, one nap, none: A systematic review and meta-analysis of napping patterns in children 0–12 years. *Sleep Medicine Reviews*, *50*, 101247.
Steer, C., & Wolke, D. (2004). An index of family adversity. Poster presented on the 3rd conference of epidemiological longitudinal studies in Europe.
Sullivan, K., Rochani, H., Huang, L.-T., Donley, D.K., & Zhang, J. (2019). Adverse childhood experiences affect sleep duration for up to 50 years later. *Sleep*, *42*(7), zsz087. https://doi.org/10.1093/sleep/zsz087
Taylor, B.J., & Hasler, B.P. (2018). Chronotype and mental health: Recent advances. *Current Psychiatry Reports*, *20*, 59.
Tham, E.K.H., Xu, H.-Y., Fu, X., Schneider, N., Goh, D.Y.T., Lek, N., … & Broekman, B.F.P. (2021). Variations in longitudinal sleep duration trajectories from infancy to early childhood. *Sleep Health*, *7*, 56–64.
Touchette, E., Côté, S.M., Petit, D., Liu, X., Boivin, M., Falissard, B., … & Montplaisir, J.Y. (2009). Short nighttime sleep-duration and hyperactivity trajectories in early childhood. *Pediatrics*, *124*, e985–e993.
Touchette, E., Servot, S., Lemieux, R., & Berthelot, N. (2020). 0859 types of childhood maltreatment and sleep regulation during pregnancy. *Sleep*, *43*(Supplement_1), A327.
Tuohino, T., Morales-Muñoz, I., Saarenpää-Heikkilä, O., Kiviruusu, O., Paunio, T., Hovi, P., … & Paavonen, E.J. (2019). Short sleep duration and later overweight in infants. *Journal of Pediatrics*, *212*, 13–19.
Tyler, D., Donovan, C.L., Scupham, S., Shiels, A.L., & Weaver, S.A. (2019). Young Children's sleep problems: The impact of parental distress and parenting style. *Journal of Child and Family Studies*, *28*, 2098–2106.
Urbanek, J.K., Spira, A.P., Di, J., Leroux, A., Crainiceanu, C., & Zipunnikov, V. (2018). Epidemiology of objectively measured bedtime and chronotype in US adolescents and adults: NHANES 2003-2006. *Chronobiology International*, *35*, 416–434.
Weightman, A.L., Morgan, H.E., Shepherd, M.A., Kitcher, H., Roberts, C., & Dunstan, F.D. (2012). Social inequality and infant health in the UK: Systematic review and meta-analyses. *BMJ Open*, *2*, e000964.
Werner, H., Lebourgeois, M.K., Geiger, A., & Jenni, O.G. (2009). Assessment of chronotype in four- to eleven-year-old children: Reliability and validity of the children's chronotype questionnaire (CCTQ). *Chronobiology International*, *26*, 992–1014.
Wiggins, J.L., Mitchell, C., Hyde, L.W., & Monk, C.S. (2015). Identifying early pathways of risk and resilience: The codevelopment of internalizing and externalizing symptoms and the role of harsh parenting. *Development and Psychopathology*, *27*(4 Pt 1), 1295–1312.
Williamson, A.A., Mindell, J.A., Hiscock, H., & Quach, J. (2019). Sleep problem trajectories and cumulative socioecological risks: Birth to school-age. *The Journal of Pediatrics*, *215*, 229–237.
Wood, D., Crapnell, T., Lau, L., Bennett, A., Lotstein, D., Ferris, M., & Kuo, A. (2018). Emerging adulthood as a critical stage in the life course BT. In N. Halfon, C.B. Forrest, R.M. Lerner, & E.M. Faustman (Eds.), *Handbook of life course health development* (pp. 123–143). Cham, Switzerland: Springer International Publishing.
Zerbini, G., & Merrow, M. (2017). Time to learn: How chronotype impacts education. *PsyCh Journal*, *6*, 263–276.
Zheng, M., Hesketh, K.D., Wu, J.H.Y., Heitmann, B.L., Downing, K., & Campbell, K.J. (2021). Nighttime sleep duration trajectories were associated with body mass index trajectories in early childhood. *Pediatric Obesity*, *16*, e12766.

Accepted for publication: 25 March 2024