SLEEP, 2026, **49**, 1–16
doi:10.1093/sleep/zsag161
Advance Access publication 13 June 2026
Original Article

Original Article

Charting infant sleep cycle development using actigraphy: longitudinal evidence for ultradian cycle lengthening within the first year of life from 35 000 h of sleep

Grégory Hammad [1,2,3,*,†], Sarah F. Schoch [4,†], Maximilian Engelmann[5], Zoe Spock [2], Salome Kurth[6,7,‡] and Eva C. Winnebeck [1,2,8,*,‡]

[1]Neurogenetics, School of Medicine and Health, Technical University of Munich, Munich, Germany, [2]Section of Chronobiology, Faculty of Health and Medical Sciences, University of Surrey, Guildford, United Kingdom, [3]GIGA - CRC human imaging, University of Liège, Liège, Belgium, [4]Radboud University Medical Center, Institute for Brain, Cognition and Behavior, Nijmegen, Netherlands, [5]Institute of Medical Psychology, Faculty of Medicine, Ludwig Maximilian University Munich, Munich, Germany, [6]Department of Psychology, University of Fribourg, Fribourg, Switzerland, [7]Department of Pulmonology, University Hospital Zurich, Zurich, Switzerland and [8]Institute of Neurogenomics, Computational Health Center, Helmholtz Center Munich, Munich, Germany

*Corresponding authors. Grégory Hammad, Email: gregory.hammad@uliege.be; Eva C. Winnebeck, Email: e.winnebeck@surrey.ac.uk.
†Grégory Hammad and Sarah F. Schoch contributed equally to this work and share first authorship.
‡Salome Kurth and Eva C.Winnebeck contributed equally to this work and share last authorship.

Abstract

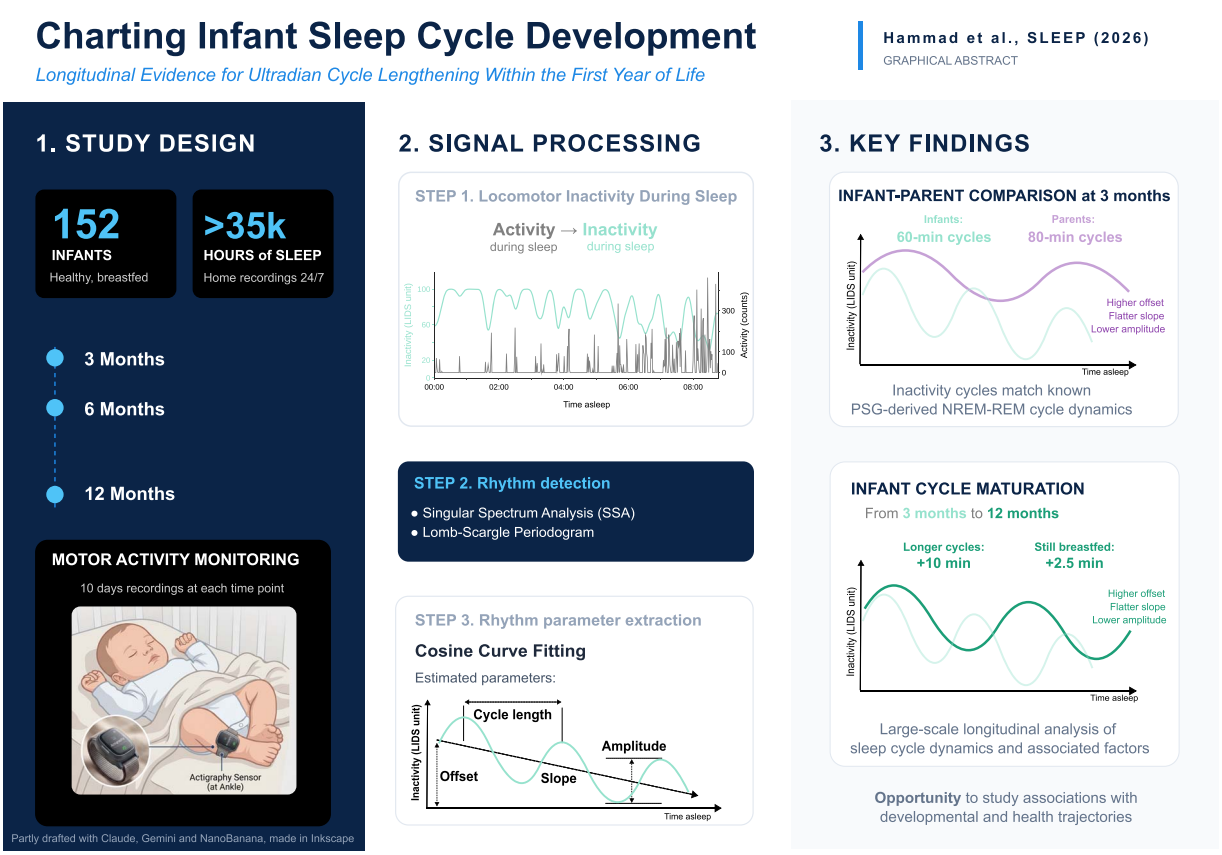
Sleep is marked by ultradian cycles coinciding with the alternation of rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. These sleep cycles are a striking feature exhibited from infancy through adulthood, yet their underlying mechanisms and functional relevance remain elusive, calling for large-scale longitudinal studies. Here, we leverage Locomotor Inactivity During Sleep (LIDS), an accessible marker of sleep cycles in adults, to chart ultradian sleep cycle dynamics in infants at scale. Specifically, we analyzed > 35 000 h of sleep from a longitudinal dataset of 152 infants with actigraphy at 3, 6, and 12 months of age. Using complementary signal processing techniques, we demonstrate the existence of rhythmic patterns in infant LIDS with cycle lengths of approximately 60 min. Cycles were shorter in infants than parents (62 min 95%CI [56, 67] vs. 81 min [74, 88]) and increased by approximately 10 min from 3 to 12 months, mirroring previous results in smaller samples for NREM–REM sleep cycles. This increase was partially mediated by increasing sleep bout duration (1.0 min/h [0.9,1.2]). Longer cycles were also found in infants still breastfed at 12 months (2.5 min [0.4, 4.5]) and their breastfeeding mothers (6.7 min [0.5, 12.9]). Furthermore, inactivity was lower at sleep onset, declined more rapidly and showed a greater amplitude in infants than parents, but all three parameters began to mature already within the first year. Overall, our results support a link between patterns in limb inactivity and sleep cycle physiology in infancy, underscoring the potential of studying these in large-scale cohorts for developmental and health outcomes.

Key words: infant; ultradian; sleep; NREM–REM cycles; actigraphy; wearables; early childhood; baby; sleep regulation; longitudinal

Submitted: 14 January, 2026; **Revised:** 2 May, 2026; **Accepted:** 27 May, 2026
© The Author(s) 2026. Published by Oxford University Press on behalf of Sleep Research Society.
This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (https://creativecommons.org/licenses/by-nc/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact reprints@oup.com for reprints and translation rights for reprints. All other permissions can be obtained through our RightsLink service via the Permissions link on the article page on our site—for further information please contact journals.permissions@oup.com.

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Graphical Abstract



Statement of Significance

Infancy is a time of profound change in sleep, spurring theories about underlying drivers, and links with developmental outcomes. Studying sleep repeatedly at this early age, however, is difficult, but wearables may help to overcome the data shortage. Here, we used easy-to-record limb movement from ankle actigraphs as a window into infant sleep physiology, going beyond its established use in sleep–wake monitoring. We demonstrate the presence of 60-min cycles in movement during sleep compatible with the pioneering discoveries of alternating rapid eye movement (REM) and non-REM (NREM) sleep in infants. The gradual lengthening of these cycles in our large sample underscores previous evidence of NREM–REM cycle lengthening during infancy, paving the way for studies linking cycle dynamics with developmental trajectories in large birth cohorts.

Introduction

Ultradian rhythms, biological rhythms with cycle lengths in the hour range and no obvious environmental synchronizers, are manifest across the animal kingdom [1] and across biochemical, physiological, and behavioral processes [2, 3] such as gene expression [4, 5], hormone release [6], body temperature [7], feeding habits [8], and motor activity [9, 10]. Despite the ubiquity of ultradian rhythms, their underlying generative mechanisms are largely unknown [11], and their biological function(s) remains equally elusive [12].

Around 70 years ago, in 1955, Aserinsky and Kleitman observed ultradian rhythms in "bodily movements" in infants during sleep, synchronized with the peculiar ultradian rhythms of ocular motility they had previously noticed [13], confirming an even earlier report of this phenomenon by Denisova [14, 15]. Two years later, Dement and Kleitman described in detail similar observations in adults: brain activity, eye, and body movements vary in an ultradian cyclic manner throughout the night [16]. These discoveries led to the first definition of ultradian sleep cycles based on cyclic changes in sleep physiology. Following the shift to an EEG-oriented definition of sleep and sleep stages, Feinberg and Floyd updated this early definition in 1979 [17], which is still in use today: Cycles are defined as the alternation between non-rapid eye movement (NREM) sleep periods (including all stages of NREM sleep and intermittent wake) and REM sleep periods (REM sleep and intermittent wake over 5 min total) and are accordingly called NREM–REM cycles. A recent retrospective study of approximately 6000 polysomnographic recordings reported a median sleep cycle length of 96 min in adults with wide variation in cycle length between and within individuals [18].

Despite these early observations of ultradian rhythms in sleep, much about their cyclic dynamics remains elusive today. As for other ultradian rhythms, mechanisms of sleep cycle generation and the functional relevance of rhythm characteristics are still

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

enigmatic (although see recent advances on their regulation in rodents [19]). This lack of understanding may be largely driven by the difficulty of monitoring sleep cycles at scale, which is necessary to overcome the variability in cycle dynamics characteristic of all ultradian rhythms. Hence, using simpler recording methods than polysomnography that can be more easily deployed in large numbers of individuals, longitudinally and in everyday environments, appears to be an intuitive path to gaining more data and thus a better understanding of ultradian sleep cycles.

In 2018, we demonstrated that Locomotor Inactivity During Sleep (LIDS), derived from simple wrist–worn actigraphy in everyday life, is an easy method to detect ultradian rhythms in movement during sleep in school-age children, adolescents, and adults, and their dynamics are highly similar to NREM–REM sleep cycles assessed with polysomnography [20]. The detected inactivity rhythms oscillate with a cycle length of approximately 90–110 min and show characteristic variability, while the mean inactivity levels were found to decline progressively overnight. This decline as well as the oscillation amplitudes were reduced with increasing age, suggesting not only usefulness for ultradian monitoring but also potentially for depth of sleep and homeostatic sleep pressure. Here, we show that the LIDS methodology can also be applied during a critical developmental period in life that is marked by rapid and substantial changes in sleep: infancy.

During the first year of life, sleep undergoes remarkable development: starting from a polyphasic sleep–wake pattern after birth with multiple short sleep bouts scattered almost evenly across the 24-h day, sleep bouts gradually consolidate, and move increasingly into the night [21–23]. This change in sleep–wake organization is attributed to the maturation of two important sleep regulatory systems: the circadian system, which promotes wakefulness during the biological day and sleep during the biological night, and the homeostatic system, which tracks the wake-dependent accumulation of sleep pressure [24, 25].

In early infancy, sleep states are commonly referred to as quiet (later: NREM) and active (later: REM) sleep, suggesting differences in motor behavior which help demarcate sleep cycles, which also initially led to their discovery by Denisova and by Aserinsky [13–15]. Only after the first two to three months of life do the differences in EEG appear that clearly distinguish REM and NREM sleep and the NREM substages. This is intriguing as it might indicate that rhythmic motor activity predates the appearance of specific brain activity patterns that allow the classification of sleep epochs into discrete stages. It might also suggest that while sleep physiology is still maturing, the ultradian cycling mechanism is already functional at an early life stage and that its manifestation might be greater in motor activity than in brain activity. Indeed, monitoring of infants' breathing and body movements with a pressure sensitive mattress [26] revealed cycles in quiet sleep with mean cycle lengths of 63 min at two to five postnatal weeks. Evidence for the presence of such cycles as early as the first postnatal days was also put forward by Freudigman and Thoman in 1994 [27]. Therefore, already from the first weeks of life, infant sleep alternates between two states [28], a clear manifestation of ultradian sleep cycles. These sleep cycles are on average 50–60 min long [13–15, 28–31] and thus markedly shorter than the 90–110-min cycles observed in adults. Studies based on small or cross-sectional samples suggest that cycles may gradually lengthen over the course of infancy and early childhood to reach adult-length cycles by school-age [30–32]. However, strong longitudinal evidence is lacking given the cumbersome nature of polysomnographic recordings especially in this age group.

Here, we used a longitudinal infant and parent dataset with actigraphy recordings at 3, 6, and 12 months after birth to explore the potential of using motor patterns during sleep via our LIDS methodology to study infant sleep dynamics, particularly those of ultradian sleep cycles. We address multiple questions: Are there ultradian rhythms detectable in infant inactivity during sleep as early as 3 months of age? Do these rhythms show characteristics typical of NREM–REM cycles at this age, i.e. a cycle length around 50–60 min with high variability and shorter lengths than those in adults and their parents? And finally, how do these ultradian rhythms change over the first year of an infant's life, both in infants and parents? For the latter question, we explored not only cycle lengthening with age but also associations with multiple other key endogenous and exogenous factors including sex, sleep–wake cycle maturation, breastfeeding, and sleep location, which are held to influence sleep quality but have never been systematically assessed in large datasets for links with ultradian sleep cycles.

Materials and methods

Participants

The work presented here analyzed data from a longitudinal study of infant sleep and gut microbiota [33–35], in which a total of 162 infants living mostly in the area of Zurich were enrolled. In addition, 100 of the infants' parents (74 mothers and 26 fathers) opted to contribute their own data to complement the data of their infant. Infants were in good general health, initially breastfed, and vaginally born between 37 and 43 weeks of gestation. Of the 152 families contributing actigraphy data (see below), 137 (90.1%) families reported a university degree (Bachelor/Master: $n = 103$, 67.8%; PhD: $n = 34$, 22.4%) and 15 (9.9%) an apprenticeship as the highest educational level for at least one parent. Details about inclusion and exclusion criteria are provided in [33–35]. Ethical approval was obtained from the cantonal ethics committee (BASEC 2016-00730), and the study was carried out in accordance with the Declaration of Helsinki. Parents provided written informed consent. Families received small non-monetary gifts for their participation.

Data collection

Activity data were recorded at 3, 6, and 12 months of age over 10 consecutive days each for a subset of 152 infant participants ($n = 150$ at 3 months, 148 at 6 months, and 143 at 12 months) and their parents ($n = 100$ at 3 months, 79 at 6 months, and 71 at 12 months), using actigraphy devices (GeneActiv, Activinsights Ltd, Kimbolton, UK). See Tables 1 and S1 for participant details. Parents were instructed to attach their own device to the non-dominant hand and the infant's device to the ankle using a modified sock or a Tyvek paper strap. In addition, parents completed 24-h diaries to document infant sleep episodes as well as times when the device was removed (primarily during bathing and swimming).

Feeding, sleeping habits as well as infants' sleep location were also reported by the parents using questionnaires. At each time point, breastfeeding frequency was assessed on a 5-item scale, ranging from "1:never" to "3:occasionally (1–2 times a week)," up to "4:regularly (3–5 times a week)" and "5:daily." For the purposes of our analysis, breastfeeding was coded as "No" for levels 1 to 3 and as "Yes" for levels 4 and 5. Parents were also asked to describe their infant's sleep location using a questionnaire providing 15 different sleep configurations (e.g. "Infant bed in separate room" or "In parents' bed"). Here, these were classified with respect to their

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Table 1. Participant summary statistics. Number and frequency of participant characteristics in the infant and parental sample per time point.

			Time points		
			3 months	6 months	12 months
Infants					
Total		*n*	150	148	143
Females		*n* (%)	67 (44.7%)	67 (45.3%)	64 (44.7%)
Breastfed		*n* (%)	150 (100%)	137 (92.6%)	51 (36.4%)*
Sleep location	Separate room	*n* (%)	20 (13.3%)	41 (27.9%)	83 (58.0%)
	Separate bed	*n* (%)	72 (48.0%)	61 (41.5%)	33 (23.1%)
	Parents' bed	*n* (%)	31 (20.7%)	29 (19.7%)	26 (18.2%)
	Attached cot	*n* (%)	27 (18.0%)	16 (10.9%)	1 (0.7%)
Nighttime sleep (%)		Median [IQR]	72.3 [69.5, 72.5]	78.6 [76.1,80.9]	82.5 [80.1, 84.9]
Parents					
Total		*n*	100	79	71
Mothers		*n* (%)	74 (74%)	61 (77.2%)	56 (78.9%)
Families with diadic data		*n*	54	45	40
Families with triadic data		*n*	23	17	15

*out of 140.
Infants were considered breastfed if breastfeeding frequency was reported as at least "4:regularly (3–5 times a week)." Nighttime sleep indicates the relative amount of daily sleep occurring during nighttime per infant per time point. A family was counted as providing diadic or triadic data whenever data from the infant plus one parent (diadic) or infant plus both parents (triadic) were available for the same time point. Abbreviation: IQR, interquartile range.

parents as "Separate room" or "Same room." The latter category was further divided into "Separate bed," "Same bed" or "Attached cot" (a semi-enclosed cot attached to the parental bed).

Actigraphy processing

Actigraphy data pre-processing as well as sleep bout detection were performed with Matlab (R2016b), while all subsequent processing steps were performed in Python with the *pyActigraphy* software (v0.2) [36].

Pre-processing. As described elsewhere [33], raw accelerometry data acquired at a sampling frequency of 30 Hz were converted to activity counts in order to use validated sleep detection algorithms. In brief, following the method by Te Lindert *et al.* [37], a 3–11 Hz bandpass Butterworth (order 5) filter was applied to the magnitude of the acceleration vector along the 3 axes, which was then converted into counts by discretizing this value between 0 and 5 g into 128 bins and summing the peak value measured every second over a period length of 1 min.

Sleep bout detection and selection. Bouts of likely sleep (relative immobility), henceforth referred to as "sleep" for simplicity, were automatically identified from actigraphy recordings using the same procedure as in prior publications of this dataset, a modified version [33] of the algorithm developed by Sadeh and colleagues (1994) [38]. Modifications improved agreement between diary and actigraphy data and included an adapted threshold and a consolidation step where short active bouts (≤5 min) surrounded by sleep were relabeled as sleep. Biases in recorded activity levels were reduced by excluding bouts where the infant was reportedly sick. Finally, from the sleep–wake scoring, the nighttime sleep percentage was calculated per infant per time point as the fraction of sleep duration occurring in the 12 h between 19:00 and 07:00 with respect to the total sleep duration.

From all such estimated sleep bouts, we then selected only those occurring during the putative main sleep episode (defined as starting between 18:00 and 08:00) to minimize recording artifacts from external movement common during daytime at that age (from strollers, prams, carriers, etc.). We further selected bouts of a minimum duration of 90 min to enable adequate characterization of rhythms in the range of 40–90 min as informed by singular spectrum analysis (SSA, see below). To prevent undue loss of data from this 90-min criterion and bias towards consolidated sleepers and later time points, we fused sleep bouts that were less than 15 min apart by padding the gaps with "Not a Number" (NaN) to represent missing data and retain the original temporal distance between the recorded activity patterns. Fused bouts were only included if they contained not more than 20% NaNs across their final duration. As a result, we were able to retain a median of 83%–88% of sleep data per infant across all three time points (Tables 2 and S2).

Locomotor inactivity during sleep. To study ultradian rhythms during sleep based on movement, activity counts during nighttime sleep bouts were converted to LIDS following the procedure first laid out in Winnebeck *et al.* [20] with minor optimizing modifications. Formally, each detected sleep bout was processed as follows:

1. Non-linear transformation of activity to inactivity: $\mathrm{LIDS}_i = 100/(1 + x_i)$, where x_i is the activity count at epoch i.
2. Smoothing using a Gaussian kernel with a standard deviation (σ) of 5 min within a 30-min sliding window ($[-3\sigma, +3\sigma]$).

An inactivity count of 100 represents an absence of movements, while the count tends to zero as the quantity (intensity and frequency) of movements increases. For ease of understanding, we refer to LIDS as "inactivity" throughout the manuscript outside of technical descriptions.

Singular spectrum analysis (SSA). To establish the presence of an oscillatory component in the infant inactivity signal, SSA was performed. To avoid misinterpreting noise components as significant regular oscillations, a Monte Carlo SSA (MC-SSA) hypothesis test [39] from the python software *MCSSA* (v0.0.1) [40] was also used.

SSA aims to construct an additive decomposition of the original signal into a trend, regular oscillations, and a noise component [41]. Each component is associated with a singular value accounting for its partial variance. The MC-SSA then compares this signal

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Table 2. Infant sleep bout summary statistics. Number and duration of actigraphically identified nighttime sleep bouts of infants before and after processing and application of inclusion criteria.

		Time points		
		3 months	**6 months**	**12 months**
Recording length (d)				
Total/infant	Mean ± *SD*	11.1 ± 1.2	10.6 ± 1.9	10.6 ± 1.9
Identified bouts				
Sleep bouts (*n*)				
Total		8491	5600	3673
Total/infant	Median [IQR]	57 [46,68]	38 [28,45]	24 [19,31]
Total/infant/night	Median [IQR]	5 [4,7]	4 [3,5]	2 [1,4]
Bout duration (h)				
Total		14 698	13 743	13 565
Total/infant/night	Median [IQR]	9.9 [9.1,10.7]	10.2 [9.4,10.9]	10.6 [9.8,11.3]
Median/infant/night	Median [IQR]	1.1 [0.8,1.9]	2.1 [1.2,3.5]	4.4 [2.0,6.1]
Analyzed bouts (fused if within 15 min; >90 min; 1 <offset< 99)				
Sleep bouts (*n*)				
Total		2479	1940	1524
Total/infant	Median [IQR]	17 [13,20]	13 [10,15]	10 [8,13]
Total/infant/night	Median [IQR]	2 [1,2]	1 [1,2]	1 [1,2]
Bout duration (h)				
Total		11 940	11 978	11 709
Total/infant/night	Median [IQR]	8.9 [7.1,10.1]	9.8 [8.6,10.7]	10.3 [9.4,11.1]
Median/infant/night	Median [IQR]	4.8 [3.4,8.0]	7.0 [4.6,10.0]	9.9 [5.6,10.8]

"Identified bouts" refers to the set of sleep bouts as detected by the modified Sadeh algorithm. "Analyzed bouts" refers to the final set of sleep bouts used in the inactivity analysis, after fusing of bouts within 15 min of each other and filtering for a 90-min minimum duration and a cosine fit offset outside of extreme values. Abbreviations: SD, standard deviation; IQR, interquartile range.

decomposition to the decomposition obtained from an ensemble of random time series generated from a first-order autoregressive process (red noise). For each component, it is then possible to test the null hypothesis that this component originates from stochastic fluctuations.

We chose an SSA embedding window length of 2.5 h to resolve periodicities of up to 150 min. This meant that the required minimum sleep bout duration for SSA was 5 h, i.e. double the window length, as recommended for maximal separation of oscillations and noise [42]. Selecting for sleep bouts >5 h reduced the overall infant sample to 127 but allowed the analysis of a median of 4, 5, and 8 bouts in each of these at 3, 6, and 12 months, respectively. Components with the 15 highest partial variances were kept, and components with a relative difference below 0.15 between their singular values were merged. These components were then statistically tested with MCSSA. Only statistically significant components were retained. Finally, to coarsely characterize each component, the main cycle length associated with its maximal power was derived with a Fast Fourier Transform.

Lomb-Scargle periodogram. To cross-check the presence of oscillations in infant inactivity, we also ran Lomb-Scargle periodograms [43, 44] as implemented in the R package *MetaCycle* [45]. For each sleep bout's inactivity signal, the estimated cycle length was determined as the one corresponding to the highest periodogram peak. The null hypothesis that such a peak arose from white noise was rejected at the alpha-threshold of 0.05. Two detection ranges (30–180 min, 15–240 min) were used as coarse assessment of the robustness of the estimated periods.

Cosine curve fitting for LIDS parameter estimation. To quantify central features of the inactivity signal beyond cycle length (i.e. amplitude, phase, level at start and slope), a sloped cosine model was fitted to the LIDS data using a non-linear least-squares minimization from the package *lmfit* (1.0.3) [46]):

$$f(t) = \alpha \cos\left(\frac{2\pi}{T_{LIDS}}t\right) + \beta \sin\left(\frac{2\pi}{T_{LIDS}}t\right) + b_{LIDS} + s_{LIDS} \times t$$

with

$A_{LIDS} = \sqrt{\alpha^2 + \beta^2}$: the LIDS amplitude, T_{LIDS}: the LIDS cycle length or period, $\phi_{LIDS} = -1 \times atan2(\beta, \alpha)$: the LIDS phase at bout start (0 = peak at bout start; 180 = through at bout start), b_{LIDS}: the LIDS offset or vertical shift or inactivity level at bout start, s_{LIDS}: the LIDS slope or linear trend across the bout.

Similar to the approach in the original study, the fit procedure was performed iteratively; for each iteration, estimations of the model parameters were obtained by minimizing the sum of squared residuals, while the cycle length was fixed to a value ranging from 30 to 180 min in steps of 2 min. The selected best fit was the one corresponding to the peak value of the Munich Rhythmicity Index (MRI), defined as: $\mathbf{MRI} = 2 \times A_{LIDS} \times r$, with r, the Pearson correlation coefficient between the inactivity data and the fitted model. Only sleep bouts with an offset greater than 1 and smaller than 99 were retained for statistical analyses to remove bouts with spurious activity patterns (e.g. no activity throughout the bout due to loss of the device during sleep).

Systematic uncertainties. In addition to reporting estimated mean parameter values and their statistical uncertainties (mainly arising from finite sample sizes), we also analyzed the systematic uncertainties arising from the methodological choices. Since the latter cannot simply be reduced by repeating the experiment and/or increasing sample size, their assessment is crucial to compare current results with those obtained with different methodologies from past and future studies. See Supplementary Information for details and results.

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Statistical analysis

Generalized Bayesian linear mixed models, accounting for the repeated measures nature of the data, were used to test for relationships between cosine-derived LIDS parameters (outcomes) and internal and external factors (predictors) such as time point, sex, sleep bout duration, percentage of nighttime sleep, breastfeeding, sleep location, and/or family role.

First, for simple comparisons between infant and parent LIDS parameters at time point 1, models included a random intercept (u_j) for each family (j) and as predictors the status (infant or parent), bout duration, and their interaction for each participant (i):

$$\begin{aligned}\eta_{ij} = \beta_0 + u_j \\ + (\beta_1 + u_{j1}) * (\text{Status} == \text{Parent}) \\ + (\beta_2 + \beta_3 * (\text{Status} == \text{Parent})) * \text{Bout duration} \\ + \epsilon_{ij}\end{aligned}$$

with β_0 the estimated average LIDS parameter value for infants, β_1 the estimated difference in the average LIDS parameter value between parents and infants, β_2 and $\beta_2+\beta_3$ the estimated increase in the average LIDS parameter value with sleep bout duration (h) for infants and parents, respectively, and ϵ the matrix of residuals.

Second, for more detailed analyses of LIDS parameters in infants across all bouts including differences between time points, models included a random intercept (u_i) for each participant (i) and a random slope (u_{ij}) for each time point (j), with time point, sex and sleep location as categorical predictor variables and sleep bout duration as well as percentage of nighttime sleep as continuous predictor variables (fixed-effects). The general model structure was:

$$\begin{aligned}\eta_{ij} = \beta_0 + u_i \\ + (\beta_1 + u_{i1}) * (\text{Time point} == \text{6 months}) \\ + (\beta_2 + u_{i2}) * (\text{Time point} == \text{12 months}) \\ + \gamma X_{ij} + \epsilon_{ij}\end{aligned}$$

with β_0 the estimated average LIDS parameter value, β_1 and β_2 the estimated differences in the average LIDS parameter value at 6 months and 12 months compared with 3 months, γ the matrix of additional fixed-effects coefficients, X the matrix of their associated predictor variables, and ϵ the matrix of residuals.

The association between breastfeeding and LIDS parameters was also investigated with the above model, separately in infants and in mothers. This was done using data collected at 12 months only since there was little variation in breast-feeding frequency at the other time points. Hence, the model did not include time point as a predictor, but breastfeeding (yes/no) was added as categorical predictor.

Third, a similar model was used to investigate the longitudinal modifications of LIDS parameters in parents. This model included a random intercept per family; random slopes for each time point; time points, role (mother/father), and sleep location as categorical predictors; sleep bout duration as continuous predictor; and an interaction between role and time points.

Across all models, the linear predictor, η, was related to the outcome variable y (LIDS parameters) with the link function g:

$$g(\mathbb{E}(y)) = \eta$$

In parents, LIDS cycle length, amplitude, slope, and offset were modeled with a Student's T distribution together with an identity link function. In infants, this was the same except for LIDS offset, for which a Gaussian distribution was the better fit. In all models for LIDS phase, the Von Mises circular distribution was used with a classic arctan link function.

A Bayesian mediation analysis [47] was performed to investigate whether increases in sleep bout duration indirectly mediated the LIDS cycle length increase with time. The model used was identical to the default model for the longitudinal analysis. However, for simplicity, time points were converted into months and used as a continuous independent variable.

All statistical analyses were performed in Python 3.8.10, using the Pandas (1.5.3) and Numpy (1.22.4) packages for data manipulation. Statistical Bayesian analyses were conducted with the Bambi (v0.13.0) software [48]. Unless stated otherwise, default weakly informative Gaussian priors were used. Posterior distributions were estimated with a Markov Chain Monte Carlo (MCMC) technique, sampling four independent chains in parallel, with 5000 draws for each chain. Convergence and overlap between chains were considered acceptable if the potential scale reduction factor ($\hat{R}$) was smaller than 1.01. For each model parameter (β), the estimated posterior distribution was considered stable if its effective sample size was higher than 90% of the total number of draws (4 × 5000). Model adequacy was visually checked by computing the mean posterior predictive distribution. Finally, for each posterior distribution, we report its mean, standard deviation, and 95% highest density interval (HDI) as the 95% credible interval (CI) in detailed tables in the Supplementary Information, and, for key numbers, its mean and the 95% CI also in the main text. Model parameter estimates were considered statistically significant if their 95% CI did not overlap with zero.

Results

Inactivity patterns in >35 000 h of nighttime sleep were analyzed in detail in 152 infants during their first year of life, based on around 10 days of ankle-actigraphy recordings at 3, 6, and 12 months of age. Almost all infants contributed to all three time points, and a consistent 45% were female (Tables 1 and 2). Additionally, in >50% of families, at least one of the parents also contributed wrist-actigraphy recordings, providing a total of >16 000 h of nighttime sleep recordings for parental inactivity pattern analyses. Mothers were the main contributors to the parental sample (74%–79%). See Tables 1, S1, and S3 for more details on the parental sample and their sleep activity data contribution.

The actigraphically detected sleep–wake structure in the infant sample and its developmental changes were well aligned with those reported in other infant cohorts (e.g. Rise&Shine cohort [49] and CHILD–SLEEP cohort [50]). Overall, sleep became progressively less polyphasic and more consolidated and moved more into the night as infants became older. The proportion of daily sleep occurring at night increased from a median of 72.3% at 3 months to 82.5% at 12 months (Table 1), paralleled by an increase in the total duration of nighttime sleep from a median of 9.9 h per night per infant to 10.6h over the same time (Table 2). Furthermore, nighttime sleep markedly consolidated over the assessment period. The median number of detected sleep bouts per night per infant decreased from 5 bouts at 3 months to 2 bouts at 12 months, with the median bout duration lengthening by 3.3 h, from 1.1 h to 4.4 h over this period (Table 2). After bout fusing for an optimal and minimally biased inactivity rhythm analysis (see Materials and Methods), sleep bout durations increased

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

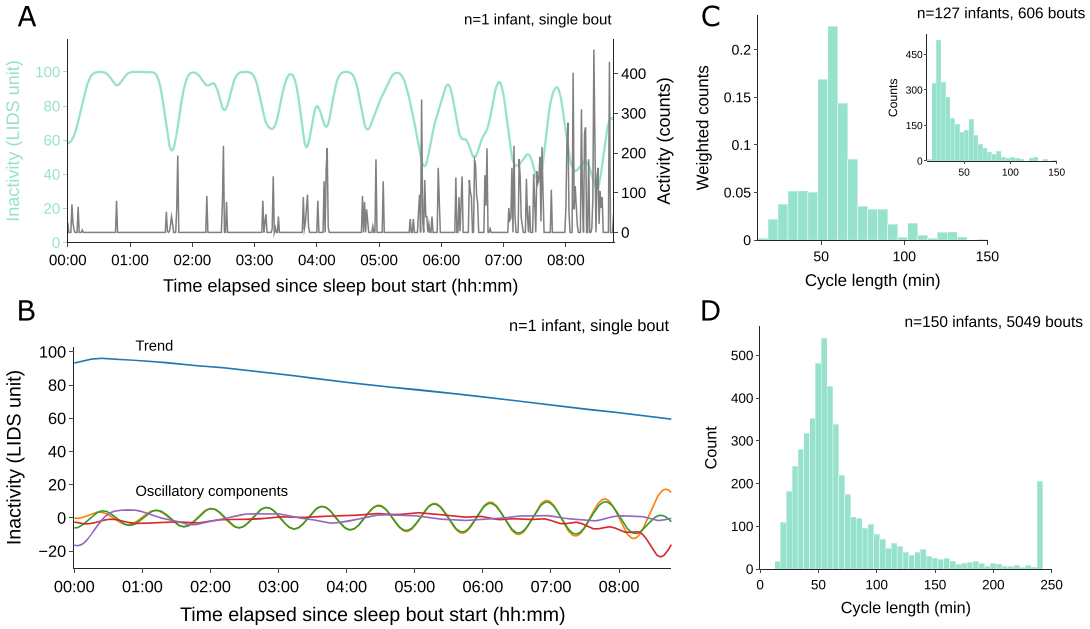


Figure 1. LIDS in 3-month old infants oscillates with an ultradian cycle length of approximately 60 min. (A) Example infant sleep bout showing locomotor activity (gray, right y-axis) and respective inactivity signal (turquoise, left y-axis). (B) Decomposition of the example inactivity signal from (A) into a trend and oscillatory components via SSA. (C) Distributions of the main cycle lengths of statistically significant oscillatory components extracted via SSA from infant inactivity during sleep at 3 months. The analysis was based on a subsample of long sleep bouts > 5 h (median 4 bouts per infant, $n = 127$ infants with sufficiently long bouts without fusing). Histogram entries were weighted according to the amount of variance explained by their corresponding oscillatory component (often multiple per sleep bout); inset displays the unweighted distribution. (D) Distribution of the statistically significant dominant cycle lengths from Lomb-Scargle periodogram analysis across all infant sleep bouts at 3 months.

but retained their characteristic variability within and between individuals and time points (Table 2).

Ultradian rhythms in infant inactivity during sleep

For an analysis of ultradian rhythmicity in infant movement during sleep, the activity signal of all sleep bouts was transformed to LIDS, enhancing the contrast between movement and non-movement (Figure 1A). This inactivity measure, ranging from 0 to 100 inactivity, formed the basis of all following analyses.

Establishing rhythmicity. For a first "agnostic" test of ultradian rhythmicity in infant inactivity during sleep, we decomposed the inactivity signal in a subsample of suitably long bouts via SSA (Figure 1B). In more than 99% of all 127 infants with bouts >5 h, this revealed statistically significant oscillatory components with main cycle lengths of 10–150 min already at 3 months. The weighted distribution of main cycle lengths, reflecting variance explained, indicated a clear dominance of approximately 60 min cycle lengths in the inactivity signals (Figure 1C), which translates into a 78% probability of an infant in our subsample displaying at least one statistically significant oscillation between 50 and 70 min at 3 months. A second rhythm analysis across the full sample via Lomb-Scargle periodograms also detected statistically significant oscillations with a cycle length distribution peaking at approximately 60 min at 3 months (Figures 1D and S1). Together, these results provide strong evidence for the existence of an ultradian rhythm in inactivity during sleep in 3-month-old infants with a main cycle length of around 60 min.

Rhythm characteristics at 3 months. The presence of such an ultradian inactivity rhythm as early as 3 months is evident also by visual inspection of the inactivity signal. While most individual bouts show clear rhythms in inactivity (see example in Figure 1A), the averaged oscillation across all sleep bouts quickly dampens due to destructive interference from differing cycle lengths and phases (Figure S2, A and B). Period-normalization of the timeline and prior phase alignment, however, reveal a strong, sustained oscillation in ankle inactivity during infant sleep across the averaged signal (Figures 2, A and B and S2). Just like previously observed in adults, there is also a gradual decline in the levels of the inactivity oscillation over the course of sleep in infants.

To systematically characterize these ultradian inactivity rhythms in infants and compare them with those of their parents, parameter values were estimated via cosine model fits for LIDS cycle length, amplitude, offset, slope, and phase across the infant and parental samples. Below we consider these characteristics in detail at the 3-month time point, the first assessment time point in the sample, and thus earliest occurrence of the rhythm. As expected, the distribution of LIDS cycle lengths in infants from cosine fits also exhibited a peak around 60 min (Figure 2C), confirming SSA and Lomb-Scargle results (Figure 1, C and D). Notably, this 60-min cycle length matches previous polysomnographically determined cycle lengths of NREM–REM cycles in infants [29, 30], suggesting that infant inactivity oscillations might be similarly linked with sleep cycles as previously demonstrated in adults [20].

A further indication of such a link between inactivity and sleep cycles is the observation that the estimated LIDS cycle lengths in infants were shorter than those in adults, a well-established characteristic of polysomnographically determined NREM–REM cycles. Importantly, at approximately 60 min, LIDS cycle lengths were not only shorter than those previously reported in adults [20] but also shorter than those detected here in their parents (Figure 2, A–C), which were recorded using the same device and

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

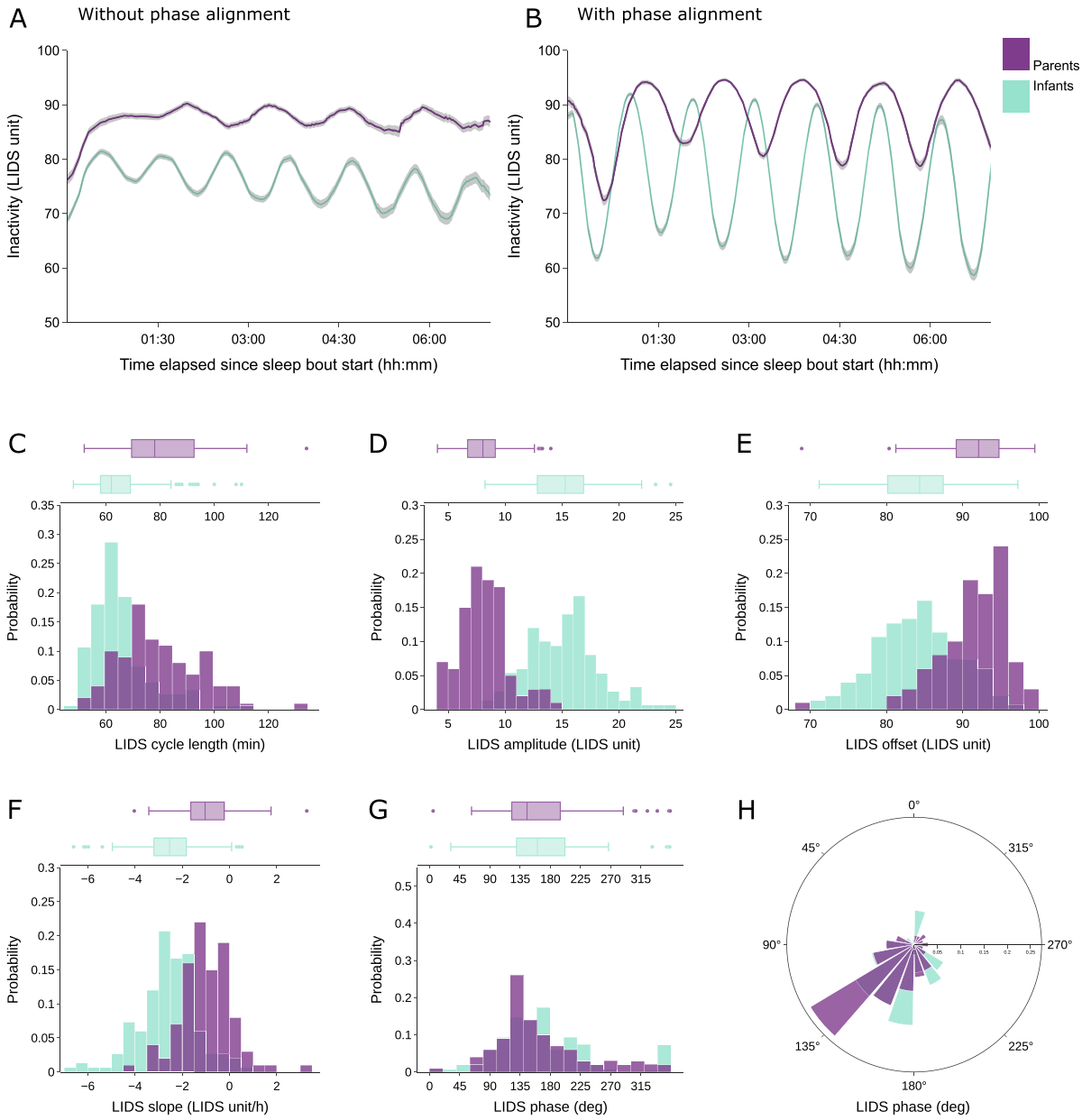


Figure 2. Comparison of inactivity rhythm parameters between infants and parents at 3 months. (A and B) Average inactivity profiles (±standard error) for infants and parents across all sleep bouts at 3 months. Signal timelines were normalized for inter-bout differences in cycle length before averaging and rescaled to infant and parent median LIDS cycle lengths for illustration, (A) without phase-alignment, (B) with phase-alignment. (C–H) Distributions of inactivity parameters for infants and parents at 3 months. Parameters were estimated via cosine fits to LIDS of individual sleep bouts and presented as medians per individual (C, cycle length; D, amplitude; E, offset; F, slope; G and H, phase). Boxplots are Tukey boxplots with whiskers spanning all data within 1.5 times the inter-quartile range above or below the central 50%.

the same sleep bout detection algorithm. Statistical testing for differences between infants and parents using Bayesian generalized mixed model regressions (Tables S4 and S5) predicted a difference in LIDS cycle lengths of approximately 19 min: 62 min (95% CI: [56, 67]) for infants and 81 min ([74, 88]) for parents at their median bout durations at the 3-months time point of 4.8 h and 5.8 h, respectively. It is worth noting that parental cycle lengths were predicted to reach the expected magnitude around 90-min for standard sleep bout lengths of 7.5 h (Table S5).

Further visual and statistical comparisons between infants and parents at the 3-month time point revealed differences not only in LIDS cycle length but also in all other LIDS parameters except for LIDS phase (Figure 2 and Table S4). At their respective median bout durations at 3 months, infants had a larger LIDS amplitude (18.2 [16.7, 19.7] vs. 7.8 [6.7, 9.0] LIDS units), a lower LIDS offset (84.8 [81.2, 88.1] vs. 91.2 [89.4, 94.2] LIDS units), and a steeper LIDS slope (−3.1 [−3.8, −2.4] vs. −1.0 [−1.7, −0.3] LIDS units/h) than their parents. This indicates a larger oscillatory range, lower inactivity at the beginning of sleep and a steeper decline of inactivity over the course of sleep. These results are in general agreement with those from the original LIDS analysis in children and adults [20], where LIDS cycle length was found to

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

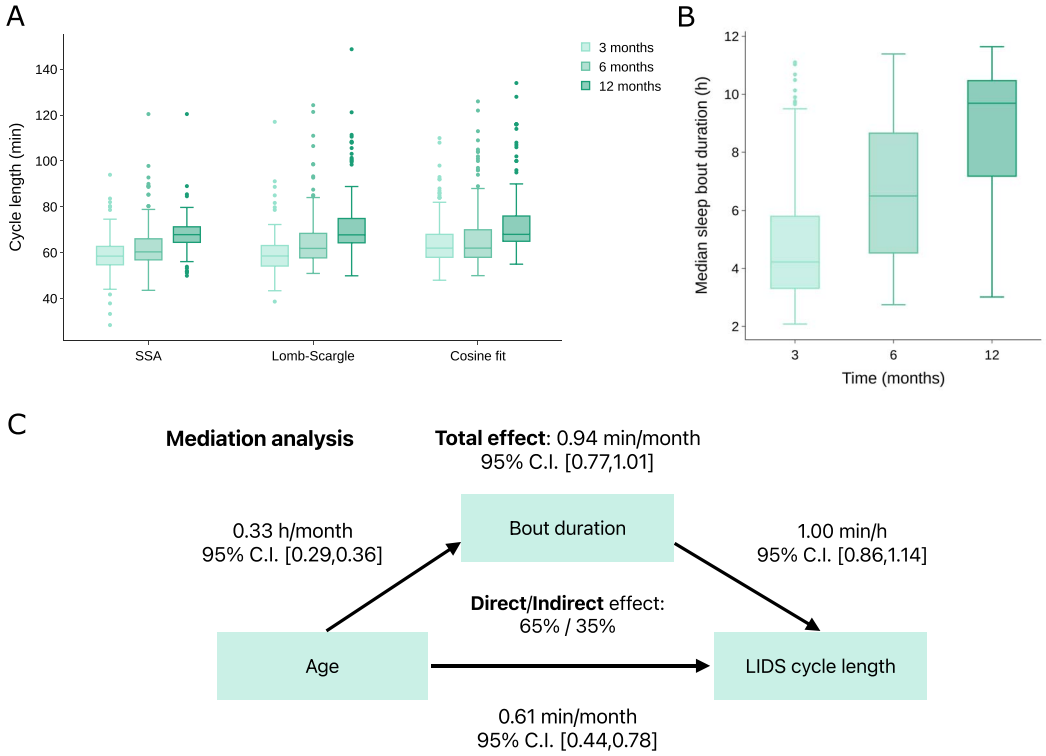


Figure 3. Infant inactivity rhythms lengthened over the first year of life. (A) Infant median LIDS cycle lengths at 3, 6, and 12 months obtained via three different methods: SSA, Lomb-Scargle periodogram and cosine fits. Note that samples differed between methods as SSA was run only on sleep bouts >5 h in duration, while the other two methods were applied to the full bout sample. (B) Infant median sleep bout durations at 3, 6, and 12 months. (C) Schematic diagram of the mediation model to test the hypothesis that sleep bout duration mediated the increase in LIDS cycle length over time. Numerical values represent the corresponding fixed-effect coefficients and their 95 ~ %CI. Boxplots are Tukey boxplots with whiskers spanning all data within 1.5 times the inter-quartile range above and below the central 50%. Note that (A) shows unadjusted data and averages per individual. For more informative numbers, refer to the statistically adjusted estimates reported in the text and tables.

lengthen with age, from 5 to 92 years, while amplitude and decline were reduced. The results for phase did not provide evidence that infant and parental LIDS rhythms started at different points of the oscillation in relation to the actigraphy-defined sleep bout onset. With the modified Sadeh algorithm employed here, the inactivity rhythm was, on average, just before the trough at detected sleep onset, which is slightly earlier than with the MASDA algorithm in Winnebeck *et al.* [20], where it was around the peak.

Taken together, these observations suggest that infant inactivity during sleep is rhythmic, shows a cycle length of around 60 min, and differs systematically from parental inactivity patterns during sleep in multiple parameters except phase.

Factors associated with infant rhythm characteristics

Development: cycle length. To test for a lengthening of ultradian sleep cycles during infancy, we analyzed the evolution of inactivity cycle length in infants across the three assessment time points: 3, 6, and 12 months. SSA analysis of long sleep bouts indicated that the probability for an infant to display statistically significant oscillatory components with main cycle lengths between 50 and 70 min remained high also at 6 and 12 months of age (75%–78%). However, as illustrated in Figure 3A, all three methods used for LIDS cycle length determination, SSA, Lomb-Scargle periodogram and cosine fits, indicate a gradual lengthening of inactivity cycles over the first year of life. The statistical analysis (Table S6) based on the cosine fit estimates puts the covariate-adjusted increase in LIDS cycle length with age at 4.7 min ([2.9, 6.5]) between 3 months and 12 months, while not detecting a statistically significant difference between 3 months and 6 months.

Importantly, inactivity cycle length was also found to increase with sleep bout duration (1.0 min/h [0.9, 1.2], Table S6), an effect previously also detected in adults [20]. Since infant sleep bout duration increased markedly with age, doubling from 4.8 h at 3 months to 9.9 h at 12 months (Figure 3B; Table 2), we conducted a dedicated mediation analysis to quantify how much of the effect of age on cycle length was actually mediated by the increase in bout duration. We found that 35% of the overall effect of age on LIDS cycle length was mediated by the increase in bout duration with age, whereas 65% occurred independently (Figure 3C), highlighting the possibility of (at least) two different mechanisms responsible for the increase in cycle length with age. Overall, the increase in LIDS cycle length between 3 and 12 months was predicted to be on average 9.8 min based on the average sleep bout lengthening of 5.1 h (4.7 min + 1.0 min/h × 5.1 h).

Finally, when selecting only bouts >5 h and restricting the cosine fit to the first 3 h of each bout, LIDS cycle length was still found to be longer at 12 months (3.1 min [1.5, 4.7]) compared with 3 months (Table S7), demonstrating that cycle lengthening with age was detectable also in a sample of homogeneous bout durations and identical cosine fit lengths.

Together, these results provide strong evidence for a gradual lengthening of ultradian sleep cycles during infancy and suggest potential concomitant lengthening mechanisms.

Development: other inactivity characteristics. All other LIDS characteristics except phase also changed throughout the

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

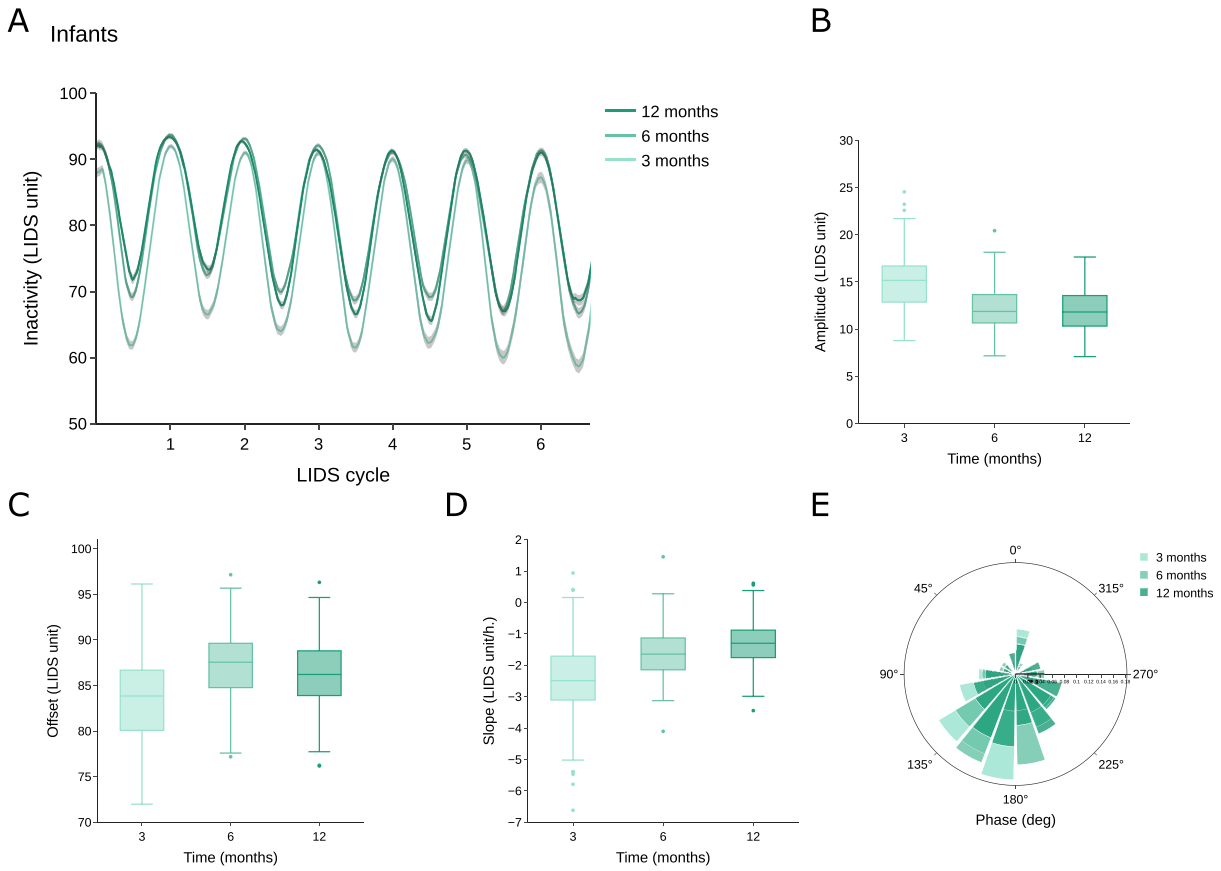


Figure 4. Development of other infant LIDS parameters over the first year of life. (A) Average inactivity profiles (±standard error) for infants at 3, 6, and 12 months. Signals were period-normalized and phase-aligned before averaging. (B–E) LIDS parameter estimates for infants at 3, 6, and 12 months. Parameters were estimated via cosine fits to LIDS of individual sleep bouts and are presented as medians per individual (B, amplitude; C, offset; D, slope; E, phase). Boxplots are Tukey boxplots with whiskers spanning all data within 1.5 times the inter-quartile range above or below the central 50%. Note that the figures show unadjusted data and averages per individual. For more informative numbers, refer to the statistically adjusted estimates reported in the text and tables.

first year of life (Figure 4; Table S6). Compared with 3-months, infants at 6 and 12 months had a lower LIDS amplitude (−2.3 LIDS units [−2.7, −1.8] at 6 months; −1.6 LIDS units [−2.2, −0.9] at 12 months), a higher LIDS offset (3.3 [2.4, 4.1]; 1.5 [0.3, 2.7] LIDS units) and an increased LIDS slope (0.7 [0.5, 0.8]; 0.8 [0.6, 1.1] LIDS units/h). The increase over time of LIDS offset shows that LIDS oscillations were initiated with a higher level of inactivity as the infants grew older, while the increase in the generally negative LIDS slope indicates a shallower decline in inactivity over the course of sleep at later time points. Together, this means that the overall level of inactivity remained higher throughout the sleep bout for older infants. As noted before [20], the probability of awakening most likely increases as the level of inactivity decreases. These results are thus compatible with infants having more consolidated sleep bouts and, therefore, longer sleep bouts during the night at 6 and 12 months compared with 3 months (Table 2).

No systematic differences in LIDS phase between time points were observed. However, it can be seen in the inactivity profiles (Figure S2) that rescaling bout timelines to their individual LIDS cycle length was sufficient to reveal clear oscillations at 3 months, while at 6 and 12 months this required additional phase-alignment. This points towards an increased variability in LIDS phase at the later time points.

Endogenous factors: sleep–wake cycle maturation and sex. In none of the statistical models did the proportion of nighttime sleep, included as a coarse marker of circadian and homeostatic maturation of sleep, show any association with any inactivity parameters. This is likely due to the low variability in this marker between infants within a time point and hence its colinearity with bout duration and age. Sex also explained little of the variation in infant inactivity parameters, with none of the effects reaching statistical significance. However, two patterns were notable: firstly, male infants tended towards longer sleep cycles (1.3 min [−0.08, +2.83]) across the full sample (Table S6). Secondly, LIDS offset tended towards lower levels in male infants (−0.8 LIDS units [−1.77, +0.21]; Table S6) suggesting greater movement during sleep in males. This matches several previous reports on sex differences in infant sleep motility, despite a lack of consistency across studies [51], as well as LIDS results in adults [20] and our result here in fathers (see below).

Exogenous factors: breastfeeding and sleep location. At 3 and 6 months, all or nearly all infants in this study were regularly breastfed (100% and 93%, respectively) while this was the case for just 36% of the cohort at 12 months (Table 1). Therefore, we explored differences in LIDS characteristics linked to differences in breastfeeding status at the last recording time point via a

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

dedicated statistical model including breastfeeding status (Table S8). At 12 months, infants that were still breastfed at least three to five times per week had a longer LIDS cycle length (2.5 min [0.4, 4.5]) as well as a higher LIDS offset (1.4 LIDS units [0.2, 2.6]), meaning that breastfed infants had longer inactivity cycles and initiated their sleep at a higher inactivity level compared with non-breastfed infants of the same age. Other LIDS parameters were not statistically linked to breastfeeding status.

In addition, infants differed in their nighttime sleep location, both within and between time points, from predominantly the parents' rooms and parents' beds at 3 months (87%) to mostly separate rooms at 12 months (58%, Table 1). Exploring relationships between infants' nighttime sleep location and LIDS characteristics across all time points (Table S6), we found that, compared with infants sleeping in a separate room, infants sleeping in their parents' bed had longer LIDS cycle lengths (2.9 min [1.2, 4.6]), lower LIDS amplitudes (−0.9 LIDS units [−1.5, −0.3]), and a flatter LIDS decline (0.3 LIDS units/h [0.1, 0.6]). These results were qualitatively identical to those obtained with the model including breastfeeding using sleep bouts collected at 12 months only (Table S8). Interestingly, the direction of the effects (increase or decrease) of sleeping predominantly in the parental bed was similar to the effect of age.

Factors associated with parental rhythm characteristics

Parental role. In parents, there was no direct effect of time points on LIDS cycle length. However, fathers exhibited a shorter cycle length (−4.7 min [−9.1, −0.3]) as well as a lower LIDS amplitude (−0.5 LIDS units [−0.9, −0.1]). However, when considering the shorter sleep durations of mothers versus fathers in this sample (Table S3) and how cycle length and amplitude scale with sleep duration, the exhibited cycles would be predicted to be longer in fathers than in mothers. Furthermore, at 3 months, fathers initiated their sleep at lower inactivity levels than mothers as reflected by their lower LIDS offset (−1.2 LIDS units [−2.1, −0.2]), with the difference to mothers even further increased at 6 months by an additional −1.5 LIDS units ([−2.9, −0.2]) until both showed reduced LIDS offsets at 12 months (−1.1 LIDS units [−2.0, −0.2]). Since the level of activity/inactivity in adults has been correlated with sleep depth [20, 52, 53], these results might be interpreted as deeper sleep/higher sleep pressure when the infant is younger due to greater partial sleep deprivation common in parents with young infants [54], with mothers initially more affected—potentially due to breastfeeding and uneven child care split.

Other LIDS parameters were not statistically different between mothers and fathers.

Breastfeeding (mothers only). As breastfeeding might induce physiological changes that can, in turn, alter sleep in women, potential links between LIDS characteristics and maintained breastfeeding at the 12-months time point were also statistically assessed (Table S10). Indeed, at 12 months, compared with non-breastfeeding mothers, breastfeeding mothers had a longer LIDS cycle length (6.7 min [0.5, 12.9]) as well as a steeper LIDS slope (−0.5 LIDS unit/h [−0.9, −0.1]), indicating a faster decline of the inactivity levels. Other LIDS parameters were not statistically linked to breastfeeding habits.

It is interesting to note that both breastfed infants and breastfeeding mothers had a longer LIDS cycle length compared with their non-breastfeeding/fed counterparts.

Sleep location. The same statistical model for mothers at 12-months indicates associations between the sleep location of the infant and the mother's LIDS characteristics (Table S10). Compared with mothers with an infant sleeping in a separate room, mothers sharing their room but not necessarily their bed with their infant had a longer LIDS cycle length (8.6 min [0.0, 17.0]), and mothers sharing a bed with their infant had a greater LIDS amplitude (1.8 LIDS units [0.5, 3.0]) and a lower LIDS offset (−2.8 LIDS units [−4.9, −0.6]). Other LIDS parameters were not statistically linked to the mother–infant sleep settings.

Similar associations were found in the full parental model incorporating all time points and also the fathers (Table S9), although the smaller sample size of fathers still biases this analysis towards the mothers. When families reported that their infant was sleeping predominantly in the parental bed, parents' LIDS amplitude was again greater (1.4 LIDS units [0.7, 2.1]) and LIDS offset was lower (−1.6 LIDS units [−2.8, −0.5]) than when infants slept in a separate room. In contrast, in this model, the longest ultradian cycle lengths for parents were found when the infant slept in an attached cot (6.3 min [1.8, 10.9]), not in a separate bed as at 12 months.

Discussion

Our study analyzed limb movement patterns during sleep as a window into infant and parental sleep physiology during infants' first year of life. We extracted both rhythmic and non-rhythmic patterns of LIDS from actigraphy and investigated associations with key endogenous and exogenous factors such as development, sex, sleep bout duration, breastfeeding, and sleep location to explore the potential of this approach for studying the functional relevance of sleep cycle dynamics in health and disease. Most notably, employing a range of analytic techniques, we demonstrated the existence of ultradian rhythms in infant inactivity that are compatible with inactivity patterns reflecting ultradian sleep cycles. These cycles lengthened between the 3- and 12-month time points in concert with age and also with consolidation of sleep bouts, providing strong longitudinal evidence that sleep cycles lengthen gradually during development. Below we discuss the interpretation of the different inactivity pattern characteristics extracted from the LIDS signal.

A rich signal such as limb movement during sleep inherently possesses many quantifiable features. Characterizing the LIDS signal using a cosine fit with a linear slope, we extracted five parameters of the inactivity signal during sleep. LIDS cycle length, amplitude, and phase are inherently quantifications of the rhythmic aspects of the signal and might be interpreted as reflecting the periodicity, amplitude, and phase of an underlying ultradian process. Phase, which quantifies the state of the inactivity rhythm at the beginning of the sleep bout, needs to be carefully interpreted as it is greatly dependent on the sleep–wake detection algorithm in cases where this is also based on movement like in our sample. In contrast, LIDS offset and LIDS slope quantify non-rhythmic aspects of inactivity during sleep, indicating the density and intensity of movement at the beginning of the bout (offset) and the gradual decline in inactivity with time asleep (slope). The latter reflects the previously observed gradual increase in movement during infant sleep [55]. Notably, the parameter amplitude also carries information about the amount of movement and is influenced by the intensity of movement also through the non-linearity in the LIDS transform, therefore, reflecting both rhythmic and non-rhythmic aspects of inactivity.

Turning first to the rhythmic patterns in inactivity during sleep, the central observation of our study was that motor inactivity during sleep is rhythmic in infants at 3 months, with a cycle

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

length of approximately 60 min. This was established using three different analysis methods, which were not only in agreement with each other but also with the expected length of the NREM–REM sleep cycle for infants of this age [28–30]. Moreover, these findings go full circle with the original discovery of sleep cycles, where motility cycles were one of the main features characterized by Denisova [14] and by Aserinsky and Kleitman [13, 56]. Additionally, infants showed shorter cycle lengths in inactivity than their parents, aligning with previous findings of different NREM–REM cycle lengths in infants and adults. This leads us to consider that both NREM–REM cycles and LIDS cycles are the manifestation of the same underlying ultradian process, rather than the co-occurrence of separated processes. Although formal testing of this hypothesis is required, there is good evidence of shared control circuits between motor activity and brain sleep states both for REM and NREM sleep in vertebrates [57–59]. In addition, when it comes to the rhythmicity aspects, rhythmic motor control and ultradian NREM–REM rhythms share common features such as phase-resetting and entrainment by external stimuli [60, 61]. Future investigations will hopefully substantiate the neurophysiological links between movement, sleep states, and ultradian regulation.

Furthermore, we demonstrated that inactivity cycles lengthened between 3 and 12 months of age by an average of 9.8 min. This closely paralleled the previously reported lengthening of NREM–REM sleep cycles during infancy, for which, over the exact same developmental period, mean NREM–REM cycle lengthening from single night recordings has been put at 11 min in a longitudinal study of 15 infants [30] and at around 15 min in a cross-sectional comparison in 48 infants of diverse ages [31]. Notably, the lengthening in our sample between 3 and 12 months was evident across all three analysis techniques, but SSA and Lomb-Scargle periodogram detected increases in cycle length already at 6 months of age (contrary to the cosine fits underlying the statistical analyses), corroborating previous suggestions (and difficulties of detection) of the gradual nature of lengthening over the first year of life [30, 31]. Our study, therefore, adds substantial longitudinal evidence for gradual sleep cycle lengthening in a large sample of 150 infants over multiple consecutive nights per time point.

Intuitively, this central finding of developmental lengthening of ultradian sleep cycles may be attributed to just one cause, the maturation of the ultradian process underlying both NREM–REM and LIDS rhythmicity. However, our mediation analysis revealed at least two mechanisms at play, pinpointing one to act via increased sleep bout duration. Since consolidation of sleep is often considered a consequence of maturing circadian and homeostatic sleep regulation [49, 50], this bout-length-driven portion of cycle lengthening suggests a potential interplay of circadian and ultradian development. The remaining effects on sleep cycle length may partially or fully reflect broader maturational processes in infant sleep regulation beyond bout consolidation. Infancy is characterized by rapid maturation of neuronal networks, including dynamics in cortical synaptic density, progressive myelination, and strengthening of functional connectivity, which collectively shape emerging sleep architecture. These processes also reflect increasing individual specialization in brain network organization, which cannot be captured with wearables or behavioral sleep quantification alone but may benefit from longitudinal high-spatial-resolution sleep EEG approaches to become visible [62].

When it comes to interpreting the exogenous effects on sleep cycles detected here, the literature is considerably sparser and our interpretations accordingly more speculative in nature. We observed that breastfed infants at 12 months had longer LIDS cycles in our sample, reflecting potentially a more mature ultradian rhythm than in non-breastfed infants akin to maturation with age. However, also improved circadian maturation may play a role. Human breast milk exhibits daily variations in hormone concentrations (higher cortisol during daytime, higher melatonin at night [63]), making breastfeeding a hormonal *zeitgeber*. Hence, breastfeeding may facilitate circadian entrainment in infants and contribute to the maturation of the circadian system, as evidenced by an earlier appearance of a circadian rhythm in core body temperature in breastfed compared with non-breastfed infants [64]. This observation aligns with our previously stated hypothesis that circadian and ultradian rhythms could be interrelated. Another possible explanation is that breastfeeding may also promote microbial diversity [65] and thus interact with the sleep–gut axis [35]. Indeed, within the same infant sample, a more diverse gut microbiome was linked to reduced daytime sleep. This could again be interpreted as the result of a more mature circadian system promoting daytime wakefulness and/or a more mature homeostatic system leading to slower accumulation of homeostatic sleep pressure during wakefulness. With many interpretations plausible, also the reverse should not be dismissed, where longer, more mature sleep cycles may make e.g. breastfeeding more sustainable for parents (in line with longer cycles also associated with room sharing). These observation may stimulate future studies, as interventions could be designed around modifiable factors that alter sleep and/or the gut microbiome during early infancy. Interestingly, breastfeeding mothers also showed longer LIDS cycles, and further investigation may shed light on any potential hormonal or microbial influences.

Turning to the non-rhythmic features of the inactivity signal, the question of their interpretation becomes paramount to interpreting their developmental changes and associations with endogenous and exogenous factors. Based on prior research into body movements and sleep, including our own work on LIDS [20, 52, 66], we believe the most parsimonious interpretation is that the non-rhythmic components extracted from LIDS reflect sleep depth and sleep pressure. Deep sleep is more prominent in the first half of the night and is linked with lower motor activity, thus, higher inactivity levels towards the beginning of a sleep bout likely correspond to deeper sleep early on in the bout [52, 53]. Another key feature of sleep is sleep homeostasis: the accumulation of sleep pressure during wakefulness and its gradual dissipation during subsequent sleep, in parallel with the decrease of deep sleep/slow wave activity over the course of the sleep bout [24, 67]. Our analysis in infants here shows that, similar to adults [20], mean inactivity levels decreased overnight in line with the gradual change from deeper sleep to lighter sleep. Thus, the non-rhythmic components extracted from the LIDS signal may reflect both sleep depth and the dissipation of sleep pressure, an interpretation we use below for the findings in this study. However, it is without question that formal testing of this interpretation, using studies of experimental sleep restriction, will be essential to validate it.

For the non-rhythmic inactivity parameters, we observed multiple noteworthy associations with endogenous factors. Firstly, LIDS offset (inactivity level at sleep onset) was lower in infants than in parents. This can be explained by the generally higher activity level of infants during sleep compared with adults, which is well documented [51]. We also observed an increase in LIDS offset with age. If interpreted in the context of sleep homeostasis, this could be indicative of higher sleep pressure in older infants, in line with our previous observation about decreased daytime sleep and consolidated, longer sleep bouts during nighttime.

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Additionally, LIDS slope (inactivity decline over the course of the sleep bout) showed a steeper decline in infants than in parents, possibly indicating a faster dissipation of homeostatic sleep pressure in infants, which is in line with mathematical models of infant sleep [68] and also with higher occurrence of slow-wave sleep in infants, a hallmark of sleep pressure dissipation, during the night [69]. However, infants maintained higher inactivity levels throughout the night as they grew older. It remains unclear if this is a cause or a consequence of sustained sleep episodes in older infants. Finally, LIDS amplitude was higher in infants than in parents, but decreased as infants aged. This phenomenon likely also reflects generally higher activity levels in infants. It remains to be investigated whether the pronounced LIDS amplitude in early infancy reflects the distinct neurophysiological features of infant sleep, as may be revealed by EEG markers of active and quiet sleep. The great amplitude in inactivity oscillations during sleep in early infancy, however, particularly recommends the use of LIDS in this age group for sleep cycle studies, possibly even over EEG.

Finally, we also observed exogenous effects on the non-rhythmic aspect of inactivity in what is a highly exploratory analysis of this topic, given the small prior number of studies on the subject. Breastfeeding mothers showed a steeper decline in inactivity over the course of sleep compared with non-breastfeeding mothers. This is interesting in light of the increased sleep pressure signature previously demonstrated for breastfeeding mothers, based on higher delta power in their nocturnal sleep electroencephalogram (EEG) and more time spent in deep sleep [70, 71]. These increases have been shown to remain even when controlling for partial sleep deprivation, suggesting that the differences in nighttime sleep might be, at least partially, due to physiological adaptations related to increased circulating prolactin levels induced by breastfeeding. When it comes to sleep environmental influences, various sleep characteristics, such as sleep duration or quality, have been shown to be modulated by socioeconomic and environmental factors [72, 73]. Parent–infant sleep settings (solitary sleeping infants or infants sharing a room and/or bed with their parents) seem to affect both infants and mothers, as well as mothers' postpartum sleep trajectory [74, 75]. Interestingly, our exploratory analysis revealed that LIDS characteristics were linked to bed/room sharing habits, both in infants and parents, suggesting that dedicated studies on the impact of the environment on sleep might benefit from the LIDS methodology.

Limitations

While this study demonstrates the great potential of using large longitudinal activity records as a window into infant and parent sleep, we note several limitations for the interpretation of this study. It cannot be excluded that differences in inactivity patterns between infants and parents partially originated from the different placement of devices between the two groups. Actigraphy data were collected using ankle-worn devices in infants while parents wore the actigraph at their wrists. However, it was demonstrated that, at least in adults, ankle-derived and wrist-derived LIDS patterns are highly correlated within individuals and sleep bouts [20]. Additionally, both infants and parents were recorded using the exact same device model, and the same sleep–wake algorithm was used on all recordings, minimizing methodological differences from these aspects. Another limiting factor arose from the natural settings in which actigraphy data were collected; while measuring sleep in the habitual sleep environment underscores the study's relevance, it also hampers exclusion of confounding factors, such as environmental noise. This is particularly relevant for our exploratory analyses of breastfeeding and sleep location, which need to be interpreted carefully (including considering reverse causation) and used only for hypothesis generation to be followed up in more controlled studies. Finally, the social, economic, and cultural characteristics of the participants should also be taken into account when generalizing the study findings, as they have been shown to exert an influence on sleep characteristics [72]. Our family sample had a high educational background, even exceeding Zurich's high education level (the city with the greatest proportion of tertiary degrees in Switzerland at 45.5% of all residents over the age of 15 [76]). Reproducing our results with socioeconomically, culturally, and ancestrally diverse cohorts is therefore paramount.

Conclusions

Epidemiological, longitudinal, and experimental research has demonstrated the importance of sleep for infant brain development and cognitive functioning. Methods that allow large-scale characterization of sleep beyond simple sleep duration commonly obtained from self-reports or actigraphy will be essential in enabling better understanding of the interplay between sleep physiology and development. Even though movement records will unlikely ever convey as much detail as polysomnography, the simplicity and unobtrusiveness of data recording enables large, longitudinal samples that are instrumental for hypothesis generation, in discovering potential genetic mechanisms through genome-wide association studies or identifying potential biomarkers for developmental progress that are suitable as screening tools. Using limb movement from actigraphy or other wearables as a window into sleep physiology, and particularly the thus far still enigmatic ultradian sleep cycles, promises great potential in advancing our understanding of the intricate relationship between sleep and developmental trajectories.

Supplementary material

Supplementary material is available at *SLEEP* online.

Acknowledgments

We would like to thank the families for their participation in this study.

Author contributions

Inactivity pattern analysis: Grégory Hammad (Formal analysis, Methodology, Software, Validation, Visualization, Writing—original draft preparation, Writing—review & editing), Sarah F. Schoch (Data curation, Formal analysis, Writing—review & editing), Maximilian Engelmann (Formal analysis, Validation), Zoe Spock (Validation, Writing—review & editing), Salome Kurth (Writing—review & editing), and Eva C. Winnebeck (Conceptualization, Funding acquisition, Methodology, Project administration, Supervision, Validation, Writing—original draft preparation, Writing—review & editing)

Infant cohort: Sarah F. Schoch (Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration) and Salome Kurth (Conceptualization, Funding acquisition, Investigation, Project administration)

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

Funding

This analysis project was funded by the Deutsche Forschungsgemeinschaft (DFG, German Research Foundation: 450622422 to E.C.W.). Data collection, curation and sleep–wake classification was funded by the Swiss National Science Foundation (PCEFP1-181279 to S.K.; P0ZHP1-178697 and P5R5PS-230534 to S.F.S.), University of Zurich (Medical Faculty; Forschungskredit FK-18-047, Clinical Research Priority Program "Sleep and Health," to S.K.), Foundation for Research in Science and the Humanities (STWF-17-008, to S.K.), and Olga Mayenfisch Foundation (to S.K.)

Disclosure statement

Financial disclosure: None declared.

Non-financial disclosure: None declared.

Data availability

Data and scripts were deposited on Zenodo: https://doi.org/10.5281/zenodo.18199381.

References

1. Jaggard JB, Wang GX, Mourrain P. Non-REM and REM/paradoxical sleep dynamics across phylogeny. *Curr Opin Neurobiol*. 2021;**71**:44–51. https://doi.org/10.1016/j.conb.2021.08.004
2. Honma KI, Hiroshige T. Endogenous ultradian rhythms in rats exposed to prolonged continuous light. *Am J Physiol Regul Integr Comp Physiol*. 1978;**235**(5):R250–R256. https://doi.org/10.1152/ajpregu.1978.235.5.R250
3. Prendergast BJ, Zucker I. Ultradian rhythms in mammalian physiology and behavior. *Curr Opin Neurobiol*. 2016;**40**:150–154. https://doi.org/10.1016/j.conb.2016.07.011
4. van der Veen DR, Gerkema MP. Unmasking ultradian rhythms in gene expression. *FASEB J*. 2017;**31**(2):743–750. https://doi.org/10.1096/fj.201600872R
5. Zhu B, Liu S. Preservation of ~ 12-h ultradian rhythms of gene expression of mRNA and protein metabolism in the absence of canonical circadian clock. *Front Physiol*. 2023;**14**:1195001. https://doi.org/10.3389/fphys.2023.1195001
6. Focke CMB, Iremonger KJ. Rhythmicity matters: circadian and ultradian patterns of HPA axis activity. *Mol Cell Endocrinol*. 2020;**501**:110652. https://doi.org/10.1016/j.mce.2019.110652
7. Blessing W, Ootsuka Y. Timing of activities of daily life is jaggy: how episodic ultradian changes in body and brain temperature are integrated into this process. *Temperature*. 2016;**3**(3):371–383. https://doi.org/10.1080/23328940.2016.1177159
8. Gerkema MP, Daan S, Wilbrink M, Hop MW, van der Leest F. Phase control of ultradian feeding rhythms in the common vole (*Microtus arvalis*): the roles of light and the circadian system. *J Biol Rhythms*. 1993;**8**(2):151–171. https://doi.org/10.1177/074873049300800205
9. Daan S, Aschoff J. Short-Term Rhythms in Activity. In: Aschoff J, ed. *Biological Rhythms*. Boston, MA: Springer US; 1981:491–498, https://doi.org/10.1007/978-1-4615-6552-9_25
10. Grau C, Escera C, Cilveti R, *et al*. Ultradian rhythms in gross motor activity of adult humans. *Physiol Behav*. 1995;**57**(3):411–419. https://doi.org/10.1016/0031-9384(94)00274-9
11. Goh GH, Maloney SK, Mark PJ, Blache D. Episodic ultradian events—ultradian rhythms. *Biology*. 2019;**8**(1):15. https://doi.org/10.3390/biology8010015
12. van der Veen DR, Gerkema MP. Re-scoping ultradian rhythms in the context of metabolism. *Front Physiol*. 2024;**15**:1504879. https://doi.org/10.3389/fphys.2024.1504879
13. Aserinsky E, Kleitman N. A motility cycle in sleeping infants as manifested by ocular and gross bodily activity. *J Appl Physiol*. 1955;**8**(1):11–18. https://doi.org/10.1152/jappl.1955.8.1.11
14. Denisova M, Figurin N. Periodic phenomena in the sleep of children. *Novoe v refleksologii i fiziologii nervnoi sistemy (Beiträge zur reflexologie und physiologie des nervensystems; News in reflexology and physiology of the nervous system)*. 1926;**2**:338–345.
15. Denisova K. English translation of the first study reporting cyclical periods of increased respiration and eye and body motility during sleep in infants in 1926, with commentary. *Sleep*. 2024;**47**(1):zsad219. https://doi.org/10.1093/sleep/zsad219
16. Dement W, Kleitman N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalogr Clin Neurophysiol*. 1957;**9**(4):673–690. https://doi.org/10.1016/0013-4694(57)90088-3
17. Feinberg I, Floyd TC. Systematic trends across the night in human sleep cycles. *Psychophysiology*. 1979;**16**(3):283–291. https://doi.org/10.1111/j.1469-8986.1979.tb02991.x
18. Cajochen C, Reichert CF, Münch M, *et al*. Ultradian sleep cycles: frequency, duration, and associations with individual and environmental factors-a retrospective study. *Sleep Health*. 2023;**10**:S52–S62. https://doi.org/10.1016/j.sleh.2023.09.002
19. Osorio-Forero A, Foustoukos G, Cardis R, *et al*. Infraslow noradrenergic locus coeruleus activity fluctuations are gatekeepers of the nrem–rem sleep cycle. *Nat Neurosci*. 2025;**28**(1):84–96. https://doi.org/10.1038/s41593-024-01822-0
20. Winnebeck EC, Fischer D, Leise T, Roenneberg T. Dynamics and ultradian structure of human sleep in real life. *Curr Biol*. 2018;**28**(1):49–59.e5. https://doi.org/10.1016/j.cub.2017.11.063
21. Coons S, Guilleminault C. Development of consolidated sleep and wakeful periods in relation to the day/night cycle in infancy. *Dev Med Child Neurol*. 1984;**26**(2):169–176. https://doi.org/10.1111/j.1469-8749.1984.tb04428.x
22. Armstrong KL, Quinn RA, Dadds MR. The sleep patterns of normal children. *Med J Aust*. 1994;**161**(3):202–205. https://doi.org/10.5694/j.1326-5377.1994.tb127383.x
23. Henderson JMT, France KG, Blampied NM. The consolidation of infants' nocturnal sleep across the first year of life. *Sleep Med Rev*. 2011;**15**(4):211–220. https://doi.org/10.1016/j.smrv.2010.08.003
24. Borbély AA. A two process model of sleep regulation. *Hum Neurobiol*. 1982;**1**(3):195–204.
25. Borbély AA, Daan S, Wirz-Justice A, Deboer T. The two-process model of sleep regulation: a reappraisal. *J Sleep Res*. 2016;**25**(2):131–143. https://doi.org/10.1111/jsr.12371
26. Thoman EB, McDowell K. Sleep cyclicity in infants during the earliest postnatal weeks. *Physiol Behav*. 1989;**45**(3):517–522. https://doi.org/10.1016/0031-9384(89)90067-X
27. Freudigman K, Thoman EB. Ultradian and diurnal cyclicity in the sleep states of newborn infants during the first two postnatal days. *Early Hum Dev*. 1994;**38**(2):67–80. https://doi.org/10.1016/0378-3782(94)90218-6
28. Jenni OG, Borbély AA, Achermann P. Development of the nocturnal sleep electroencephalogram in human infants. *Am J Physiol Regul Integr Comp Physiol*. 2004;**286**(3):R528–R538. https://doi.org/10.1152/ajpregu.00503.2003
29. Stern E, Parmelee AH, Harris MA. Sleep state periodicity in prematures and young infants. *Dev Psychobiol*. 1973;**6**(4):357–365. https://doi.org/10.1002/dev.420060409
30. Louis J, Cannard C, Bastuji H, Challamel MJ. Sleep ontogenesis revisited: a longitudinal 24-hour home polygraphic study on 15

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

normal infants during the first two years of life. *Sleep*. 1997;**20**(5):323–333. https://doi.org/10.1093/sleep/20.5.323
31. Ficca G, Fagioli I, Salzarulo P. Sleep organization in the first year of life: developmental trends in the quiet sleep–paradoxical sleep cycle. *J Sleep Res*. 2000;**9**(1):1–4. https://doi.org/10.1046/j.1365-2869.2000.00172.x
32. Lopp S, Navidi W, Achermann P, LeBourgeois M, Behn CD. Developmental changes in ultradian sleep cycles across early childhood: preliminary insights. *J Biol Rhythms*. 2017;**32**(1):64–74. https://doi.org/10.1177/0748730416685451
33. Schoch SF, Jenni OG, Kohler M, Kurth S. Actimetry in infant sleep research: an approach to facilitate comparability. *Sleep*. 2019;**42**(7):zsz083. https://doi.org/10.1093/sleep/zsz083
34. Schoch SF, Huber R, Kohler M, Kurth S. Which are the central aspects of infant sleep? The dynamics of sleep composites across infancy. *Sensors*. 2020;**20**(24):7188. https://doi.org/10.3390/s20247188
35. Schoch SF, Castro-Mejía JL, Krych L, *et al*. From alpha diversity to zzz: interactions among sleep, the brain, and gut microbiota in the first year of life. *Prog Neurobiol*. 2022;**209**:102208. https://doi.org/10.1016/j.pneurobio.2021.102208
36. Hammad G, Reyt M, Beliy N, *et al*. pyActigraphy: open-source python package for actigraphy data visualization and analysis. *PLoS Comput Biol*. 2021;**17**(10):e1009514. https://doi.org/10.1371/journal.pcbi.1009514
37. te Lindert BHW, Van Someren EJW. Sleep estimates using microelectromechanical systems (MEMS). *Sleep*. 2013;**36**(5):781–789. https://doi.org/10.5665/sleep.2648
38. Sadeh A, Sharkey M, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. *Sleep*. 1994;**17**(3):201–207. https://doi.org/10.1093/sleep/17.3.201
39. Allen MR, Smith LA. Monte Carlo SSA: detecting irregular oscillations in the presence of colored noise. *J Climate*. 1996;**9**(12):3373–3404. https://doi.org/10.1175/1520-0442(1996)009<3373:MCSDIO>2.0.CO;2
40. Garnot VSF, Daon Y, Schmidt V. *Monte Carlo Singular Spectrum Analysis*.
41. Golyandina N. Particularities and commonalities of singular spectrum analysis as a method of time series analysis and signal processing. *WIREs Comput Stat*. 2020;**12**(4):1–39. https://doi.org/10.1002/wics.1487
42. Khan MAR, Poskitt DS. A note on window length selection in singular Spectrum analysis. *Aust N Z J Stat*. 2013;**55**(2):87–108. https://doi.org/10.1111/anzs.12027
43. Lomb NR. Least-squares frequency analysis of unequally spaced data. *Astrophys Space Sci*. 1976;**39**(2):447–462. https://doi.org/10.1007/BF00648343
44. Scargle JD. Studies in astronomical time series analysis. II. Statistical aspects of spectral analysis of unevenly spaced data. *Astrophys J*. 1982;**263**:835–853. https://doi.org/10.1086/160554
45. Gang W, Anafi RC, Hughes ME, Kornacker K, Hogenesch JB. Metacycle: an integrated R package to evaluate periodicity in large scale data. *Bioinformatics*. 2016;**32**(21):3351–3353. https://doi.org/10.1093/bioinformatics/btw405
46. Newville M, Otten R, Nelson A, *et al*. LMFIT: Non-Linear Least-Squares Minimization and Curve-Fitting for Python. Version 1.0.3. Zenodo; 2021. https://doi.org/10.5281/zenodo.5570790
47. Yuan Y, MacKinnon D. Bayesian mediation analysis. *Psychol Methods*. 2009;**14**(4):301–322. https://doi.org/10.1037/a0016972
48. Capretto T, Piho C, Kumar R, Westfall J, Yarkoni T, Martin OA. Bambi: a simple interface for fitting bayesian linear models in python. *J Stat Softw*. 2022;**103**(15):1–29. https://doi.org/10.18637/jss.v103.i15
49. Xinting Y, Quante M, Rueschman M, *et al*. Emergence of racial/ethnic and socioeconomic differences in objectively measured sleep–wake patterns in early infancy: results of the rise & SHINE study. *Sleep*. 2020;**44**(3):zsaa193. https://doi.org/10.1093/sleep/zsaa193
50. Paavonen EJ, Saarenpää-Heikkilä O, Morales-Munoz I, *et al*. Normal sleep development in infants: findings from two large birth cohorts. *Sleep Med*. 2020;**69**:145–154. https://doi.org/10.1016/j.sleep.2020.01.009
51. Mimi L, Fitzgerald DA, Norman MB, Sullivan CE, Waters KA. Pediatric sleep movements: a review of methodologies, normative data, disease associations and research gaps. *J Clin Sleep Med*. 2025;**21**(10):1773–1785. https://doi.org/10.5664/jcsm.11748
52. Naitoh P, Muzet A, Johnson LC, Moses J. Body movements during sleep after sleep loss. *Psychophysiology*. 1973;**10**(4):363–368. https://doi.org/10.1111/j.1469-8986.1973.tb00793.x
53. Wilde-Frenz J, Schulz H. Rate and distribution of body movements during sleep in humans. *Percept Mot Skills*. 1983;**56**(1):275–283. https://doi.org/10.2466/pms.1983.56.1.275
54. Hagen EW, Mirer AG, Palta M, Peppard PE. The sleep-time cost of parenting: sleep duration and sleepiness among employed parents in the Wisconsin Sleep Cohort Study. *Am J Epidemiol*. 2013;**177**(5):394–401. https://doi.org/10.1093/aje/kws246
55. Tonetti L, Scher A, Atun-Einy O, Samuel M, Boreggiani M, Natale V. Actigraphic motor activity during sleep from infancy to adulthood. *Chronobiol Int*. 2017;**34**(2):246–253. https://doi.org/10.1080/07420528.2016.1219362
56. Aserinsky E, Kleitman N. Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science*. 1953;**118**(3062):273–274. https://doi.org/10.1126/science.118.3062.273
57. Peever J, Fuller PM. The biology of REM sleep. *Curr Biol*. 2017;**27**(22):R1237–R1248. https://doi.org/10.1016/j.cub.2017.10.026
58. Torontali ZA, Fraigne JJ, Sanghera P, Horner R, Peever J. The sublaterodorsal tegmental nucleus functions to couple brain state and motor activity during REM sleep and wakefulness. *Curr Biol*. 2019;**29**(22):3803–3813.e5. https://doi.org/10.1016/j.cub.2019.09.026
59. Liu D, Li W, Ma C, *et al*. A common hub for sleep and motor control in the substantia nigra. *Science*. 2020;**367**(6476):440–445. https://doi.org/10.1126/science.aaz0956
60. Marder E, Bucher D. Central pattern generators and the control of rhythmic movements. *Curr Biol*. 2001;**11**(23):R986–R996. https://doi.org/10.1016/S0960-9822(01)00581-4
61. Fenk LA, Riquelme JL, Laurent G. Central pattern generator control of a vertebrate ultradian sleep rhythm. *Nature*. 2024;**636**(8043):681–689. https://doi.org/10.1038/s41586-024-08162-w
62. Beaugrand M, Jaramillo V, Mühlematter C, *et al*. Tracing infant sleep neurophysiology longitudinally from 3 to 6 months: EEG insights into brain development. *npj Biol Timing Sleep*. 2026;**3**(1):9. https://doi.org/10.1038/s44323-026-00071-7
63. Wong S, Wright K, Spencer R, Vetter C, Hicks L, Oskar LBMJ. Development of the circadian system in early life: maternal and environmental factors. *J Physiol Anthropol*. 2022;**41**(1):22. https://doi.org/10.1186/s40101-022-00294-0
64. Lodemore MR, Petersen SA, Wailoo MP. Factors affecting the development of night time temperature rhythms. *Arch Dis Child*. 1992;**67**(10):1259–1261. https://doi.org/10.1136/adc.67.10.1259
65. van den Elsen LWJ, Garssen J, Burcelin R, Verhasselt V. Shaping the gut microbiota by breastfeeding: the gateway to allergy prevention? *Front Pediatr*. 2019;**7**:1–10. https://doi.org/10.3389/fped.2019.00047

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

66. Muzet A, Naitoh P, Townsend RE, Johnson LC. Body movements during sleep as a predictor of stage change. *Psychon Sci*. 1972;**29**(1):7–10. https://doi.org/10.3758/BF03336549
67. Daan S, Beersma DG, Borbely AA. Timing of human sleep: recovery process gated by a circadian pacemaker. *Am J Physiol Regul Integr Comp Physiol*. 1984;**246**(2):R161–R183. https://doi.org/10.1152/ajpregu.1984.246.2.R161
68. Webb L, Phillips AJK, Roberts JA, *et al*. Mapping the physiological changes in sleep regulation across infancy and young childhood. *PLoS Comput Biol*. 2024;**20**(10):e1012541. https://doi.org/10.1371/journal.pcbi.1012541
69. Bes F, Schulz H, Navelet Y, Salzarulo PA. The distribution of slow-wave sleep across the night: a comparison for infants, children, and adults. *Sleep*. 1991;**14**(1):5–12. https://doi.org/10.1093/sleep/14.1.5
70. Blyton DM, Sullivan CE, Edwards N. Lactation is associated with an increase in slow-wave sleep in women. *J Sleep Res*. 2002;**11**(4):297–303. https://doi.org/10.1046/j.1365-2869.2002.00315.x
71. Nishihara K, Horiuchi S, Eto H, Uchida S, Honda M. Delta and theta power spectra of night sleep EEG are higher in breast-feeding mothers than in non-pregnant women. *Neurosci Lett*. 2004;**368**(2):216–220. https://doi.org/10.1016/j.neulet.2004.07.021
72. Rojo-Wissar DM, Bai J, Benjamin-Neelon SE, Wolfson AR, Spira AP. Development of circadian rest-activity rhythms during the first year of life in a racially diverse cohort. *Sleep*. 2022;**45**(6):zsac078. https://doi.org/10.1093/sleep/zsac078
73. Blume C, Schoch SF, Vienneau D, *et al*. Association of transportation noise with sleep during the first year of life: a longitudinal study. *Environ Res*. 2022;**203**:111776. https://doi.org/10.1016/j.envres.2021.111776
74. Volkovich E, Ben-Zion H, Karny D, Meiri G, Tikotzky L. Sleep patterns of co-sleeping and solitary sleeping infants and mothers: a longitudinal study. *Sleep Med*. 2015;**16**(11):1305–1312. https://doi.org/10.1016/j.sleep.2015.08.016
75. Volkovich E, Bar-Kalifa E, Meiri G, Tikotzky L. Mother–infant sleep patterns and parental functioning of room-sharing and solitary-sleeping families: a longitudinal study from 3 to 18 months. *Sleep*. 2017; **1**(2):zsx207. https://doi.org/10.1093/sleep/zsx207
76. Schweizerischer Städteverband (SSV) Federal Statistical Office (BFS). *Statistik der Schweizer Städte 2019*. Number 8086839. Bern/Neuchâtel; 2019.

Downloaded from academic.oup.com/sleep/article/49/10/zsag161/8707366 by guest on 16 October 2026

© The Author(s) 2026. Published by Oxford University Press on behalf of Sleep Research Society. This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (https://creativecommons.org/licenses/by-nc/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact reprints@oup.com for reprints and translation rights for reprints. All other permissions can be obtained through our RightsLink service via the Permissions link on the article page on our site—for further information please contact journals.permissions@oup.com.
SLEEP, 2026, **49**, 1–16
https://doi.org/10.1093/sleep/zsag161
Original Article