

RESEARCH ARTICLE



Fetal and infant growth patterns, sleep, and 24-h activity rhythms: a population-based prospective cohort study in school-age children

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Summary

The study objective was to explore associations of fetal and infant weight patterns and preterm birth with sleep and 24-h activity rhythm parameters at school-age. In our prospective population-based study, 1327 children were followed from birth to age 10–15 years. Fetal weight was estimated using ultrasound in the second and third trimester of pregnancy. Birth weight and gestational age were available from midwife registries. Infant weight was measured at 6, 12 and 24 months. Fetal and infant weight acceleration or deceleration were defined as a change of >0.67 standard deviation between the corresponding age intervals. At school-age, sleep duration, sleep efficiency, wake after sleep onset, social jetlag, inter-daily stability, and intra-daily variability were assessed using tri-axial wrist actigraphy for 9 consecutive nights. We observed that low birth weight (<2500 g) was associated with 0.24 standard deviation (95% confidence interval [CI] 0.04; 0.43) longer sleep duration compared to normal weight. Compared to normal growth, growth deceleration in fetal life and infancy was associated with 0.40 standard deviation (95% CI 0.07; 0.73) longer sleep duration, 0.44 standard deviation (95% CI 0.14; 0.73) higher sleep efficiency, and –0.41 standard deviation (95% CI –0.76; –0.07) shorter wake after sleep onset. A pattern of normal fetal growth followed by infant growth acceleration was

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associated with -0.40 standard deviation (95% CI -0.61 ; -0.19) lower inter-daily stability. Preterm birth was not associated with any sleep or 24-h rhythm parameters. Our findings showed that children with fetal and infant growth restriction had longer and more efficient sleep at school-age, which may be indicative of an increased need for sleep for maturational processes and development after a difficult start in life.

KEYWORDS

birth, inter-daily stability, intra-daily variability, preterm, social jetlag, wake after sleep onset

1 | INTRODUCTION

Sleep development starts during fetal life and is associated with the development, maturation, and connectivity of neural networks in the brain (Bennet et al., 2018). Adverse events in fetal life or infancy can disturb this development and may have persistent effects on sleep and 24-h rhythm patterns in childhood (Brockmann et al., 2020; Yiallourou et al., 2018). Studies in children born preterm suggest that preterm birth itself as well as related comorbidities, such as being born small for gestational age (SGA) and cerebral haemorrhage, are associated with disturbed sleep (e.g., difficulties falling asleep and frequent awakenings) at child age (Gogou et al., 2019; Stangenes et al., 2018). Apart from neonatal comorbidities, the premature disconnection to maternal circadian cues and adverse environmental factors (e.g., during hospital admission) may also influence sleep development (Gogou et al., 2019).

A previous study among 52 children using polysomnography, showed that fetal growth restricted (FGR) and preterm born children had reduced sleep duration and efficiency, and altered rapid eye movement (REM) sleep at the age of 5–12 years (Yiallourou et al., 2018). A larger study ($n = 787$) using parental questionnaires, reported different sleep habits (e.g., earlier bedtimes and longer sleep duration) and more sleep problems in children aged 11 years born preterm as compared to children born full-term (Stangenes et al., 2017). Studies of fetal or infant determinants of 24-h activity rhythms in childhood are scarce. Findings from two Finnish actigraphy studies among young adults born preterm and full-term suggest that very low birth weight children might show an earlier chronotype in adult life (Björkqvist et al., 2018, 2020). Possible mechanisms include a longer period of melatonin deficiency after birth and adverse effects on circadian rhythm programming by prenatal hypoxia, and protein malnutrition and environmental factors in the early postnatal period (Björkqvist et al., 2018). The age of onset of developing the suggested earlier chronotype, as well as its relationship with cardiometabolic health, require further investigation. Most previous studies are based on specific clinical neonatal populations, exposed to extreme circumstances before or after birth. To the best of our knowledge, the associations of fetal and infant growth patterns and gestational age across the full range, with sleep and 24-h activity rhythms in childhood, have not been studied yet.

We hypothesised that altered fetal and infant developmental patterns, reflected by gestational age and weight growth are associated

with disturbed sleep and 24-h activity rhythms at school-age in a population-based sample. In a birth cohort including 1327 mothers and children, we examined the associations of preterm birth and growth in early life with actigraphy estimated measures of sleep and 24-h activity rhythms at the age of 10–15 years.

2 | METHODS

2.1 | Study design and participants

This analysis was performed in the Generation R Study, a prospective population-based cohort study from early fetal life onward in Rotterdam, The Netherlands (Kooijman et al., 2016). The study has been approved by the Medical Ethical Committee of the Erasmus MC Rotterdam (MEC 198.782/2001/31). Written informed parental consent was obtained for all participants. Pregnant women living in Rotterdam with an expected delivery between April 2002 and January 2006 were eligible for study participation (61% included $n = 9778$). We had information on fetal or infant growth of 9257 singleton births (Vogelezang et al., 2019). Between September 2015 and June 2018, a subsample of 1910 children who attended the regular 11- ($n = 1152$, median age 11.7 years) or 14-year study visit ($n = 758$, median age 14.7 years) was asked to participate in an actigraphy sub-study, of whom 1483 (77.6%) gave consent. In this subgroup, children born preterm were oversampled because of specific interest in the long-term consequences of preterm birth. We excluded children from multiple pregnancies, children without any data on weekday sleep, and children in whom none of the recorded nights passed standard quality control (>6 h wear time, >4 h detected sleep time) (Koopman-Verhoeff, Bolhuis, et al., 2019; Koopman-Verhoeff, Serdarevic, et al., 2019).

2.2 | Fetal and infant growth measures

Fetal ultrasound examinations were performed in all three trimesters by well-trained researchers according to clinical standards, as described previously (Kooijman et al., 2016; Vogelezang et al., 2019). Last menstrual period or first-trimester ultrasound was used to establish gestational age (Tunón et al., 1996). Fetal ultrasounds in the second trimester were performed at a median (interquartile range [IQR])

of 20.5 (20.0–21.2) weeks, and in the third trimester at 30.4 (29.8–30.9) weeks. Head circumference, abdominal circumference, and femur length were measured to the nearest millimetre. Fetal weight was estimated by measuring head circumference, abdominal circumference, and femur length (to the nearest millimetre) using the formula by Hadlock et al. (1984). We calculated sex-adjusted standard deviation scores (SDS) for estimated fetal weight. Gestational age at birth was divided in categories of preterm birth (<37 weeks), normal term birth (37–41 weeks), and late term birth (>41 weeks). Birth weight was obtained from community midwife and hospital registries and divided in categories of <2500, 2500–4250, and >4250 g. We calculated sex- and gestational age-adjusted SDS for birth weight within our study population using the Growth Analyzer 3.5 (Dutch Growth Research Foundation), based on North European reference charts (Niklasson et al., 1991). Children born SGA were defined as sex- and gestational age-adjusted SDS for birth weight below the tenth percentile, and those born large for gestational age as sex- and gestational age-adjusted SDS for birth weight above the 90th percentile (Vogelezang et al., 2019).

Infant weight was measured in community health centres with a mechanical personal scale at a median (IQR) of 6.2 (6.0–6.4), 11.0 (10.7–11.4), and 24.8 (24.2–25.6) months, further referred to as the 6-, 12-, and 24-month visits (Kooijman et al., 2016; Vogelezang et al., 2019). Age- and sex-adjusted SDS were created using Dutch reference growth charts (Fredriks et al., 2000). Fetal growth was defined as growth between the second trimester and birth, and infant growth as growth from birth to 24 months. For both fetal and infant growth, we defined a decrease of >0.67 SD between time-points as growth deceleration, an increase of >0.67 SD between time-points as growth acceleration, and growth in between (–0.67 to +0.67 SD) as normal growth (Vogelezang et al., 2019). Combining the growth categories of the fetal period with the growth categories of the infant period, yielded nine different growth patterns for the total period of second trimester until 24 months.

2.3 | Sleep and 24-h activity rhythm measures

At both the 11- and 14-year visit, we assessed sleep using a tri-axial wrist actigraph (GENEActiv; Activinsights, UK), as described previously (Koopman-Verhoeff, Serdarevic, et al., 2019). In short, children wore the device for 9 consecutive nights (5 school nights and 4 weekend nights) on their non-dominant wrist. A recent study showed acceptable reliability of the GENEActiv actigraph to estimate sleep in children aged 7–12 years when a minimum of 3–5 nights were measured (Antczak et al., 2021). Additionally, each morning children completed a sleep diary with questions about their previous night's sleep. No sleep measurements were performed during school holidays or within 7 days after the start or end of daylight saving time. Actigraphs were set at a frequency of 50 Hz and raw sleep data were processed with the R-package GGIR using an algorithm with 5-s epochs (van Hees et al., 2014). All days with >16 h wear time per 24 h, were included in the analyses. This procedure generated the following sleep

measures: (i) sleep duration (total duration of estimated sleep between sleep onset and final waking in minutes); (ii) sleep efficiency (percentage of time spent asleep between sleep onset and final waking time); and (iii) wake after sleep onset (WASO, total time awake between sleep onset and final awakening). Additionally, we calculated 24-h activity rhythm parameters: (iv) social jetlag (average midpoint sleep during the weekend subtracted by the average midpoint sleep during the week, in hours), (v) intra-daily variability (indication of fragmentation of the sleep rhythm, ranging from 0 to 2, with higher scores indicating more fragmentation), and (vi) inter-daily stability (indicating the stability of the 24-h activity rhythm across days, ranging from 0 to 1, with higher scores indicating more stable rhythms) (Mitchell et al., 2017; van Hees et al., 2015; van Someren et al., 1996). Intra-daily variability and inter-daily stability were selected as non-parametric indicators of the 24-h activity rhythm as previous research showed that these measures correlated highly with the relative amplitude but were more specific (Luik et al., 2013). For homogeneity, we only used school days for the measures of sleep duration, sleep efficiency, and WASO, to minimise the influence of atypical weekend events (Koopman-Verhoeff, Bolhuis, et al., 2019; Koopman-Verhoeff, Serdarevic, et al., 2019). We included weekend sleep in a separate sensitivity analysis to test the robustness of this assumption.

2.4 | Covariates

Information on maternal factors included educational level (based on highest attained educational level and categorised as low, middle, or high), pre-pregnancy body mass index (BMI), folic acid use during pregnancy (yes/no), smoking and alcohol use during pregnancy (continued/no), and breastfeeding at 2 months (yes/no) were assessed by questionnaires. The sex and age of the child were obtained from medical records. Child ethnicity was based on country of birth of the parents (Dutch or non-Dutch). Season of sleep assessment was defined as 'Spring', 'Summer', 'Autumn', or 'Winter'.

2.5 | Statistical analysis

First, we checked the correlations of all fetal and infant growth exposure measures with all sleep and 24-h activity rhythm outcome variables. Second, we used linear regression to estimate the associations of the main birth outcomes (gestational age, birth weight, size for gestational age; both continuous and per category) with sleep outcomes (sleep duration, sleep efficiency, WASO) and 24-h activity rhythm outcomes (social jetlag, intra-daily variability, inter-daily stability). Third, we used linear regression to estimate the associations of the nine different growth patterns of combined fetal and infant growth (with the pattern of normal fetal growth-normal infant growth as the reference period), with sleep and 24-h activity rhythms. All assumptions of linear regression were met for all analyses. We checked whether covariates were associated with both exposures and main outcomes (sleep duration, WASO, social jetlag, intra-daily variability), or changed the effect

estimate >10% when added to the models. We used three models: a 'basic model' adjusted for season and age at sleep assessment; a main 'confounder' model that was additionally adjusted for maternal education, pre-pregnancy BMI, folic acid use, smoking, and alcohol during pregnancy, as well as child sex, ethnicity, and breastfeeding at 2 months; and a 'BMI' model in which we additionally adjusted for child BMI at sleep assessment. Because of skewed distributions of sleep duration, WASO, and inter-daily stability, we used natural logged values for sleep duration and inter-daily stability, and square root values for WASO, in all linear regression analyses. For comparison of effect estimates, we calculated SDS (observed value-mean/SD) for all outcome measures. To take into account multiple testing, we present results based on statistical significance on $p < 0.05$ and $p < 0.025$ (based on two main outcomes groups: sleep and 24-h activity rhythm). We considered more extensive Bonferroni correction too strict because of several intercorrelated exposures and outcomes.

As the amount and timing of sleep changes during adolescence (Carskadon et al., 2004), we first explored whether associations were different between those measured at the 11- and 14-year visit. We observed statistically significant interactions of timing of visit with gestational age at birth and multiple weight parameters (birth weight; weight SDS at birth, third trimester, 24 months) for sleep efficiency, WASO, social jetlag and inter-daily stability. We therefore performed additional stratified analyses on study visit group as sensitivity analyses. No interactions were observed between sex and gestational age or weight parameters in the associations with sleep or 24-h rhythm. Second, to test the robustness of associations, we reran the confounder models using combined weekend and weekday sleep. For all analyses, we used multiple imputations for missing covariates using the Markov chain Monte Carlo approach. Five datasets were created, and pooled results were reported. Statistical analysis was carried out using the IBM Statistical Package for the Social Sciences (SPSS), version 25.0 (IBM Corp., Armonk, NY, USA).

3 | RESULTS

3.1 | Subject characteristics

The final population for analysis of this study comprised 1327 children (Figure 1). Table 1 shows that 10% of children were born preterm (<37 weeks) and 7% with low birth weight (<2500 g). In all, 95% of the children wore the device for ≥ 7 days. The median (IQR) sleep duration was 7.5 (6.9–8.0) h and mean (SD) social jetlag was 1.0 (0.7) h. The correlations between exposures and outcomes are presented in Supporting information: Table S1.

3.2 | Birth outcomes, sleep, and 24-h rhythms

Low birth weight was associated with 0.24 SDS (95% confidence interval [CI] 0.04;0.43) longer sleep duration (Table 2). The associations between preterm birth and higher intra-daily variability, between low birth weight and shorter WASO, and between small birth size for gestational age and longer social jetlag, all attenuated to non-significance after correction for multiple testing. Gestational age, birth weight or birth size across the full range were not associated with sleep or 24-h activity rhythms.

3.3 | Fetal and infant growth patterns, sleep, and 24-h rhythms

As compared to normal fetal and infant growth, a pattern of continued growth deceleration in both fetal life and infancy was associated with 0.40 SDS (95% CI 0.07;0.73) longer sleep duration, 0.44 SDS (95% CI 0.14;0.73) higher sleep efficiency, and -0.41 SDS (95% CI -0.76 ; -0.07) shorter WASO (Table 3). Furthermore, a pattern of

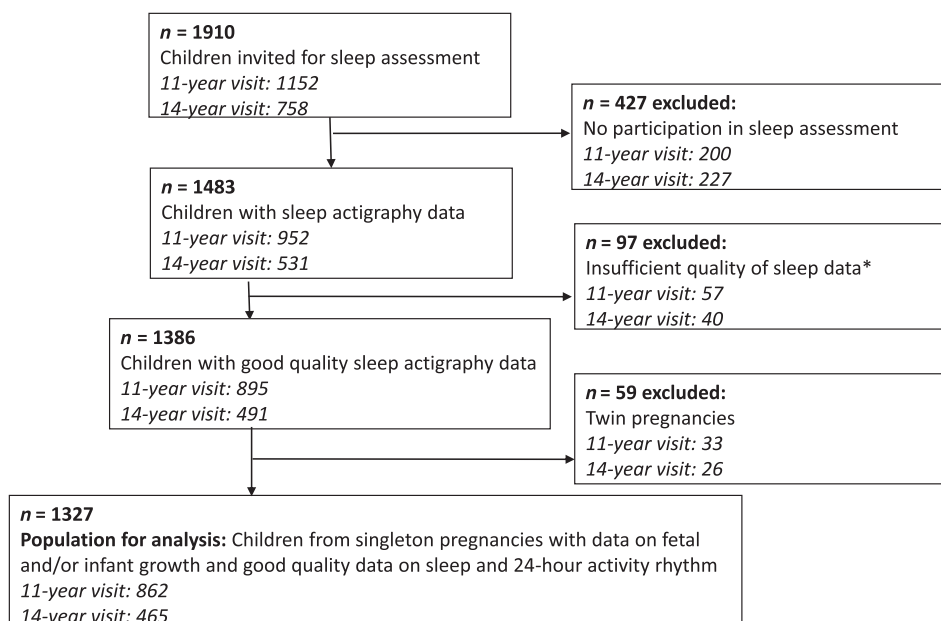


FIGURE 1 Flowchart of the study population. *No data available on weekday sleep, or none of the night data passing standard quality control (>6 h wear time, >4 h detected sleep time).

TABLE 1 Baseline characteristics of the study population

Characteristic	N	Total population (N = 1327)
Maternal characteristics		
Age at intake, years, mean (SD)	1327	32.1 (4.1)
Pre-pregnancy BMI, kg/m ² , median (IQR)	1086	22.5 (20.8–24.9)
Education, n (%)	1291	
Low/middle		487 (37.7)
High		804 (62.3)
Folic acid use during pregnancy, yes, n (%)	1027	925 (90.1)
Smoking during pregnancy, continued: yes, n (%)	1196	147 (12.3)
Alcohol use during pregnancy, continued: yes, n (%)	1147	611 (53.3)
Fetal and child characteristics		
Sex, female, n (%)	1327	707 (53.3)
Ethnicity, n (%)	1326	
Dutch		1093 (82.4)
Non-Dutch		233 (17.6)
Fetal period		
Second trimester, median (IQR)		
Gestational age, weeks	1240	20.5 (20.0–21.2)
Estimated fetal weight, g	1221	367 (326–420)
Third trimester, median (IQR)		
Gestational age, weeks	1245	30.4 (29.8–30.9)
Estimated fetal weight, g	1234	1619 (1462–1771)
Birth		
Gestational age at birth, weeks, median (IQR)	1325	40.1 (39.0–41.0)
Preterm birth, <37 weeks gestational age, n (%)		133 (10.0)
Late birth, >41 weeks gestational age, n (%)		327 (24.7)
Birth weight, g, median (IQR)	1327	3500 (3100–3860)
Low birth weight, <2500 g, n (%)		93 (7.0)
High birth weight, >4250 g, n (%)		97 (7.3)
Birth weight SD-score, SD, mean (SD)	1325	0.1 (1.0)
Birth size	1325	
Small for gestational age, <10th percentile, n (%)		102 (7.7)
Large for gestational age, >90th percentile, n (%)		167 (12.6)
Infancy		
Breastfeeding at 2 months, yes, n (%)	1145	798 (69.7)
At 6 months, median (IQR)		
Age at visit, months	1129	6.2 (6.0–6.4)
Weight, kg	1126	7.8 (7.2–8.4)
At 12 months, median (IQR)		
Age at visit, months	1059	11.0 (10.7–11.4)
Weight, kg	1052	9.6 (8.9–10.0)
At 24 months, median (IQR)		
Age at visit, months	1025	24.8 (24.2–25.6)
Weight, kg	1024	12.8 (12.0–13.8)
Childhood (10–15 years)		
Age at sleep assessment, years, median (IQR)	1327	11.8 (11.6–14.5)
BMI at centre visit, kg/m ² , mean (SD)	1322	17.9 (2.9)
Overweight/obesity, n (%)		160 (12.1)

(Continues)

TABLE 1 (Continued)

Characteristic	N	Total population (N = 1327)
Season of sleep assessment, n (%)	1325	
Winter		402 (30.3)
Spring		485 (36.6)
Summer		387 (29.2)
Autumn		51 (3.8)
Actigraphy		
Sleep duration, h, median (IQR)	1325	7.5 (6.9–8.0)
Sleep efficiency, %, mean (SD)	1325	84.6 (5.5)
Wake after sleep onset, min, median (IQR)	1325	79 (60–100)
Social jetlag, h, mean (SD)	1321	1.0 (0.7)
Sleep midpoint weekdays, time, mean (SD in min)	1325	02:51 (42)
Sleep midpoint weekends, time, mean (SD in min)	1324	03:52 (59)
Intra-daily variability, mean (SD)	1324	0.56 (0.10)
Inter-daily stability, median (IQR)	1324	0.17 (0.14–0.20)
Sleep diary		
Sleep duration, h, mean (SD)	1258	9.1 (1.1)
Nightly awakenings, n, mean (SD)	1277	0.5 (0.7)

Abbreviations: BMI, body mass index; IQR, interquartile range; SD, standard deviation.

normal fetal growth followed by infant growth acceleration was associated with a -0.40 SDS (95% CI -0.61 ; -0.19) lower inter-daily stability. Results from the basic models are presented in Supporting Information: Tables S2 and S3. Supporting Information: Tables S4 and S5 show that findings were similar after additional adjustment for BMI.

3.4 | Sensitivity analyses

Stratified analyses per study visit revealed that the associations of low birth weight with sleep duration and WASO, and of small birth size with social jetlag were similar but stronger at the 14-year visit (Supporting Information: Table S6). The associations of the growth patterns of continued fetal and infant growth deceleration with sleep duration, sleep efficiency and WASO, and of normal fetal growth with infant growth acceleration with inter-daily stability, were in the same direction at both visits (Supporting Information: Table S7). However, the smaller group sizes in the subgroup analyses yielded small differences in effect estimates and reaching significance. Results were very similar when we included weekend sleep in the sleep measures (Supporting Information: Tables S8 and S9).

4 | DISCUSSION

In this large prospective population-based birth cohort, we observed that both low birth weight and a pattern of continued fetal and infant growth deceleration were associated with longer sleep duration at the age of 10–15 years. The pattern of continued growth deceleration

was also associated with higher sleep efficiency and shorter WASO, whereas a pattern of normal fetal growth followed by infant growth acceleration was associated with lower inter-daily stability. Subgroup analyses per study visit showed that associations of gestational age across the full range and low birth weight with WASO were stronger and only significant in children aged 14–15 years, as compared to children aged 10–11 years. The same observation applied to the association of birth size, both small and across the full range, with social jetlag. We observed no interactions with child sex.

Our findings that children with low birth weight or continued fetal and infant growth restriction showed more favourable sleep outcomes, as expressed by longer sleep duration, higher sleep efficiency and shorter WASO, partly aligned with previous studies (Yiallourou et al., 2017, 2018). Only a limited number of studies have investigated the effects of fetal growth restriction on childhood sleep, showing conflicting results (Leitner et al., 2002; Pesonen et al., 2009; Yiallourou et al., 2018). Using polysomnography in children aged 5–12 years, Yiallourou et al. presented comparable results to ours by demonstrating that FGR children born preterm showed longer sleep duration, higher sleep efficiency and shorter WASO than preterm children who were born with appropriate birth weight for gestational age (AGA), but not as compared to their term AGA peers (Yiallourou et al., 2017, 2018). By contrast, actigraphy studies of Leitner et al. (2002) and Pesonen et al. (2009) showed shorter sleep, lower sleep efficiency and/or more awakenings in children aged 4–8 years with fetal growth restriction or low birth weight, respectively, as compared to children with normal birth weight. A possible explanation for these remarkable conflicting results is that in FGR children, sleep characteristics evolve differently over the course of childhood as compared to their normal birth weight peers; but further research at school-age is

TABLE 2 Associations of birth outcomes with childhood sleep and 24-h activity rhythms (confounder model)

Birth outcomes	N	Difference (95% confidence interval) in SDS					
		Sleep duration	Sleep efficiency	WASO	Social jetlag	Intra-daily variability	Inter-daily stability
Gestational age							
<37 weeks	133	0.15 (−0.01;0.32)	0.08 (−0.10;0.26)	−0.10 (−0.28;0.07)	−0.02 (−0.20;0.16)	0.20 (0.02;0.38)	−0.11 (−0.29;0.06)
37–41 weeks	863	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
>41 weeks	327	−0.04 (−0.15;0.08)	0.01 (−0.12;0.13)	−0.01 (−0.13;0.11)	0.08 (−0.04;0.20)	−0.05 (−0.17;0.07)	0.07 (−0.05;0.19)
Trend (per week)	1323	−0.02 (−0.04;0.00)	−0.02 (−0.04;0.01)	0.02 (−0.00;0.05)	0.01 (−0.02;0.03)	−0.02 (−0.04;0.01)	0.02 (−0.01;0.04)
Birth weight							
<2500 g	93	0.24 (0.04;0.43)*	0.17 (−0.04;0.38)	−0.21 (−0.42;−0.01)	0.04 (−0.17;0.25)	0.09 (−0.12;0.30)	−0.12 (−0.32;0.08)
2500–4250 g	1135	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
>4250 g	97	−0.02 (−0.20;0.17)	0.20 (−0.01;0.40)	−0.20 (−0.39;0.00)	−0.01 (−0.21;0.19)	0.09 (−0.11;0.29)	−0.02 (−0.21;0.18)
Trend (per 500 g)	1325	−0.04 (−0.08;0.00)	−0.01 (−0.06;0.03)	0.02 (−0.03;0.06)	−0.02 (−0.06;0.03)	−0.01 (−0.05;0.03)	0.02 (−0.02;0.06)
Size for gestational age							
Small (<p10)	102	−0.01 (−0.20;0.17)	−0.02 (−0.22;0.18)	0.05 (−0.14;0.24)	0.22 (0.02;0.41)	0.08 (−0.12;0.27)	0.03 (−0.16;0.22)
Appropriate (p10–p90)	1054	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
Large (>p90)	167	−0.06 (−0.20;0.09)	0.08 (−0.09;0.24)	−0.07 (−0.22;0.09)	0.02 (−0.14;0.21)	0.05 (−0.11;0.20)	−0.00 (−0.15;0.15)
Trend (per SDS)	1323	−0.02 (−0.06;0.03)	0.02 (−0.03;0.07)	−0.03 (−0.08;0.02)	−0.04 (−0.09;0.01)	0.02 (−0.03;0.07)	0.01 (−0.04;0.06)

Note: Values are linear regression coefficients (95% confidence intervals) and reflect the change in sleep and 24-h activity rhythm parameters per birth outcome. Models are adjusted for season and age at sleep assessment; child sex and ethnicity; maternal pre-pregnancy body mass index, educational level, smoking, alcohol, and folic acid use during pregnancy; and breastfeeding at 2 months. Bold = $p < 0.05$.

Abbreviations: p, percentile, SDS, standard deviation score; WASO, wake after sleep onset (min).

TABLE 3 Associations of fetal and infant growth patterns with childhood sleep and 24-h activity rhythms (confounder model)

Growth patterns	N	Difference (95% confidence interval) in SDS					
		Sleep duration	Sleep efficiency	WASO	Social jetlag	Intra-daily variability	Inter-daily stability
Fetal growth deceleration							
Infant growth deceleration	30	0.40 (0.07;0.73)*	0.44 (0.14;0.73)*	-0.41 (-0.76;-0.07)*	-0.08 (-0.43;0.27)	-0.08 (-0.44;0.28)	-0.22 (-0.57;0.13)
Infant normal growth	113	-0.02 (-0.21;0.18)	-0.05 (-0.27;0.16)	0.01 (-0.19;0.22)	0.02 (-0.19;0.22)	-0.04 (-0.25;0.17)	-0.20 (-0.40;0.00)
Infant growth acceleration	115	-0.01 (-0.20;0.19)	0.12 (-0.09;0.33)	-0.12 (-0.32;0.09)	0.03 (-0.18;0.23)	-0.16 (-0.37;0.06)	0.09 (-0.11;0.30)
Fetal normal growth							
Infant growth deceleration	113	-0.07 (-0.26;0.13)	-0.03 (-0.24;0.19)	-0.04 (-0.24;0.16)	-0.04 (-0.24;0.17)	0.02 (-0.19;0.24)	0.01 (-0.19;0.21)
Infant normal growth	258	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]	[Reference]
Infant growth acceleration	104	-0.07 (-0.27;0.13)	-0.04 (-0.26;0.18)	-0.01 (-0.22;0.20)	-0.03 (-0.24;0.18)	-0.00 (-0.22;0.21)	-0.40 (-0.61;-0.19)*
Fetal growth acceleration							
Infant growth deceleration	163	-0.03 (-0.20;0.15)	-0.02 (-0.20;0.17)	0.01 (-0.17;0.19)	-0.03 (-0.22;0.15)	0.01 (-0.17;0.20)	-0.15 (-0.33;0.03)
Infant normal growth	164	-0.13 (-0.30;0.04)	0.05 (-0.13;0.24)	-0.10 (-0.28;0.08)	-0.04 (-0.23;0.14)	-0.11 (-0.30;0.08)	-0.15 (-0.33;0.03)
Infant growth acceleration	41	0.07 (-0.22;0.36)	0.13 (-0.19;0.45)	-0.10 (-0.41;0.20)	-0.09 (-0.39;0.22)	0.09 (-0.22;0.41)	-0.28 (-0.58;0.02)

Note: Values are linear regression coefficients (95% confidence intervals) and reflect the difference in sleep and 24-h activity rhythm parameters compared to children with normal fetal and infant growth. Models are adjusted for season and age at sleep assessment; child sex and ethnicity; maternal pre-pregnancy body mass index, educational level, smoking, alcohol, and folic acid use during pregnancy; and breastfeeding at 2 months. Bold = $p < 0.05$, bold* = $p < 0.025$.

Abbreviations: SDS, standard deviation score; WASO, wake after sleep onset (min).

required. Although there is overlap between children born preterm and children with low birth weight or fetal growth restriction, we did not observe any associations with preterm birth or gestational age across the full range in our full group analyses. These findings only partly agree with results of previous studies on sleep duration in school-age children born preterm, which are also not consistent (Bennet et al., 2018; Gogou et al., 2019; Visser et al., 2021). Some studies reported longer sleep duration (Stangenes et al., 2017), while others reported no differences (Brockmann et al., 2020; Iglowstein et al., 2006; Perkinson-Gloor et al., 2015), or shorter sleep in those born preterm (Biggs et al., 2016; Yiallourou et al., 2018).

As for 24-h rhythms, we showed that at the 14-year visit, birth size was linked to social jetlag, with children born SGA having greater misalignment in sleep/wake timing between weekdays and weekends than children with normal size at birth. The rapid changes of the sleep-wake rhythms during adolescence may explain why we did not observe this association at the 11-year visit, as social jetlag may become more prominent at an older age (Carskadon et al., 2004). To the best of our knowledge, this is the first study to investigate early growth and social jetlag in childhood, which hampers comparison of our results with previous research. A suggested risk factor for social jetlag is a late(r) chronotype, when sleep debt builds up during the week to be compensated during the weekend (Wittmann et al., 2006). Chronotype has previously been investigated in relation to birth outcomes. However, those studies have not described a later but in fact an earlier chronotype (earlier bed and waking times) in young adults and adolescents born preterm, especially in those born SGA (Björkqvist et al., 2014, 2020; Hibbs et al., 2014). This earlier chronotype may reflect developmental influences, as well as genetic alterations and/or parenting styles, and may be protective for later mental and physical health (Hibbs et al., 2014; Mainieri et al., 2021). Based on these findings, the association of birth size and social jetlag would have been expected in the opposite direction as observed in our study. Interestingly, a growth pattern of normal fetal growth and accelerated infant growth was associated with a decrease in inter-daily stability. This may suggest that catch-up growth might affect the consolidation of a stable 24-h activity rhythm at school-age. However, as there are no studies for comparison, further research on perinatal and infant growth influences on 24-h rhythms in childhood is essential.

The mechanisms underlying the associations of early growth, sleep, and 24-h rhythms are not fully understood and are likely multifactorial. Development of sleep and 24-h rhythms already starts during the early fetal period when brain maturation commences and neural networks become more coherent (Bennet et al., 2018). As from 32 weeks gestational age, four different sleep-wake states can be distinguished. Preterm birth affects this developmental process of sleep and 24-h rhythm, likely mostly as a result of adverse brain growth (Bennet et al., 2018; Kocevskaja et al., 2018; Pascal et al., 2018). This is even more prominent in FGR infants, in whom even lower neural myelination and a larger reduction in structure and organisation of neural connections between brain regions have been observed (Bennet et al., 2018). Other mediating factors are thought to be fetal and neonatal hypoxia, the loss of placental steroids and hormones,

inflammation, genetic alterations, and environmental factors during the neonatal period (Bennet et al., 2018). Previous research in infants 0–6 months corrected age suggested that those born preterm show an earlier emergence of a circadian 24-h rhythm than their full-term peers, potentially due to longer exposition to external time cues such as light (Guyer et al., 2015). How this finding relates to the previously described 'early' chronotype later in life in this population, requires further investigation.

The higher sleep quantity and quality observed in children with low birth weight and early growth restriction could be the result of earlier bed times (possibly related to more protective parenting) and/or an increased need for restorative sleep to benefit (catch-up) growth, but could also be related to certain behavioural traits more often seen in preterm born or FGR children, such as requiring extra time for processing of stimuli experienced during the day (Caravale et al., 2017). Although further research is needed to understand their increased sleep (need) and its consequences for future health, our findings emphasise the importance of sleep and 24-h rhythm at school-age in this vulnerable group of children. Therefore, exploration of sleep and 24-h rhythms should be integrated into neonatal follow-up of children born preterm and SGA.

4.1 | Strengths and limitations

Major strengths of this study are the prospective analysis in an ongoing birth cohort study with a large sample size and extensive information on perinatal and sociodemographic risk factors, which permitted correction for multiple confounders. Using weight measurements at six different time-points from second trimester until 24 months of age enabled us to construct and study nine different growth patterns of combined fetal and infant growth. Another strength is the use of objectively measured sleep and 24-h rhythms using actigraphy rather than subjective (parental) report (Meltzer et al., 2012). Lastly, this is, to our knowledge, the first study to investigate how pre-, peri- and postnatal growth associates to 24-h activity rhythms in school-age children.

Some study limitations should also be considered. Being a population-based study, the number of children born preterm in this study (10%) was a fairly good representation of the national prevalence of preterm birth (7%) (van Zijl et al., 2020). However, the proportion of children born very preterm (<30 weeks gestation) in our study was low ($n = 11$, 0.8%). This might have limited statistical power to detect significant associations for preterm birth/gestational age and affects the generalisability of our findings to this specific group, in which sleep problems were described to occur more frequently (Stangenes et al., 2017). Furthermore, the observed differences in associations between the 11- and 14-year visit suggest that the onset of puberty may play a role. However, we were unable to correct for pubertal status as insufficient information on Tanner stages was available. Follow-up studies are needed to assess the observed differences between late childhood and (early) adolescence. Finally, although many covariates were included, residual confounding might still be an issue, as in any observational study. For instance, the influence of parenting and other socio-ecological factors were not included in this

study but could have confounded our results (Meltzer et al., 2021). Second, although we corrected for BMI at sleep assessment, future research should further explore the possible mediating role of physical activity and adiposity in childhood in the association of early growth, sleep, and 24-h rhythms.

5 | CONCLUSION

We observed that children with fetal and/or infant growth restriction showed better sleep quantity and quality at school-age. These findings may be indicative of an increased need for sleep in these children, which they may use for maturational processes and development after a more complex start in life.

AUTHOR CONTRIBUTIONS

Ms Victoria A. A. Beunders conceptualised and designed the study, carried out the analyses and interpretation of the data, drafted the initial manuscript, and reviewed and revised the manuscript. Dr M. Elisabeth Koopman-Verhoeff conceptualised and designed the study, co-ordinated and supervised data collection, assisted in data analysis and interpretation, and reviewed and revised the manuscript. Dr Carolina C. V. Silva assisted in data analysis and interpretation, and critically reviewed the manuscript for important intellectual content. Prof. Jansen, Dr Annemarie I. Luik, Dr Marijn J. Vermeulen, Prof. Irwin K. M. Reiss and Prof. Koen F. M. Joosten assisted in data interpretation and critically reviewed the manuscript for important intellectual content. Prof. Vincent W. V. Jaddoe conceptualised and designed the study, co-ordinated and supervised data collection, interpreted the data and reviewed and revised the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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CONFLICT OF INTEREST

The authors declare no conflict of interest or financial ties to products in this study.

DATA AVAILABILITY STATEMENT

The data underlying this article will be shared on reasonable request to the corresponding author.

DISCLOSURE STATEMENT

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