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ORIGINAL ARTICLE



Influence of radiofrequency electromagnetic fields exposure on sleep patterns in preterm neonates

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ABSTRACT

Purpose: The study objective was to assess the influence of radiofrequency electromagnetic fields (RF-EMF) exposure on sleep patterns in preterm newborns. We hypothesized that an increase in RF-EMF exposure levels would alter infants' sleep structure parameters.

Materials and methods: Individual, continuous measurements of RF-EMF levels were performed in 29 hospitalized preterm newborns throughout the first 21 days after birth. The last day, overnight sleep structure was recorded by polysomnography. Relationships between both chronic (three-week period) and acute (polysomnographic period) RF-EMF levels with sleep parameters were computed.

Results: At median levels, the main chronic effect was an increase in indeterminate sleep with RF-EMF exposure. At the highest exposure levels found in our study, an increase in RF-EMF levels increased sleep fragmentation. No significant relationship was found between acute RF-EMF levels and sleep parameters.

Conclusions: Despite no consolidated disruption in sleep structure, this study is the first to show that some sleep parameters seem to have a certain sensitivity to chronic – but not acute – RF-EMF exposure in preterm newborns. Further studies are needed to confirm our results and examine possible mid- to long-term, sleep-related cardiorespiratory and neurodevelopmental outcomes.

Abbreviations: ECG: electrocardiogram; EEG: electroencephalographic leads; EOG: electrooculogram; IS: indeterminate sleep; NICU: neonatal intensive care unit; NREMs: non-rapid eye movements; P99: 99th percentile; RF-EMFs: radiofrequency electromagnetic fields; REM: rapid eye movements sleep; SPT: sleep period time; TST: total sleep time; WASO: wakefulness after sleep onset

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Introduction

Neonates spend a major part of their early life asleep. The early developmental period features major changes in sleep patterns, reflecting the degree of brain maturation and plasticity. Sleep–wake state integrity is also of paramount importance in the neurophysiological development (e.g. body growth and vital functions) of neonates (Curzi-Dascalova et al. 1993; Mirmiran and Ariagno 2009) and preterm neonates in particular.

We recently reported that preterm neonates are chronically exposed to low environmental radiofrequency electromagnetic field (RF-EMF) levels while hospitalized in neonatal intensive care units (NICUs) (Besset et al. 2020). Preterm neonates are very sensitive to their environment such as EMF (Peyman 2011). Strikingly, there are currently no data on the impact of RF-EMF exposure on sleep in this population. In adults, most of polysomnographic studies in the literature showed that RF-EMF exposure may not disturb clinical features (macrostructure) of sleep in humans

(Lowden et al. 2019; Danker-Hopfe et al. 2020) or in rats (Crouzier, Debouzy, et al. 2007; Crouzier, Testylier, et al. 2007). However, it also reported structural modifications of sleep with either a delayed appearance of rapid eye movement sleep (REM) (Loughran et al. 2005) or a 30% increase in REM sleep (Danker-Hopfe et al. 2016) after mobile phone exposure in other studies. In juvenile rats, studies by our group showed an increase in the frequency of REM episodes in mobile phone exposed animals (Pelletier et al. 2013).

Above studies do not show definitive evidence about deleterious effects of RF-EMF exposure on sleep in adults. However, RF-EMF should merit special attention in preterm neonates as regards to the crucial role of sleep in neurophysiological development and their high sensitivity to the environment (Tamashiro and Moran 2010).

The goal of the present study was to assess the influence of RF-EMF hospital exposure on sleep patterns in the preterm neonate. We hypothesized that an increase in RF-EMF exposure levels would alter sleep structure parameters.

Materials and methods

Infants

Twenty-nine preterm neonates (18 girls and 11 boys) were recruited from the NICU at Amiens University Medical Center (France) after their parents had been informed of the protocol and had given their written consent. The anthropomorphic characteristics are presented in Table 1. The protocol was approved by the protection of persons committee ('Comité de protection des personnes du Sud-Est III', project no. 18.10.12.31915). All neonates were free of neurological, respiratory, and cardiac diseases. Some infants received mechanical ventilation. Recordings were performed after determining by practitioner that there had been no changes in health status in previous days. None of the infants had suffered from sleep deprivation in the 48 hours prior to the sleep recording session.

Radiofrequency electromagnetic field measurements

Methods, equipment and exposure values have been previously described (Besset et al. 2020, 2021). Briefly, recordings were performed using three-axis exposimeters (EME Spy 200, Microwave Vision Group, Paris, France). Attached to the incubator wall of each newborn. In this way,

measurements of RF-EMF levels were made, for each newborn, at only one single location, 30 cm away from the child's head in order to reflect individual, specific exposure. RF-EMF measurements were made every two minutes throughout the first 3 weeks after birth (15,120 measurement points per infant). Individual RF-EMF exposure level was expressed as the median and the 99th percentile (P99). The median represented half of the exposure period, i.e. 10.5 days, whereas P99 represented 1% of the highest values, i.e. 5h24 over the whole recording period. The same RF-EMF measurements and analyses were also performed for each infant during his polysomnographic period (Figure 1).

Sleep analysis

Overnight polysomnographic recordings were performed 3 weeks after birth between 8:00 p.m. and 8:00 a.m. Electrophysiological recordings included six right and left electroencephalographic (EEG) derivations (F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1), an electrooculogram (EOG), and an electrocardiogram (ECG). Body movements, respiratory movements by actimeter and transcutaneous arterial oxygen saturation were also recorded. All recordings were continuously monitored on a polysomnograph

Table 1. Clinical characteristics of the subjects.

Gestational age (weeks of amenorrhea)	29 (27–33)
Birth weight (g)	1230 (550–2344)
Percentile weight (%)	58 (2–96)
Artificial ventilation (number of days)	20 (1–21)
O ₂ supplementation (>21%) (number of days)	3 (0–20)
weight at study (g)	1578 (740–2578)
# of infants under artificial ventilation at study (%)	55
# of infants under O ₂ supplementation (>21%) at study (%)	24
# of infants under caffeine treatment at 3 days before study (%)	93
# of infants under neurological/cardiological treatment at 3 days before study (%)	0

Values are quoted as the median (range), percentile weight calculated according to Chou et al. (2020) and Fenton and Kim (2013).

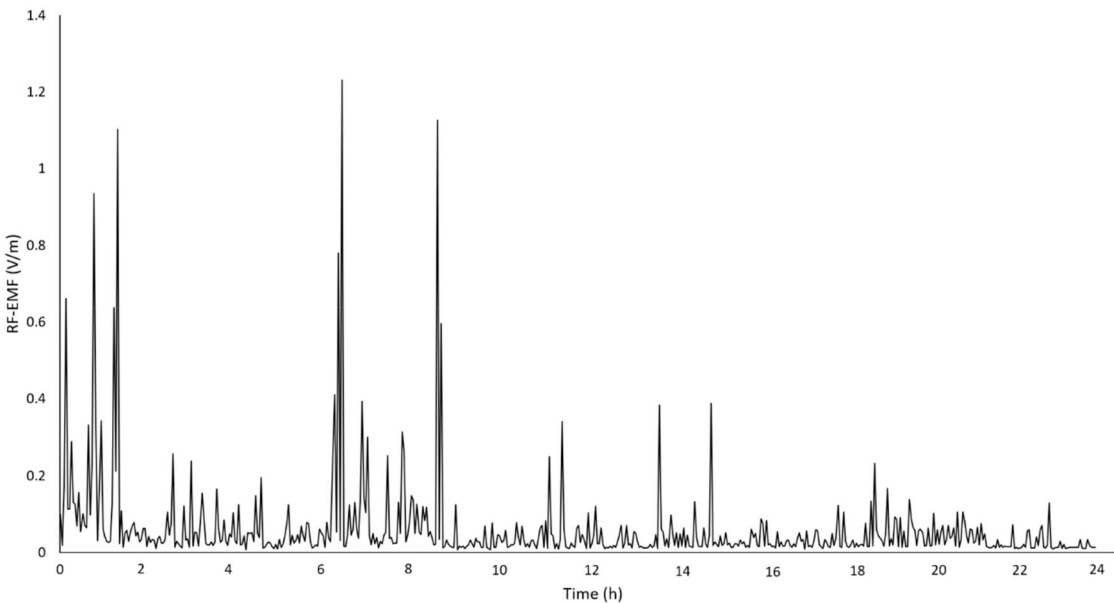


Figure 1. An example of continuous RF levels over one typical day (extraction of 24 h data from the same infant as in Besset et al. 2021).

(Deltamed LTM Brainbox 1166DC, Natus Medical Incorporated, Pleasanton, CA).

Sleep states were scored (always by the same operator) visually in 30-second epochs as recommended by the Pediatric Task Force (Grigg-Damberger 2016). Sleep states were differentiated on the basis of the concordance between EEG and EOG signals. As necessary, breathing and cardiac parameters were superimposed on these measurements to help define sleep stages. Infants were also video-recorded throughout the night to help excluding nursing interventions and feeding episodes from the analysis. Wakefulness was defined as a state in which the infant's eyes were open (whether scanning the environment or not) and when frequent body movements occurred. REM sleep was defined as continuous EEG activity with eye movements, while non-rapid eye movement (NREM) sleep was defined as discontinuous EEG activity without eye movements. Periods that did not meet the criteria for either REM or NREM were defined as indeterminate sleep (IS).

The following criteria were taken into account: sleep period time (SPT), beginning at the first sleep onset and ending at the last awakening; the frequency, mean duration, and proportion of wakefulness after sleep onset (WASO), expressed as a percentage of the SPT; total sleep time (TST), defined as the difference between SPT and WASO durations; and the frequency, mean duration, and proportion of the different sleep states, expressed as a percentage of TST.

Statistics

Statistics were computed using Statview software (version 5.0; SAS Institute, Cary, NC). Normality of data distribution was checked by applying the Kolmogorov-Smirnov test. Since most of the data did not follow a normal distribution, non-parametric tests were used. The influence of chronic RF-EMF exposure on sleep was assessed by using Spearman's correlations to test the relationship between all sleep parameters and RF-EMF levels calculated over the 3-week period (median and P99).

To ensure that the effects were actually due to the chronic RF-EMF exposure (and not to acute RF-EMF exposure *per se*), we performed the same statistical analyses with the median and P99 RF-EMF levels extracted from the polysomnographic period only. Due to multiple testing (76 correlations in total: 19 sleep parameters correlated to both the median and P99 RF levels over both the 3-week and polysomnographic periods), a Bonferroni correction was applied to calculate the threshold for statistical significance, which was set to $p < .0007$.

Results

Sleep structure parameters extracted from the polysomnographic recordings at 3 weeks postnatal age are shown in Table 2. For the chronic RF-EMF exposure over the 3-week period, average exposure of the 29 infants was 0.03 ± 0.01 V/m at the median level and 0.24 ± 0.11 V/m at

Table 2. Sleep data.

SPT (min)	672.5 (515–713)
TST (min)	605.5 (312–699)
Sleep efficiency (% SPT)	89.3 (51–100)
WASO (min)	75.5 (0–300)
WASO frequency (h^{-1})	0.6 (0–1)
WASO mean duration (min)	12.3 (2–76)
WASO (% SPT)	10.7 (0–49)
Sleep state change frequency (h^{-1})	4.2 (2–6)
REM frequency (h^{-1})	1.1 (0.3–2)
NREM frequency (h^{-1})	0.8 (0.5–1)
IS frequency (h^{-1})	1.7 (1–2)
REM mean duration (min)	21.9 (10–38)
NREM mean duration (min)	15.5 (5–29)
IS mean duration (min)	9.5 (6–25)
REM (% TST)	41.1 (20–65)
NREM (% TST)	26.5 (11–36)
IS (% TST)	34.9 (17–57)
Longest REM period	40 (29–72.5)
Longest NREM period	28.5 (17.5–64)

SPT: sleep period time; TST: total sleep time; WASO: wakefulness after sleep onset; REM: rapid eye movement sleep; NREM: non-rapid eye movement sleep; IS: indeterminate sleep.

Values are quoted as the median (range).

Table 3. Correlations between sleep data and chronic RF-EMF exposure levels over the 3-week period.

Sleep structure parameters	RF-EMF median level		RF-EMF P99 level	
	Correlation (ρ)	p Value	Correlation (ρ)	p Value
SPT (min)	0.039	.8358	–0.083	.6596
TST (min)	0.113	.5488	0.044	.8145
Sleep efficiency (% SPT)	0.082	.6648	0.076	.6857
WASO (min)	–0.093	.6209	–0.076	.6881
WASO frequency (h^{-1})	–0.155	.4127	0.09	.6347
WASO mean duration (min)	0.001	.9957	–0.278	.1558
WASO (% SPT)	–0.077	.6828	–0.076	.6867
Sleep state change frequency (h^{-1})	0.098	.6058	0.45	.0172
REM frequency (h^{-1})	0.181	.3394	0.44	.0198
NREM frequency (h^{-1})	–0.016	.9315	0.033	.8593
IS frequency (h^{-1})	0.131	.4889	0.407	.0311
REM mean duration (min)	–0.156	.4094	–0.412	.0292
NREM mean duration (min)	–0.205	.2791	–0.0278	.1558
IS mean duration (min)	0.437	.0208	–0.102	.5895
REM (% TST)	–0.297	.1166	0.116	.5384
NREM (% TST)	–0.157	.4067	–0.23	.2235
IS (% TST)	0.457	.0156	0.044	.8165
Longest REM period	–0.477	.0116	–0.249	.1883
Longest NREM period	–0.100	.5953	–0.143	.4485

ρ : Spearman's rank correlation; NS: non-significant; see Table 2 for other abbreviations.

None of the correlations was significant after Bonferroni's correction.

the P99 level. For the acute RF-EMF exposure during the polysomnographic period only, average exposure was 0.03 ± 0.02 V/m at the median level and 0.12 ± 0.09 V/m at the P99 level.

Results of the correlations between sleep structure parameters and both chronic and acute RF-EMF exposure levels are shown in Tables 3 and 4, respectively.

For the chronic RF-EMF exposure, the median level was associated positively with both IS mean duration and proportion, and negatively with the longest REM period. At the highest exposure levels, P99 was associated negatively with the mean duration of REM episodes, and positively with the sleep state change frequency. The latter result was mainly due to an increase in the frequencies of REM and IS episodes with the increase in P99.

Table 4. Correlations between sleep data and acute RF-EMF exposure levels during the polysomnographic period.

Sleep structure parameters	RF-EMF median level		RF-EMF P99 level	
	Correlation (ρ)	<i>p</i> Value	Correlation (ρ)	<i>p</i> Value
SPT (min)	-0.539	.0044	-0.153	.4191
TST (min)	-0.307	.1045	-0.170	.3685
Sleep efficiency (% SPT)	-0.083	.6596	-0.139	.4611
WASO (min)	0.048	.8014	0.100	.5949
WASO frequency (h^{-1})	0.099	.6003	0.403	.0329
WASO mean duration (min)	0.111	.5726	-0.215	.2732
WASO (% SPT)	0.084	.6577	0.140	.4603
Sleep state change frequency (h^{-1})	-0.050	.7908	0.263	.1639
REM frequency (h^{-1})	0.152	.4202	0.250	.1863
NREM frequency (h^{-1})	-0.342	.0705	-0.069	.7152
IS frequency (h^{-1})	-0.201	.2867	0.080	.6709
REM mean duration (min)	-0.028	.8809	-0.306	.1051
NREM mean duration (min)	0.296	.1175	-0.089	.6366
IS mean duration (min)	-0.136	.4731	0.212	.2623
REM (% TST)	0.106	.5756	-0.169	.3713
NREM (% TST)	-0.092	.6283	-0.131	.4881
IS (% TST)	-0.163	.3872	0.340	.0721
Longest REM period	0.115	.5415	-0.145	.4431
Longest NREM period	0.193	.3075	-0.200	.2908

ρ : Spearman's rank correlation; NS: non-significant; see Table 2 for other abbreviations.

None of the correlations was significant after Bonferroni's correction.

During acute RF-EMF exposure, an increase in the median level was significantly associated with a decrease in SPT. On the other side, a positive association was found between P99 and WASO frequency.

Discussion

Preterm neonates are chronically exposed to RF-EMF (Besset et al. 2021) at levels, which do not place them at risk (ICNIRP 2020) and, despite subtle changes in some sleep parameters, the present study shows that such exposure does not disrupt their sleep structure.

Here, sleep data were similar to those found in previous sleep studies (Stéphan-Blanchard et al. 2008). In particular, sleep parameters were highly correlated with postmenstrual age. This is an usual effect observed in the literature (Bourel-Ponchel et al. 2021). However, postmenstrual age was not correlated with chronic or acute RF-EMF exposure levels (data not shown) and cannot therefore be considered as an independent cofactor in RF-EMF effects.

At median levels, an observed trend (not significant after Bonferroni's correction) was an increase in IS with RF-EMF exposure. At the highest exposure levels in our study (P99), an increase in RF-EMF levels did not alter sleep structure but seemed to increase sleep fragmentation (greater sleep state change frequency not significant after Bonferroni's correction). More consistently, our results suggest that the RF-EMF impact on sleep was dependent on exposure duration rather than exposure levels as regards to the differences observed between chronic and acute exposures.

An increase in IS with chronic RF-EMF exposure might have neurophysiological significance. Indeed, it was shown that a greater preponderance of organized, well-defined sleep states, i.e. NREM or REM, is one of the most significant aspects of early brain maturation (Curzi-Dascalova et al.

1993; Mirmiran and Ariagno 2009). Therefore, IS may indirectly be a marker of low brain maturation. We also observed that chronic RF-EMF exposure could potentially result in sleep fragmentation, with an increase in the occurrence of IS and REM episodes. This poorer sleep stability may also suggest a lower brain maturation in the most exposed infants. Indeed, it is commonly established (Lehtonen and Martin 2004) that sleep continuity increase is a marker of brain maturation, with concomitant increase in the sleep stage duration and decrease in the sleep stage frequency. Given the crucial role of sleep for growth and brain development in preterm neonates, the effects observed here suggest that exposed infants may have more difficulty to organize and maintain their sleep, probably as a consequence of comparative immaturity.

On the other hand, analysis of the influence of RF-EMF exposure during the polysomnographic period only showed no global influence on sleep. Acute exposure levels were either not statistically different for the median levels ($p = .8095$) or significantly lower for P99 ($p = .0001$) as compared to the whole 3-week recordings. Thus, it cannot be ruled out that this may have contributed to the absence of effect in the highest acute exposure levels. Acute RF-EMF exposure was nevertheless associated with a decrease in the SPT at median levels and an increase in the frequency of wakefulness episodes at the highest levels (not significant after Bonferroni's correction). However, inherent to clinical studies, infants with the lowest SPTs in our study were those for whom the most human intervention (nursing staff, parents) periods had to be excluded from sleep analysis. Taken as a whole, our findings indicate that, at the low levels found in our study, acute RF-EMF exposure does not alter sleep patterns in preterm neonates.

These results are consistent with current knowledge on sleep neurophysiological mechanisms. Sleep is a very integrated process, known for its plasticity and ability to reorganize in response to stimulation provided by the post-natal environment (Peirano et al. 2003). Thus, very low, acute RF-EMF environmental levels may not be high enough to show sleep disturbances. Also, the impact on sleep caused by chronic exposure may have masked some of the acute effects. Current studies, reporting either sub-chronic (Danker-Hopfe et al. 2016; three nights of RF-EMF exposure) and acute (Loughran et al. 2005; 30 minutes of RF-EMF exposure) exposure in adult humans, or chronic exposure in rats (Pelletier et al. 2013, 5 weeks of RF-EMF exposure) were always performed at levels at least 100 times higher than those found in our study. Strikingly, all these studies pointed out in the same direction: a sleep fragmentation and an increase in REM when exposed to RF-EMF whatever the exposure modalities (acute or chronic). As more and more are mentioned in the most recent neurodevelopmental paradigms, our study is the first to highlight that the population, period and duration of exposure to environmental agents are key factors in the appearance of effects. Although no effect on sleep structure was found, our data suggest that some indicators of sleep quality were sensitive to RF exposure, even at very low levels. Our study was not designed to

evaluate the effects of low-frequency EMFs; however, it could be interesting to study their specific or cumulative influence with RF on the structure of sleep given the results obtained previously by other authors (Bellieni et al. 2008, 2012).

In conclusion, chronic exposure to low RF-EMF levels did not disrupt sleep structure despite some discrete alterations such as increased IS and sleep fragmentation. These changes may indicate a lower brain maturation in the most exposed infants. Although no concerns about infants' health are currently relevant, further studies are needed to confirm the present results and examine mid- to long-term, sleep-related cardiorespiratory (Chardon et al. 2004) and neurodevelopmental effects (Constantinou et al. 2005).

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Author contributions

Dimitri Besset: methodology, software, formal analysis, investigation, and writing – original draft; Brahim Selmaoui: methodology, validation, and writing – review and editing; Stéphane Delanaud: methodology, software resources, and writing – review and editing; Lisa Bessarion: investigation; Karen Chardon: methodology, validation, data curation, and writing; René de Seze: methodology, software, validation, and writing – review and editing; André Leke: methodology and writing – review and editing; Erwan Stephan-Blanchard: conceptualization, methodology, validation, data curation – writing – review and editing, and project administration.

Ethical approval

The study was approved by an independent institutional review board (*CPP Sud-Est III*; reference: RCB18.10.12.31915) and was performed in accordance with the principles of the Declaration of Helsinki Participation of human subjects and did not occur until informed consent was obtained.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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