J. Sleep Res. (1999) **8**, 77–81

A 50-Hz electromagnetic field impairs sleep

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Accepted in revised form 15 December 1998; received 19 November 1998

SUMMARY In view of reports of health problems induced by low frequency (50–60Hz) electromagnetic fields (EMF), we carried out a study in 18 healthy subjects, comparing sleep with and without exposure to a 50Hz/1μTesla electrical field. We found that the EMF condition was associated with reduced: total sleep time (TST), sleep efficiency, stages 3+4 slow wave sleep (SWS), and slow wave activity (SWA). Circulating melatonin, growth hormone, prolactin, testosterone or cortisol were not affected. The results suggest that commonly occurring low frequency electromagnetic fields may interfere with sleep.

KEYWORDS slow wave sleep, TST, melatonin, EMF

INTRODUCTION

Exposure to low frequency (50–60Hz) electromagnetic fields (EMF) has been reported to be associated with health complaints (Knave 1994) and particularly with dermatological problems (Feldman *et al.* 1985; Berg 1988; Bergqvist and Wahlberg 1994), and with depression and fatigue (Knave *et al.* 1985; Dowson *et al.* 1988; Poole *et al.* 1993; McMahan and Meyer 1994). Also, the low frequency spectrum of the EEG (Lyskov *et al.* 1993) is suppressed, as is the pineal hormone melatonin in animals (Reiter *et al.* 1988; Lombrozo *et al.* 1996) and, possibly, in humans (Arnetz and Berg 1996; Graham *et al.* 1996).

The available studies have mainly focused on exposure during the waking hours (at work), but there is a possibility that also the exposure during sleep may be of interest. One reason is that sleep constitutes one-third of the 24 h day and that the stationary nature of sleep will involve a constant exposure to a particular EMF intensity, whereas exposure during wakefulness may vary greatly over time, depending on the movement pattern of the individual (Feychting and Ahlbom 1993; Nissen and Paulsson 1995). No data on EMF and physiological sleep are available, however, but some of the complaints, for example fatigue, could be related to disturbed sleep. In addition, the EMF-induced suppression of melatonin may be related to disturbed sleep since melatonin appears to be a sleep facilitator (Arendt *et al.* 1995).

The purpose of the present study was to investigate the effects on sleep of one night's exposure to 1μTesla – a relatively high, but commonly occurring level (Feychting and Ahlbom 1993). Apart from traditional sleep polysomnography, it was also of interest to study the effects on sleep related hormones such as melatonin, human growth hormone (GH), adrenocorticotropic hormone (ACTH), cortisol, and prolactin (Sassin *et al.* 1969; Weitzman *et al.* 1983; Gronfier *et al.* 1996; von Treuer *et al.* 1996).

METHODS

Eighteen healthy, non-medicating, good sleepers (8 females, age range 18–50 years) participated in the experiment, approved by the ethical committee of the Karolinska Institute. Their average habitual sleep length was 7.54 ± 0.23 h and habitual bedtime was 23.47 ± 0.19 h. After a night of habituation, the subjects were exposed 3–5 days later to a night with a 1mTesla (μT) EMF field on or off, in a balanced order with one week in between. The subjects and experimenters were blind to the order of conditions.

The experiment took place in a special purpose wooden cabin, without uncontrolled interference from light, noise, or EMFs. The subjects were studied two at a time and slept in beds in the same large room, 10 meters apart and with a screen separating the two areas. The magnetic fields were measured using a three-axis magnetometer with a frequency response

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5 Hz-2 kHz and a resolution of 1 nT (Combinova MFM10). The average background in the beds was < 0.02μT with 0.025μT as the maximum of 25 different measurements. This is very low compared to averages of daily city living, which usually reach 0.2μT (Feychting and Ahlbom 1993), even if higher transients of several μT may be seen.

A vertical exposure field was generated by two rectangular coils (1.8×3 m) with 5 turns, each situated below the floor 0.7 m beneath each bed and 10 m apart. Thus no interaction between the two coils could occur. The coils were driven in series by a 50-Hz AC current from a low voltage transformer connected to an ordinary 230 V outlet. The current was adjusted with a variable power resistor to yield an exposure field of 1 μT. The current could be activated through a switch which was housed in a locked box together with the transformer and the resistor. The average of 40 measurements taken in 10 different positions in each bed during a one hour period was 1.02 μT (SD = 0.04 μT). The 1 μT level was chosen for the experiment since it would represent a commonly occurring high level. The generated field did not cause any sound or any other manifestation that could be perceived by the subjects or experimenters.

Overhead fluorescent lighting yielded 170 lux at the level of the bed while the lights were on. With lights turned off the level was less than 0.1 lux.

At 18.00 h, before each session, the locked box was opened by a technician not involved in any other part of the study, and the switch was set in a predetermined position (according to a previously made random selection), unknown to the experimenter and the subjects. The on-off code was not broken until all the data had been collected and analysed and were ready for entry into computer programs for statistical analysis.

After a habituation night, subjects arrived for the experiment at 22.00 h, had an 18 gauge catheter (Venflon 2) inserted into a vein of the right arm, and electrodes for conventional sleep recording applied (C3-A2, submental bipolar, outer canthi of each eye) (Rechtschaffen and Kales 1968). Thereafter the subjects would get prepared for going to bed and spent any remaining time reading.

Sleep was recorded digitally on a solid state, battery-powered unit (Embla), placed 90 cm behind the head of the subject. No EMF emission from the equipment was present at the head of the subject. Neither was there any direct effect of the EMF on the recording. The latter was established in a series of tests with the field alternately on and off in 1-min intervals. For the test was used two electrodes placed on the pillow and connected via a 10 kohm resistor. The leads were slightly curved, with a distance of 20–30 cm. The spectral analysis of the recorded data showed that the spectral power density in the 50Hz, 25Hz, the delta, theta, alpha, sigma or theta bands were the same, and within the noise level, for the 'on' and 'off' conditions. For comparison, the absolute delta power density level for both conditions corresponded to $\approx 0.1\%$ of the mean of the power density of the delta band during NREM sleep.

Sleep took place between 24.00 hours and 08.00 hours. Blood samples were drawn at: 23.00 h, bedtime, 02.30 h, 05.00, and 08.00 hours (on awakening). The catheter was kept patent with injections of heparinized saline solution after each sample. Blood was centrifuged immediately after sampling and the plasma was frozen until analysed with routine methods of the Department of Clinical Chemistry at Karolinska Institute. Prolactin, growth hormone and corisol were measured using kits and instruments from Wallace (DELPHIA, Finland – CV: 7%, 5%, an 5%, respectively) ACTH was analysed with an IRMA method (CV 7%) and melatonin with a RIA method (CV 6%). Calibrators were those of the kits.

Five minutes after awakening, the Karolinska Sleep Diary (KDS) (Åkerstedt *et al*. 1994) was completed. The items were scored from 1 to 5, where '5' indicated highest quality or greatest 'ease', and included Sleep quality, Sleep depth, Undisturbed sleep, Ease of falling asleep, and Ease awakening.

The sleep EEGs were scored conventionally (Rechtschaffen and Kales 1968), as well as subjected to a Fast Fourier Transform, with a sampling frequency of 64 Hz, a sampling epoch of 4 s, and a band pass filter of 0.35–70 Hz. Four-second epochs with wakefulness or artefacts were removed from the analysis after visual inspection. The resulting spectral power was integrated across the delta band (0.5–4.5 Hz). One minute values were averaged across all non-REM cycles to provide Slow Wave Activity (SWA) – the 'EMF off' condition was set to 100%.

The difference between the two conditions was tested through one-tailed *t*-tests (the hypothesis was directed towards EMF causing impaired sleep), using a 5% significance level. Sleep latency and REM latency were subjected to a logarithmic transformation, whereas SWA was subjected to a square root transformation due to the very high absolute values which caused a marked skewness in the data. Two subjects had to be removed from the sleep analysis because of deviating (from reported habitual sleep) values for the baseline night (compared to their self-reported habitual sleep length). A two-way repeated measures ANOVA was used to test for changes across the night, using 'Condition' and 'Time of night' as factors. Results were corrected for lack of compound symmetry (large differences between values in the variance-covariance matrix for the time points) by means of the Huyhn-Feldt epsilon coefficient. A similar analysis was carried out to compare the conditions with respect to development across hours of sleep for the significant polysomnographical parameters.

RESULTS

The condition with the EMF 'on' showed significantly less/lower: TST, sleep efficiency, SWS, SWA and subjective depth of sleep (Table 1).

To analyse whether the order of conditions had any effect, measures with significant differences between conditions were subjected to a two-factor ANOVA with 'order' as a group variable (see Fig. 1). Thus, the *F*-values of interest were those of the interaction effect (group by condition, d.f. = 1,15). TST for the order 'EMF off-EMF on' was 418 ± 12 in the 'off' condition and 404 ± 11 in the 'on' condition, whereas for the order 'EMF

Table 1 Mean ± SE for sleep variables with EMF 'off' and 'on' (t-test)

	Off *Mean ± SE*	*On* *Mean ± SE*	*P* =
Total Sleep Time	424 ± 9	407 ± 11	0.035
Sleep Efficiency	0.86 ± 0.02	0.82 ± 0.02	0.050
Awakenings	1.34 ± 0.03	2.41 ± 0.04	0.065
Sleep Latency	18 ± 4	22 ± 6	0.290
SWS Latency	12 ± 1	14 ± 2	0.200
REM Latency	81 ± 9	80 ± 9	0.440
Stage 1	8 ± 2	10 ± 1	0.155
Stage 2	219 ± 10	211 ± 10	0.095
SWS	97 ± 4	82 ± 6	0.005
SWA%	100	80 ± 9	0.018
REM	107 ± 7	104 ± 6	0.335
Stage Wake + Movem	45 ± 9	54 ± 8	0.100
Rated Ease of falling asleep	4.1 ± 0.2	4.2 ± 0.2	0.150
Rated Ease of awakening	3.6 ± 0.2	3.8 ± 0.2	0.115
Rated Sleep quality	3.7 ± 0.2	4.0 ± 0.2	0.085
Rated Sleep depth	3.9 ± 0.2	3.4 ± 0.2	0.005
Rated Undisturbed sleep	3.2 ± 0.2	3.3 ± 0.2	0.200

d.f. = 15; '*P*' indicated for one-tailed *t*-test; all values are given in minutes except for sleep efficiency (prop), SWA (%) and subjective ratings (units).

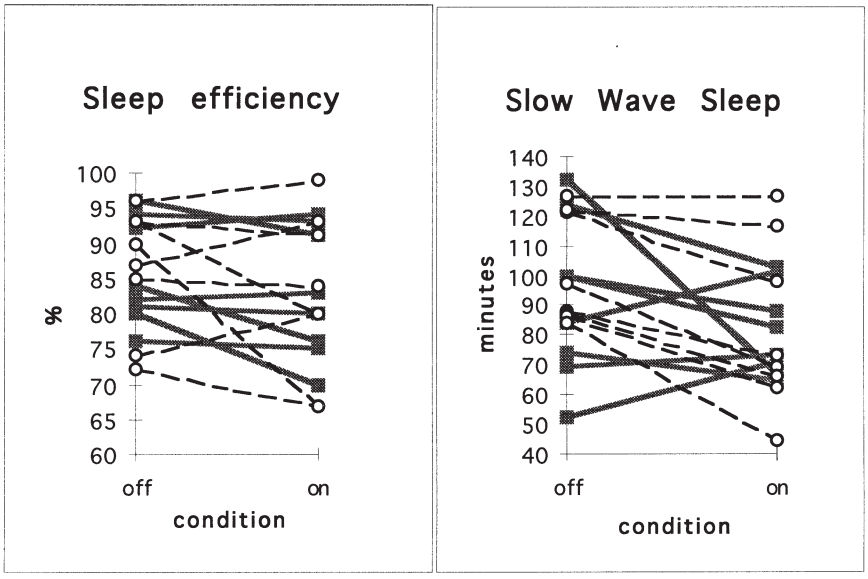

Figure 1. Sleep efficiency and SWS for each individual with the EMF 'off' and 'on', respectively. Solid lines indicate subjects undergoing the 'off-on' order, and dashed lines those with the reverse order.

on-EMF off' they were 428 ± 13 in the 'off' condition and 404 ± 20 in the 'on' condition (Fint = 0.08, ns). SWS for the order 'EMF off-EMF on' was 92 ± 10 with field off and 81 ± 5 with field on; for the reverse order it was 102 ± 7 with the field 'off' and 82 ± 10 with field 'on' (Fint = 0.5, ns). Sleep efficiency for the order 'EMF off-EMF on' was 0.86 ± 0.03 in the 'off' condition and 0.83 ± 0.03 in the 'on' condition; for those who received the reverse order it was 0.86 ± 0.03 in the 'off' condition and 0.80 ± 0.04 in the 'on' condition (Fint = 0.05, ns). Rated Sleep depth for the order 'EMF off-EMF on' was 3.8 ± 0.2 in the 'off' condition and 3.5 ± 0.2 in the 'on' condition. For those who received the reverse order it was 3.9 ± 0.03 in the 'off' condition and 3.3 ± 0.04 in the 'on' condition (Fint = 0.4, ns).

A repeated measures ANOVA was used to analyse whether there was any difference in the change across time for the significant sleep variables SWS and TST (Fig. 2). Sleep efficiency would show the same result as TST if analysed from sleep onset and SWA is not well-suited for this type of analysis by the hour. Three subjects had to be excluded from the analysis because of insufficient data to fill all 8 h slots. Results for SWS showed a significant main effect of condition ($F = 9.7$, d.f. = 1/12, $P = 0.009$) and 'hour of sleep' ($F = 23.8$, d.f. = 7/84, $P =$

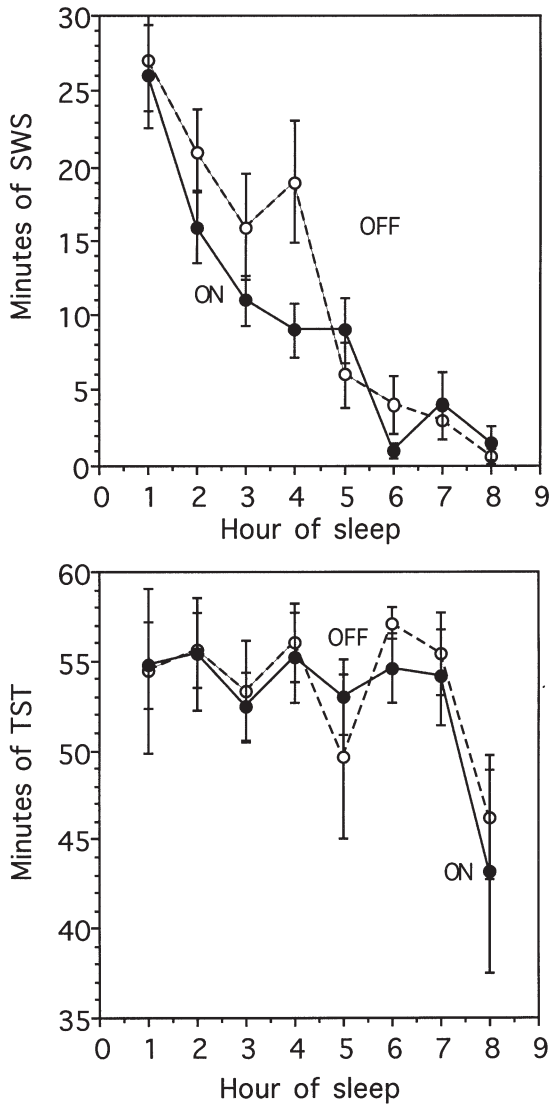

Figure 2. Hourly mean ± se for SWS and TST (starting at sleep onset) across the night, with EMF 'off' and 'on', respectively.

0.0001, epsilon = 0.87) but no significant 'time condition' interaction ($F = 1.64$, d.f. = 7/84, $P = 0.14$, epsilon = 0.98). Still, the figure shows that the effect of condition was present during the first hours only. TST per hour showed a significant effect of hour only ($F = 3.5$, d.f. = 7/84, $P = 0.006$, epsilon = 0.80). The values for condition and interaction was $F = 0.2$ (d.f. = 1/12, $P = 0.70$) and $F = 0.2$ (7/84, $P = 0.93$), respectively.

All plasma hormone levels showed a significant variation across the night, but no significant difference between conditions, nor any significant interaction (Table 2).

DISCUSSION

Clearly, sleep was affected by the low frequency EMF. The reductions of TST and sleep efficiency suggest a slight disturbance. So does the reduction of SWS and SWA, which are often assumed to reflect recuperation (Horne 1988), and which are extremely responsive to time awake/sleep loss (Webb and Agnew 1971; Borbély *et al.* 1981), as well as related to subjective sleep quality. Among the subjective variables only 'depth of sleep' showed a significant reduction, which, however, is in line with the reduction of SWS.

Considering that the present experiment is the first of its kind, and that the issue of EMF effects is sensitive, one needs to carefully consider possible confounders. One such influence might be a failure to 'blind' experimenters and subjects. This seems possible to rule out, however, since the order of the conditions was known only to a technician not involved in the study. Nor was there any sound or other emissions from the field that could be perceived by the experimenters or subjects. Another possible confounder is order effects, but such an influence was ruled out through the statistical analysis of order effects. A third possibility of confounding is direct effects of the field on the recording equipment. This was tested and ruled out – no influence at all was seen. In addition, the effect on SWS was present only during the first hours of sleep, when SWS is usually increased – any direct effects should have been present for the duration of the exposure. Thus, we feel confident in concluding that the effects must have been caused by the EMF influences on the sleep process.

Table 2 Mean ± SE and ANOVA results for plasma hormone levels at five points in time

	23.00	*24.00*	*02.30*	*05.00*	*08.00*	*F time*	*F cond*	*Ft*c*
Melatonin off	34 ± 8	53 ± 8	110 ± 40	60 ± 11	28 ± 7			
Melatonin on	25 ± 7	36 ± 7	67 ± 8	55 ± 8	35 ± 7	5.7*	1.5	0.8
GH off	1.6 ± 0.9	1.3 ± 0.4	2.0 ± 0.3	0.6 ± 0.1	0.3 ± 0.1			
GH on	1.5 ± 0.6	2.5 ± 0.6	1.2 ± 0.2	0.6 ± 0.1	0.3 ± 0.1	6.0**	0.6	1.6
Cortisol off	105 ± 15	102 ± 24	70 ± 15.5	184 ± 20	357 ± 20			
Cortisol on	103 ± 11	114 ± 24	108 ± 24	209 ± 24	365 ± 18	63.1***	3.2	0.5
ACTH off	1.8 ± 0.4	1.3 ± 0.1	1.7 ± 0.3	3.1 ± 0.5	5.5 ± 2.5			
ACTH on	1.6 ± 0.3	1.8 ± 0.7	1.2 ± 0.1	3.0 ± 0.4	4.2 ± 0.3	22.9***	3.6	2.2
Prolactin off	5.6 ± 0.5	5.9 ± 1.1	9.6 ± 1.2	9.0 ± 0.9	12 ± 1.5			
Prolactin on	5.9 ± 0.7	4.6 ± 0.4	9.3 ± 0.7	7.6 ± 0.7	11 ± 1.2	19.9***	1.3	0.4

d.f. = 1/17 for condition, 4/68 for time, and 4/68 for interaction; *F*-values are given after Huyhn-Feldt correction; *P*-values: * < 0.05, ** < 0.01, *** < 0.001. Melatonin and ACTH levels given in pmol/L, cortisol in nmol/L, GH and prolactin in μg/L.

It should be emphasized that, even if significant effects were observed, the absolute levels are still within the range of normal sleep values (Williams *et al*. 1974), and far from clinical significance. Conversley, the present study used healthy subjects, only one night of exposure, and rather moderate intensity. However, patient groups might be more sensitive and it is conceivable that increased intensity of the field or of duration of exposure might yield larger effects. This remains to be demonstrated.

With regard to the hormone parameters, all showed the expected behaviour during sleep, but no effects of the experimental condition. Again, however, intensity, repetition of exposure, and individual sensitivity might alter this.

It is concluded that low frequency EMF may interfere with sleep, but that the clinical implications will have to await further work with different levels of exposure and with patient groups.

ACKNOWLEDGEMENTS

This study was supported by the Swedish Work Environment Fund. The assistance of Mikael Westerlund and Gisela Hellgren is gratefully acknowledged.

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