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Mobile phone 'talk-mode' signal delays EEG-determined sleep onset

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Abstract

Mobile phones signals are pulse-modulated microwaves, and EEG studies suggest that the extremely low-frequency (ELF) pulse modulation has sleep effects. However, 'talk', 'listen' and 'standby' modes differ in the ELF (2, 8, and 217 Hz) spectral components and specific absorption rates, but no sleep study has differentiated these modes. We used a GSM900 mobile phone controlled by a base-station simulator and a test SIM card to simulate these three specific modes, transmitted at 12.5% (23 dBm) of maximum power. At weekly intervals, 10 healthy young adults, sleep restricted to 6 h, were randomly and single-blind exposed to one of: talk, listen, standby and sham (nil signal) modes, for 30 min, at 13:30 h, whilst lying in a sound-proof, lit bedroom, with a thermally insulated silent phone beside the right ear. Bipolar EEGs were recorded continuously, and subjective ratings of sleepiness obtained every 3 min (before, during and after exposure). After exposure the phone and base-station were switched off, the bedroom darkened, and a 90 min sleep opportunity followed. We report on sleep onset using: (i) visually scored latency to onset of stage 2 sleep, (ii) EEG power spectral analysis. There was no condition effect for subjective sleepiness. Post-exposure, sleep latency after talk mode was markedly and significantly delayed beyond listen and sham modes. This condition effect over time was also quite evident in 1–4 Hz EEG frontal power, which is a frequency range particularly sensitive to sleep onset. It is possible that 2, 8, 217 Hz modulation may differentially affect sleep onset.

Keywords: Sleep; EEG; Microwaves; Mobile phone

Mobile phone signals operating at 900 MHz of the global system for mobile communications (GSM900) include extremely low-frequency (ELF) pulse-modulated fields. These are associated with 'time division multiple access' (TDMA) and/or the 'discontinuous transmission' (DTX) technologies implemented during 'talk-mode' and 'listen-mode' transmission. The TDMA technology allows more phones simultaneously to communicate with a base-station, resulting in a basic repetition frequency of 217 Hz, with every 26 pulses being grouped together causing another ELF pulsing at around 8 Hz (by definition, the 26th pulse is idle). The latter, unlike the 217 Hz pulsing, is unaffected by the call density and is a permanent feature of GSM signals. In GSM phones utilizing DTX technology (for battery power saving), there is an additional 2 Hz pulsing during 'listen mode.' If the phone is just switched-on for registration with the base-station, without an active call ('standby mode'), the carrier frequency pulses less periodically, at <2 Hz. In addition, these pulse modulations affect a phone's power output, causing the three modes to differ in the amount of radiation absorbed by adjacent tissue (specific absorption rates – SARs, talk > listen > standby ≈ 0 mW/g, all averaged to 10 g of tissues). In sum, the 'talk', 'listen' and 'standby' signals of GSM900 mobile phones differ in their ELF spectral composition and SAR rates (cf. refs [17,19] for detailed review). These factors must be taken into account when comparing experimental results obtained from simulated signals in the laboratory.

With respect to brain function reflected in the electroencephalogram (EEG), and especially in relation to sleep or rest, there is accumulating evidence that ELF pulse modulation has some influence. However, it is not known whether the different ELF composition of talk and listen modes will have different sleep effects. This is because most studies appearing to look at GSM 'talk-mode' signals (900 MHz with 217 Hz modulation) have not considered the permanent ELF modulation at 8 Hz (e.g. refs [4–6,20,21,26,25]). Furthermore, those studies that did include this 8 Hz component, only focussed on listen mode (with the extra 2 Hz modulation) [3,13–15]. These

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Table 1
Summary of the ELF characteristics of the GSM signals used by other relevant EEG studies, in comparison with our study

Our study	ELF components	Other studies
Talk	8, 217 Hz	
Listen	2, 8, 217 Hz	Borbély et al. [3], Huber et al. [13] ('base-station-like'), Huber et al. [15] ('handset-like'), Huber et al. [14] (both 'base-station-like' and 'handset-like')
Standby	1–32 Hz	D'costa et al. [6]
Not applicable	Only 217 Hz	Mann and Röschke [21], Wagner et al. [26] (repeating Mann and Röschke [21], with more participants), Wagner et al. [25] (repeating Mann and Röschke [21], with greater power flux density from 0.5 to 50 W/m^2), Curcio et al. [5], Loughran et al. [20], Croft et al. [4], D'costa et al. [6]

See text for details.

differences are summarised in Table 1. Evidence pointing to ELF pulse modulation inducing neurophysiological changes, has been shown, for example, in recent brain imaging and EEG studies by Huber et al. [15,16]. Their earlier study [15] found that it was the ELF pulse-modulation of the 900 MHz microwave, rather than the continuously emitted 900 MHz itself, that subsequently increased waking alpha (11–11.25 Hz) and sleep spindle (12.25–13.5 Hz) EEG activities. The relative waking regional cerebral blood flow (rCBF) imaging after this exposure, compared to sham exposure, showed the dorsolateral prefrontal cortex to have been particularly affected by this pulse modulation, which led the authors to speculate that this region was a focus for the ELF pulse modulation. Their later investigation [16] revealed that the stronger was this ELF modulation, the greater was the waking rCBF effect.

Given that there may be different effects of ELF components on sleep, no sleep study has differentiated talk, listen and standby modes systematically, nor done so in relation to the potential effects on the EEG during process of falling asleep or the propensity to fall asleep. This unexplored area formed the basis of our study.

Talk, listen and standby modes were generated by a GSM900 Nokia 6310e mobile phone having a test-SIM card and controlled by a HP8922M GSM900 base-station simulator, located 1.5 m away in another room, and transmitting at about only 12.5% (23 dBm) of maximum power. SARs for the three modes were as follows: talk = 0.133 mW/g, listen = 0.015 mW/g, and standby < 0.001 mW/g (all averaged to 10 g of tissue [24]). A summary of the GSM ELF pulse modulation for these modes is included in Table 1.

Ten paid participants (healthy, un-medicated, normal-sleeping, right-handed men, mean age: 22 ± 2.7 years, range: 18–28 years) were screened and recruited from students on the campus, having given their written informed consents. They were regular mobile phone users but with an average talk-time less than 1 h/day. They maintained their regular sleep–wake schedule for at least 3 days prior to each trial (monitored by wrist-worn actimeters and personal sleep diaries). Alcohol and caffeine-containing beverages were prohibited at the night before and on the morning of each trial. Their mobile phone use ceased after 22:00 h the evening before trials. Their prior night's sleep was restricted to 6 h (by a delayed bedtime and with their sleep monitored by actimeters).

A fixed afternoon routine for experimentation ensued, beginning with the participant lying on a comfortable bed, in an individual sound-proof and lit bedroom. The experimental phone was harnessed beside the right ear, and any possible heat from the battery or any seemingly inaudible 'hum' was insulated from the ear by a 2 cm thick cotton-wool wadding. At precisely weekly intervals, participants were exposed ('blind' and 'randomly') to one of: talk, listen, standby and sham (nil signal) modes, for 30 min, commencing at 13:30 h, with the exposure orders counterbalanced between participants. Throughout all exposures the phone generated no sounds (the phone's speaker was disabled). Participants remained silent, and fixed their eyes on a wall marker. Bipolar EEGs (F3–C3; F4–C4; C3–P3; C4–P4; P3–O1; P4–O2) were sampled at 100 Hz using Embla 7000 system (Embla^{TM}-Flaga hf. Medical Devices). Band pass filtering of the EEG was at 0.3–50 Hz, with electrode impedance always <5 kΩ. EOGs and EMGs were also obtained according to the established method [22]. Subjective ratings of sleepiness were assessed using the Karolinska Sleepiness Scale (KSS [1]) every 3 min (before, after and during the exposure). After the exposure, the phone and base-station were switched off, the phone removed, the bedroom darkened, and a 90 min sleep opportunity followed with participants being instructed to close their eyes and try to fall asleep. Here, we only report on the period of falling asleep, determined from the EEGs, using two methods:

(i) Visually scored latency to sleep onset from 'lights out' – with sleep onset defined as, 'the first appearance of a consecutive period of stage 2 sleep, lasting for at least 3 min' [22]. This was determined by two independent scorers, 'blind' to the conditions.
(ii) EEG power spectral analysis (FFT routines, epoch: 5 s; sampling rate: 128 Hz; Hanning window, frequency resolution: 0.2 Hz, then averaging across six 5 s epochs, i.e. every 30 s), with emphasis on 1–4 Hz activity, which is indicative of sleepiness and sleep [22] and particularly evident in the left frontal channel (EEG derivative: F3–C3 [27]).

Before all exposures, mean subjective sleepiness [1] was similar for all conditions (score 5 – 'neither alert nor sleepy'), which rose over the 30 min exposure in a similar manner for all conditions, reaching values between levels 7 ('sleepy') or 8 ('sleepy, some effort to stay awake').

However, post-exposure, the visually scored sleep latencies (talk mode: 48.8 ± 7.9 min, listen mode: 22.1 ± 6.1 min, standby mode: 32.9 ± 8.5 min, sham mode: 23.8 ± 4.6 min) revealed a significant condition effect ($F_{[3,27]} = 3.4$, $p = 0.03$, one-

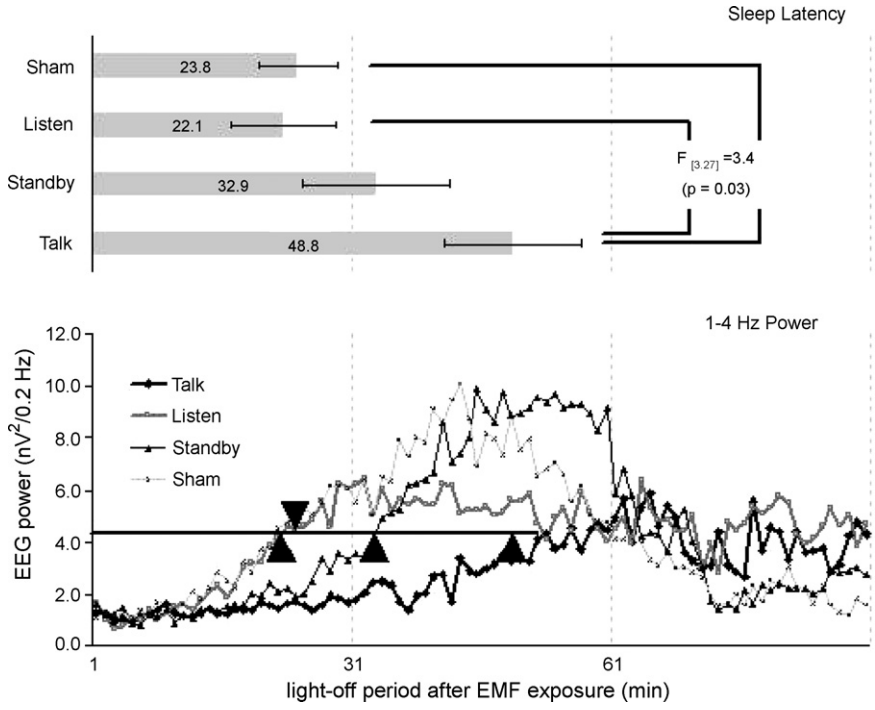

Fig. 1. 90 min sleep EEG recording after different exposure modes. Visually scored sleep-onset latency (mean and S.E.M.) for the four conditions (Upper). Changes in EEG 1–4 Hz power shown for the left frontal (F3–C3) EEG (Lower). The filled triangles superimpose the mean visually scored sleep onsets. A horizontal line can be drawn through these triangles, indicating consistency between the two independent methods of analysis.

way ANOVA for repeated measures). Student–Newman–Keuls ranges tests for multiple comparisons showed that sleep latency following talk mode was significantly delayed beyond listen and sham modes (Fig. 1, upper panel). This differentiation was quite evident with the changes in 1–4 Hz EEG frontal power, especially from the left frontal channel (Fig. 1, lower panel), where a two-way (conditions × time [10 min intervals; 9 levels]) ANOVA for repeated measures showed a significant interaction effect ($F_{[24,216]} = 1.67$, $p < 0.033$). Post hoc comparisons using SPSS Helmert tests showed this EEG 1–4 Hz power to rise significantly ($p < 0.006$ – applying Bonferroni correction) in the second 10 min period after listen and sham exposures, the third period after standby exposure, but for no period after talk exposure.

To our knowledge this is the first study where actual talk, listen and standby modes have been compared in a systematic manner, and separated in relation to sleep onset. Ostensibly, the apparent alerting effect of talk mode is inconsistent with findings from previous studies claiming to look at the same signal (involving GSM900 carrier frequency but with only a 217 Hz modulation), where sleep latency was found to be either shortened [21] or not affected [20,26,25]. However, these studies differ from ours in the ELF composition of the phone signals. Our talk-mode signal was real, having the permanent ELF component at 8 Hz. Rather than assessing sleep onset at a normal bedtime after limited control of circadian confounding and pre-trial sleep, we utilized the natural, early afternoon circadian 'dip', titrated by actimetrically monitored pre-trial sleep restriction, with all exposure sessions starting at the same time. Thereby, we produced a standard amount of sleepiness and controlled for circadian effects before each exposure within participants. These other studies also varied in exposure set-up and duration, the phone's position, power outputs and SAR levels. Thus, our finding with talk mode on sleep onset is unique, and cannot directly be compared with previous sleep studies using only 217 Hz pulsed GSM900 signals. Moreover, we compared talk mode with three other conditions (listen, standby and sham modes) within participants (single-blind) under standardised conditions, and found a specific effect.

With regard to the EEG at sleep onset, we also looked at the time course change of theta (5–7 Hz), alpha (8–10 and 10–12 Hz) and sigma (12–14 Hz) power to see how these tracked the transition to sleep (data not shown). Whilst frontal theta and spindle power did reflect increasing trends at sleep onset, with a pronounced delay of sleep onset after talk mode, compared with delta power the temporal change of these other frequencies was less distinct, largely because of their being of lower power, episodic and noisier at sleep onset. Indeed, within the EEG spectrum that has been extensively studied in the wakefulness–sleep transition, the linear increase of 1–4 Hz power (with anterior–posterior gradients, more prominent at the frontal region) is the most consistent EEG feature of increasing sleepiness from wakefulness through drowsiness to stage 2 sleep [8,27]. Furthermore, studies assessing single-Hz EEG activity from 1–11 Hz during sleep onset reported that only delta power, particularly at 3–4 Hz, best showed this change, as well as from sleep to brief awakening [2,7]. Theta power in the range of 6–7 Hz lacks significant change during this transition period [7,28]. Although alpha power at 10 Hz reflects the transition from waking to sleep, this is not the case for the reverse

transition [2]. Moreover, sleep onset differentially affects broadband alpha power (8–12 Hz, central derivation), which displays a quadratic trend: a progressive decrease during wakefulness, a minimum point during stage 1, and a subsequent increase during stage 2 [7]. Alpha-like activity at frontal, central and occipital brain regions does not always occur concurrently during the first 30 min of sleep onset [10], and the correlation between alpha and delta/theta power (at all derivations) differs between frontal and occipital alpha at sleep onset [10]; all of which creates difficulty in using alpha activity for determining sleep onset. We concur with the literature that the temporal change of delta power is a more sensitive and reliable marker for stage 2 sleep.

Our finding of a nil effect of listen mode compared with the sham condition is similar to the outcome from of Huber et al. [13], where their 30 min GSM900 'base-station-like' signals (sharing the same ELF components with our 'listen mode' at 2, 8, 217 Hz but with eight times more ELF spectral power at 2 and 8 Hz) had no influence on sleep latency in a subsequent 3 h daytime sleep in healthy young men, having had their prior night's sleep restricted to 4 h. To the extent that our ELF characteristics and findings with listen mode seem to reproduce those of Huber et al., and with a similar experimental listen-mode protocol, then we believe that the outcome from our unique incorporation of talk mode, is not a random effect, and thus the difference between talk and listen effects on sleep onset seems to be real.

The actual cause of the significant difference between talk and listen effects on sleep onset is unknown. It might be due to the typical SAR value for talk mode being about nine times higher than that for the listen mode. We cannot exclude this possibility as there are technical problems in trying to equate talk and listen mode in terms of SARs, as SARs are integral to pulse modulations. However, Regel et al. [23] have measured possible dose–response effects on the sleep EEG, by varying the intensity of SARs of the GSM900 mobile phone signals (with ELF components at 2, 8, 217 Hz) and reported nothing of note in this respect. For example, a SAR value of 0.2 W/kg resulted in a sleep-onset latency of 19.4 ± 2.4 min, compared with the similar 20.7 ± 2.8 min for a SAR value of 5 W/kg. It should also be noted that any possible 'far-field' influence of our GSM900 base-station signal can be excluded, as it was switched off during the sleep recording session.

It is unlikely that brain heating effects could be the cause for our findings with talk mode, especially when our relatively low SAR values are considered. Hirata and Shiozawa [12] calculated the SARs and resultant brain temperature rises for 660 exposure conditions (e.g. phone pressed against the ear, flattening it against the head, or thermally insulated from the ear and head, under exposure to GSM carrier frequencies between 900 MHz and 2.45 GHz, with horizontal or vertical polarisation, and 18 different antenna feed points using a dipole, monopole and helical antennae). At 2 W/kg per 10 g of tissue (about 16× our talk-mode value, and at the ICNIRP [18] recommended limit), the predicted worst-case brain temperature rise would be about 0.25 °C. Thus for our SARs, any putative, localised rise in brain temperature would probably be nominal, especially when the naturally rapid heat clearance by blood from the brain is further considered.

For the reasons just described, together with our lower SARs for both talk and listen modes compared with those utilised by Regel et al. [23], we suspect that the significant difference between talk and listen modes on sleep onset has something to do with their respective spectral composition. Both these modes share the same ELF components at 8 Hz and 217 Hz, and it seems that one or both these components (as in talk mode) seem to delay sleep onset. Furthermore, as listen mode does not affect sleep, and contains another ELF component at 2 Hz, then it is possible that the latter component may negate this sleep delaying effect.

Previous studies using 217 Hz pulse-modulated GSM900 signals have produced equivocal findings with sleep onset across laboratories, and even within the same laboratory the results are not replicated when better methodological controls are adopted. For example, Mann and Röschke [21] with their single pulse-modulated ELF component at 217 Hz reported a shortened sleep onset, but this could not be replicated by expanding participant numbers [26] nor by increasing exposure dosimetry [25].

Thus to summarise so far, it might seem that the delayed sleep-onset effect of talk mode is a result of the 8 Hz component alone or the integration of both 8 Hz and 217 Hz pulsing. Whilst this remains a poorly investigated possibility in humans, a review on animal studies suggests that ELF pulse modulations between 8 and 16 Hz may be critical for physiological effects of GSM mobile phone signals (cf. Introduction of ref. [11]). We speculate that the 8 and/or 217 Hz electromagnetic field alters the electrical properties of brain cells on the exposure side, making cells more excitable. Interestingly, a recent study [9] using transcranial magnetic stimulation to investigate the effect of a 45 min GSM900 MHz (with 217 Hz modulation) exposure reported a neuro-excitatory effect on motor neurons adjacent to the exposure area. This may lend support to the current alerting effect of the talk mode. In conclusion, our systematic study utilising talk, listen, and standby modes of GSM 900 mobile phone signals, together with a sham condition, has found a distinction in the post-exposure effect on sleep onset following talk mode, which seems to be related to its different composition of ELF modulation frequencies.

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