

Effects of cellular phone use on ear canal temperature measured by NTC thermistors

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Summary

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The earlier studies using phantom models and human subjects concerning warming effects during cellular phone use have been controversial, partly because radiofrequency (RF) exposures have been variable. In this randomized, double-blind, placebo-controlled crossover trial, 30 healthy subjects were submitted to 900 MHz (2W) and 1800 MHz (1W) cellular phone RF exposure, and to sham exposure in separate study sessions. Temperature signals were recorded continuously in both ear canals before, during and after the 35-min RF exposure and the 35-min sham exposure sessions. Temperature was measured by using small-sized NTC thermistors placed in the ear canals through disposable ear plugs. The mean temperature changes were determined during a set cardiovascular autonomic function studies: during a 5-min controlled breathing test, during a 5-min spontaneous breathing test, during 7-min head-up tilting, 1-min before, during and after two consecutive Valsalva manoeuvres and during a deep breathing test. Temperatures in the exposed ear were significantly higher during RF exposures compared with sham exposure in both 900 and 1800 MHz studies with maximum differences of $1.2 \pm 0.5^{\circ}\text{C}$ (900 MHz exposure) and $1.3 \pm 0.7^{\circ}\text{C}$ (1800 MHz exposure). Temperatures in the RF-exposed ear were also significantly higher during the postexposure period compared with post-sham exposure period with maximum differences of $0.6 \pm 0.3^{\circ}\text{C}$ for 900 MHz and $0.5 \pm 0.5^{\circ}\text{C}$ for 1800 MHz. The results of this study suggest that RF exposure to a cellular phone, either using 900 or 1800 MHz with their maximal allowed antenna powers, increases the temperature in the ear canal. The reason for the ear canal temperature rising is a consequence of mobile phone battery warming during maximal antenna power use. The earlier published articles do not indicate that temperature rising in the ear canal has any significant contribution from the RF fields emitted from mobile phones.

Introduction

Mobile personal phones are now a mass market product. The billionth GSM user was connected in the first quarter of 2004 and the second billionth connection on the GSM family was made in June 2006. Widespread use of cellular (mobile) phones has raised public and scientific concern about their possible adverse effects on health. The research interest has been focused mainly on brain activity, on subjective symptoms perceived by some users and on the development of malignant tumours (Mann & Röschke, 1996; Borbely et al., 1999; Preece et al., 1999; Hietanen et al., 2000; Koivisto et al., 2000; Krause et al., 2000; Muscat et al., 2000; Inskip et al., 2001; Johansen et al., 2001;

Sandström et al., 2001; Auvinen et al., 2002; Hietanen et al., 2002). Despite of research efforts, the clinical and epidemiological findings are discrepant and do not provide a clear pattern of adverse health effects related to RF exposure emitted by the cellular phones (Krewski et al., 2001). The major barriers in interpreting the studies are the relatively short time of the cellular phones on the markets making impossible to infer epidemiological cause-effect relationships, the difficulties in measuring the changes in the human body and its biosignals, and the difficulties to carry out studies on humans under controlled laboratory conditions. Overcoming those difficulties, some theories have been drawn in the scenario of possible deleterious effects on human health involving thermal, athermal and non-

thermal biological effects produced during the use of cellular phones (Krewski et al., 2001).

While some studies have reported biological effects in the absence of heat, warming of tissues is the major effect of exposure to RF fields. Some studies have been carried out to evaluate temperature changes during mobile phone use (Anderson & Joyner, 1995; Van Leeuwen et al., 1999; Wainwright, 2000; Paredi et al., 2001). In the experimental study by Van Leeuwen et al. the authors found a maximum rise in brain temperature of 0.11°C when a head phantom was exposed to 915 MHz with 0.25 W. Anderson et al. found an increase of 0.034°C in a head phantom during analog mobile phone RF exposure. In a recently published study by Paredi et al. the authors found an increase of 2.3°C in the skin temperature of the nostril and occipital area after 6 min of 900 MHz exposure.

Knowing that rises in temperature above 1°C might lead to behavioural or stress effects, it is important to try to establish to what extent the use of cellular phones increase the temperature in the locally exposed head area. At this moment there are no published studies on human exposure to cellular phones and simultaneous measurements of temperature changes in the ear canal. The theoretical calculations by Bernardi et al. (2001) and Gandhi et al. (2001) have indicated that the temperature increases on skin from RF fields emitted by mobile phones are negligible (a few tenths of a degree Celsius). Those observations have been argued in a recent study by Straume et al. (2005), where temperature rises up to 5.0°C were measured using infrared camera measurements. The observations in Straume et al. (900 MHz mobile with the exposure of 0.21 W maximum output power for 30 min) indicate that the reason for cheek and ear warming is from the mobile phone battery. When currents are drawn from the mobile phone battery, some electrical energy is dissipated in the electronics of the mobile phone. This elevates the temperature of mobile phone and subsequently also raises the skin and head tissue temperature, when the mobile phone is in contact with skin surface.

The objective of this study was to measure temperature changes in the ear canal before, during and after a 35-min RF exposure emitted by cellular phone at frequencies of 900 and 1800 MHz with peak powers of 2 and 1 W and average powers of 0.25 and 0.125 W respectively. Evaluation of ear canal temperature was chosen due to proximity of the ear canal to the brain tissues, and because small-sized NTC thermistor wires can be easily applied to ear canal through disposable ear plugs in clinical settings. Monitoring of ear canal temperature was made in our study parallel to several other signal measurements involved to the cardiopulmonary system during cellular phone RF exposure (Tahvanainen et al., 2004).

Materials and methods

Subjects

Thirty-four healthy volunteers, 16 females and 18 males with an average age of 38.8 ± 10.3 years (SD) were enrolled in the

study, where the objectives were to evaluate human cardiovascular responses and ear canal temperature changes during mobile phone use. Written informed consent was obtained from all subjects. The experimental design of the study was approved by the ethics committee of the Kuopio University Hospital and by the ethics committee of the Finnish Institute of Occupational Health. The clinical experiments were carried out in a quiet laboratory with a well-regulated temperature (21–23°C) and relative humidity (35–45%) at the Department of Clinical Physiology, Kuopio University Hospital. The studies were always started between 1 PM and 3 PM to control circadian effects.

Study design

The subjects in this randomized, double-blinded and placebo-controlled crossover study were exposed during two different days, 1 week apart, to 900 and 1800 MHz cellular phones. The total duration of each study (900 MHz with the antenna power of 2 W and 1800 MHz with the antenna power of 1 W) was 2 h including one 35-min RF exposure and one 35-min sham exposure.

Double randomization, one to predefine the order of the RF exposures (900 MHz followed by 1800 MHz, or 1800 MHz followed by 900 MHz) and one to define the order of the interventions (sham exposure followed by RF exposure, or RF exposure followed by sham exposure) was used. A random digit table was used to generate the random lists (Altman, 1999).

Study protocol

The detailed study protocol has been described in our earlier report (Tahvanainen et al., 2004), where blood pressure and heart rate responses to mobile phone RF exposure was evaluated. Simultaneous recordings of temperature from the left and right ear canals, electrocardiogram (ECG), non-invasively measured arterial blood pressure signal from the finger (finger cuff method) and intermittently from the arm, pneumotachometrically measured respiration signal and relative RF signal intensity produced by cellular phone was conducted. The multi-signal recording was made during physiological challenges, when a set of different autonomic function tests were conducted during and after a 35-min 900 and 1800 MHz RF exposure and sham exposure session. After the RF exposure was switched ON or sham exposure session was started, a 6-min controlled breathing test (CBT) and a 6-min spontaneous breathing test (SBT) were performed in supine position. Next, a 7-min head-up tilt table test (HUT) in 70° head-up position was followed by two consecutive Valsalva manoeuvres (VM), where the subject performed forced expirations against a close glottis with a strain of 40 mmHg for 15 s in supine position during the exposure period. After the 35-min RF or sham exposure period, a 6-min CBT, a 6-min SBT and a deep breathing test (DBT), were conducted in supine position. Each test was separated by a 2-min resting period.

A questionnaire on subjective feelings of the test subjects was applied at the end of RF and sham exposure periods. The questionnaire included three questions: (1) Could you distinguish, whether the cellular phone was ON or OFF during the previous period?, (2) If 'Yes' to the previous question, which one of the two phones was ON?, and (3) How did you feel when the cellular phone was ON?

Exposure and data acquisition

Ear canal temperatures were measured using two identical high-precision miniature NTC thermistors (BetaTHERM Corp., Shrewsbury, MA, USA). The diameter of the drop-shaped thermistors was 2.4 mm, and the resistance at 25°C was 3000 ohms. Thermistors were introduced symmetrically 1.5–2 cm deep to the ear canals through disposable, plasticized PVC foam ear plugs (E-A-R Classic; Aearo Limited, Stockport, UK) in an attempt to create a closed space inside the ear canal without the influence of environmental temperature. The wires of the thermistors were fixed to the skin of the face with micropore. The very thin isolated but non-shielded thermistor wires (length 200 mm) were connected to a custom amplifier with analog output (WinTemp; Absolute Aliens Oy, Turku, Finland) (Fig. 1). The accuracy of the absolute temperature thus measured is about 0.3°C and precision of the relative changes is about 0.05°C in the temperature range of 25–40°C. Both ear canal temperatures were measured simultaneously before, during and after the RF exposure and sham exposure periods.

Because the 900 MHz RF field generated a small (0.1–0.2°C) shift in temperature signal upwards due to direct RF field

interference on the thermistor wires when the phone was switched ON, the mean temperature values during the 900 MHz exposure were corrected by using a correction term as follows. The mean values of the ear canal temperature from two 10 s periods (just before and just after the phone was switched ON) were calculated. The difference between both mean temperature values from the 10 s periods, called as 'correction term', was then subtracted from all the temperature results measured during the 900 MHz exposure in the exposed ear canal.

A PC-controlled dual band mobile phone (Nokia 6150; Nokia Corporation, Helsinki, Finland) was used for providing the RF exposure with maximal antenna powers for the two operational frequencies. Another physically identical but inactive phone without battery was located on the opposite ear. A notebook computer (HP OmniBook XE2; Hewlett-Packard, Inc., Taipei, Taiwan) equipped with a mobile phone service software package (WinTesla; Nokia Corporation, Helsinki, Finland) was used to control the functions of the mobile phone providing RF exposure. The active mobile phone was connected via serial port to the notebook computer.

The mobile phones were located symmetrically on the ears by using a plastic head helmet. The plastic helmet was positioned in the head such that the active phone was leaning to the ear on the dominant hand side of the head, and the inactive phone was leaning to the ear on the non-dominant hand side of the head.

Two specially designed antennas were used to measure the relative intensity of 900 and 1800 MHz fields. Each antenna was connected to a microwave detector (Model 4503A-03; NARDA Microwave-East, Hauppauge, NY, USA) and to an amplifier with analogue output (WinRF; Absolute Aliens Oy) to obtain the RF



Figure 1 Set-up for head area instrumentation. Small-sized NTC thermistors were applied to ear canals through ear plugs (left figure), thermistors wires were fixed to skin and thermistors wires were connected to analog amplifiers. Instrumentation of head area (right figure) including thermistors applied to both ears canals and the thermistors amplifiers with analog outputs on the neck. Respiration was measured using heated Fleisch-type pneumotachometer connected to face mask.

field amplitude signal. The RF field-recording instrumentation was firmly positioned in a holder attached to the tilt table. The RF field-recording antenna was kept at a distance of 20 cm from the active mobile phone during the study.

A notebook computer (HP 4150; Hewlett-Packard, Inc., Lyon, France) equipped with data acquisition and analysis software for signal processing (WinCPRS; Absolute Aliens Oy) was used to record the temperature signals, the relative RF field intensity signal and the biosignals described above during the clinical experiments.

SAR characteristics of the test phone

Specific absorption rate (SAR) measurements for the two operational frequencies (900 and 1800 MHz) of the dual-band cellular phone (Nokia 6150; Nokia Corporation), used for the RF exposure, were conducted inside a head phantom. The details of RF dosimetry measurements are described in detail in our earlier communication (Tahvanainen et al., 2004). Liquid with tissue equivalent dielectric properties was used in the head

phantom in an EMF-shielded laboratory during robotic RF field measurements (DASY3; Schmid & Partner Engineering AG, Zürich, Switzerland). During the SAR measurements the same dual-band mobile phone as used during the clinical studies was programmed to operate when the battery was fully loaded with identical settings. A calibrated RF field scanning probe, moving in a robotic arm, recorded the spatial values of electric field strength (E , in $V\ m^{-1}$) within a $7\ cm \times 7\ cm \times 7\ cm$ cubic volume in the head phantom in the vicinity of mobile phone carefully positioned just below the head phantom. The measured maximal SAR values within the $7\ cm \times 7\ cm \times 7\ cm$ cubic volume of the dual-band phone were $1.58\ W\ kg^{-1}$ at the frequency of 900 MHz and $0.70\ W\ kg^{-1}$ at 1800 MHz. The corresponding penetration depths in the head phantom were 12.4 mm (900 MHz) and 11.4 mm (1800 MHz).

During the robotic SAR measurements in the head phantom, the relative intensity of the RF field for the 900 and 1800 MHz exposures were also monitored for 35 min using the same geometry and with the same recording antennas and amplifier and PC-based data acquisition software as during the clinical

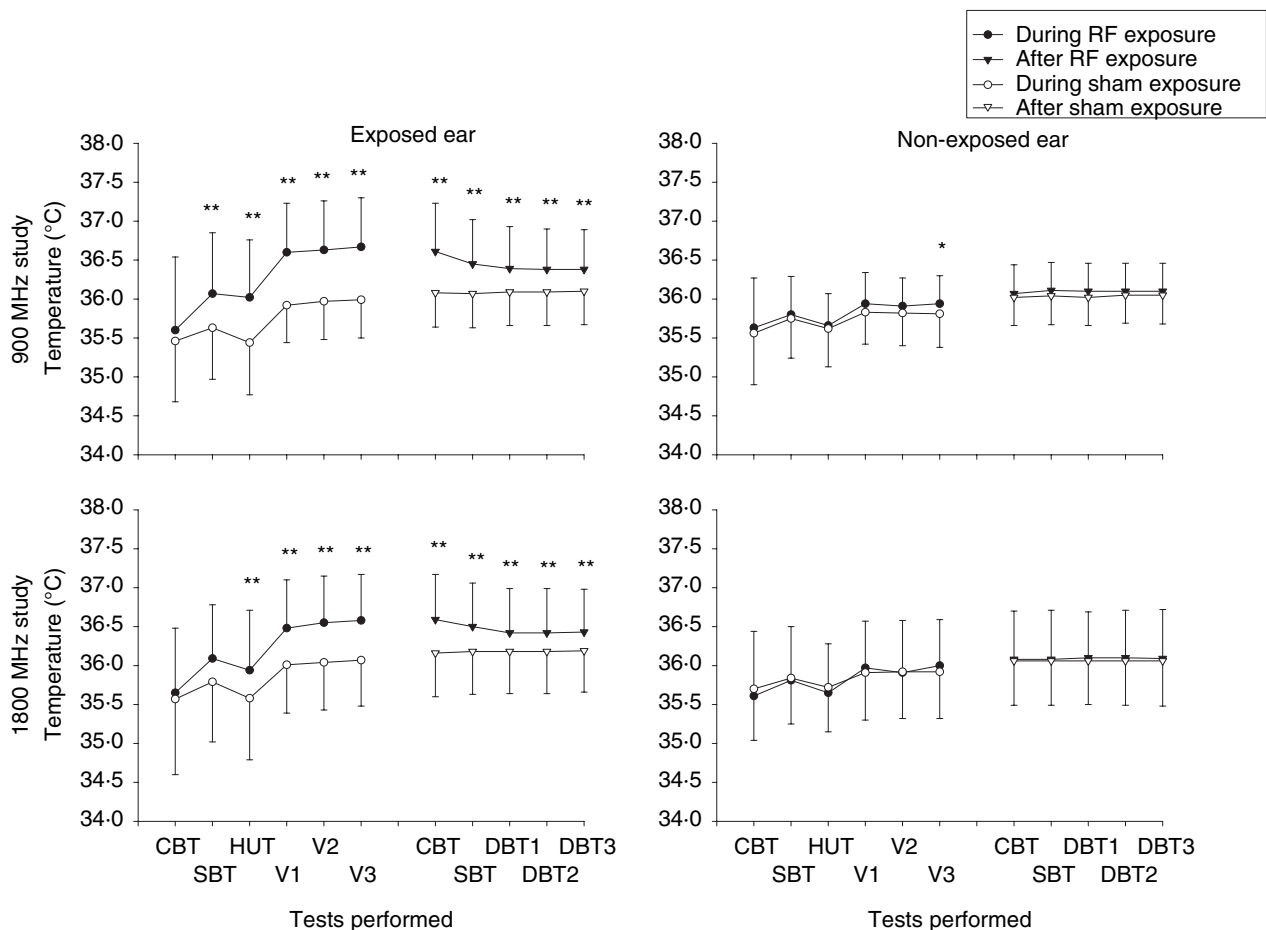


Figure 2 Average ear canal temperature values from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3). * $P < 0.05$; ** $P \leq 0.01$.

studies. The RF field intensity indicated excellent stability ($\pm 2\%$) throughout the 35-min period for both 900 and 1800 MHz frequencies.

Statistical analysis

For the statistical analysis, carry-over effect ($P < 0.1$) and the period effect ($P < 0.05$) were checked by using repeated measures analysis (general linear model). Carry-over effect means that the physical effects of the first treatment are still partly present when the subject enters the second treatment period. Period effect means that there is a systematic difference between the two periods of trial (Altman, 1999).

Student's *t*-test for paired data was used in the comparisons between both interventions (RF exposure versus sham exposure) for temperature measures. Subgroup comparisons were carried out by independent *t*-tests. Non-continuous variables were analysed by using the McNemar test. Two-sided *P*-values were considered statistically significant at $P < 0.05$. All data in the figures and tables are presented as mean values and standard deviations (SD). SPSS software package was used for the statistical analyses (SPSS for Windows, version 9.0; SPSS Inc., Chicago, IL, USA).

Results

Subjects

From the initial 34 subjects, 30 subjects (15 males, 15 females) with an average age of 39.5 years (SD 10.3 and range 23–55 years) and a mean body mass index (BMI) of $23.7 \pm 2.1 \text{ kg m}^{-2}$ were included into the statistical analyses. The reasons for the three exclusions were shoulder pain (one

subject), hypertension (one subject), pain in the ear channel (one subject). The fourth subject, an airplane pilot, was excluded from ear canal temperature measurements because of safety risks for his profession. Twenty-six (87%) right-handed subjects received the RF exposure on the right side of the head and four (13%) left-handed on the left side.

Exposed ear

There were statistically significant differences in the ear canal temperatures between RF exposure and sham exposure, both for 900 and 1800 MHz phones. Accordingly, there were statistically significant differences in the ear temperatures between post-exposure and post-sham conditions both at 900 and 1800 MHz.

When subjects were exposed to 900 MHz, temperature was significantly higher during SBT, HUT and VM compared with sham exposure intervention ($P < 0.01$) (Fig. 2 left upper panel, Table 1). The mean differences between both interventions were $0.5 \pm 0.9^\circ\text{C}$ during SBT, $0.6 \pm 0.7^\circ\text{C}$ during HUT and $0.7 \pm 0.5^\circ\text{C}$, $0.7 \pm 0.5^\circ\text{C}$ and $0.7 \pm 0.5^\circ\text{C}$ during the three analysis periods of VM ($P < 0.01$). The mean differences between post-RF exposure and post-sham exposure temperatures were $0.5 \pm 0.5^\circ\text{C}$ during CBT, $0.4 \pm 0.5^\circ\text{C}$ during SBT and $0.3 \pm 0.4^\circ\text{C}$, $0.3 \pm 0.4^\circ\text{C}$ and $0.3 \pm 0.4^\circ\text{C}$ during the three analysis periods of DBT ($P < 0.01$) (Fig. 2 left upper panel, Table 1).

The same pattern in temperature dynamics was observed in the 1800 MHz study. With RF exposure, temperature was significantly higher during the HUT (mean difference of $0.4 \pm 0.7^\circ\text{C}$) and during the three analysis periods of VM (mean differences of $0.5 \pm 0.4^\circ\text{C}$, $0.5 \pm 0.4^\circ\text{C}$ and $0.5 \pm 0.4^\circ\text{C}$) compared with sham exposure ($P < 0.01$) (Fig. 2 left lower panel, Table 2). The mean differences between post-

Table 1 Mean $\pm$ SD ear canal temperature values in the RF-exposed and non-exposed ear in the 900 MHz study. Temperatures were calculated from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3).

Tests performed	Temperature RF-exposed ear			Temperature Non-exposed ear		
	During RF exposure ($^\circ\text{C}$)	During sham exposure ($^\circ\text{C}$)	<i>P</i>	During RF exposure ($^\circ\text{C}$)	During sham exposure ($^\circ\text{C}$)	<i>P</i>
CBT	35.6 ± 0.9	35.5 ± 0.8	0.59	35.6 ± 0.6	35.6 ± 0.7	0.76
SBT	36.1 ± 0.8	35.6 ± 0.7	0.01	35.8 ± 0.5	35.7 ± 0.5	0.68
HUT	36.0 ± 0.7	35.4 ± 0.7	0.00	35.7 ± 0.4	35.6 ± 0.5	0.66
V1	36.6 ± 0.6	35.9 ± 0.5	0.00	35.9 ± 0.4	35.8 ± 0.4	0.08
V2	36.6 ± 0.6	35.9 ± 0.5	0.00	35.9 ± 0.4	35.8 ± 0.4	0.06
V3	36.6 ± 0.6	35.9 ± 0.5	0.00	35.9 ± 0.4	35.8 ± 0.4	0.02
	After RF exposure ($^\circ\text{C}$)	After sham exposure ($^\circ\text{C}$)		After RF exposure ($^\circ\text{C}$)	After sham exposure ($^\circ\text{C}$)	
CBT	36.6 ± 0.6	36.1 ± 0.4	0.00	36.1 ± 0.4	36.0 ± 0.4	0.19
SBT	36.4 ± 0.6	36.1 ± 0.4	0.00	36.1 ± 0.4	36.0 ± 0.4	0.10
DBT1	36.4 ± 0.5	36.1 ± 0.4	0.00	36.1 ± 0.4	36.0 ± 0.4	0.19
DBT2	36.4 ± 0.5	36.1 ± 0.4	0.00	36.1 ± 0.4	36.1 ± 0.4	0.34
DBT3	36.4 ± 0.5	36.0 ± 0.4	0.00	36.1 ± 0.4	36.1 ± 0.4	0.33

Data were mean $\pm$ SD.

Table 2 Mean $\pm$ SD ear canal temperature values in the RF-exposed and non-exposed ear in the 1800 MHz study. Temperatures were calculated from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3).

Tests performed	Temperature RF-exposed ear		<i>P</i>	Temperature Non-exposed ear		<i>p</i>
	During RF exposure ($^{\circ}$ C)	During sham exposure ($^{\circ}$ C)		During RF exposure ($^{\circ}$ C)	During sham exposure ($^{\circ}$ C)	
CBT	35.7 $\pm$ 0.8	35.6 $\pm$ 1.0	0.78	35.6 $\pm$ 0.8	35.7 $\pm$ 0.7	0.63
SBT	36.1 $\pm$ 0.7	35.8 $\pm$ 0.8	0.11	35.8 $\pm$ 0.7	35.8 $\pm$ 0.6	0.81
HUT	35.9 $\pm$ 0.8	35.6 $\pm$ 0.8	0.01	35.7 $\pm$ 0.6	35.7 $\pm$ 0.6	0.35
V1	36.5 $\pm$ 0.6	36.0 $\pm$ 0.6	0.00	36.0 $\pm$ 0.6	35.9 $\pm$ 0.6	0.38
V2	36.6 $\pm$ 0.6	36.0 $\pm$ 0.6	0.00	35.9 $\pm$ 0.7	35.9 $\pm$ 0.6	0.89
V3	36.6 $\pm$ 0.7	36.1 $\pm$ 0.6	0.00	36.0 $\pm$ 0.6	35.9 $\pm$ 0.6	0.18
	After RF exposure ($^{\circ}$ C)	After sham exposure ($^{\circ}$ C)		After RF exposure ($^{\circ}$ C)	After sham exposure ($^{\circ}$ C)	
CBT	36.6 $\pm$ 0.6	36.2 $\pm$ 0.6	0.00	36.1 $\pm$ 0.6	36.1 $\pm$ 0.6	0.58
SBT	36.5 $\pm$ 0.6	36.2 $\pm$ 0.6	0.00	36.1 $\pm$ 0.6	36.1 $\pm$ 0.6	0.67
DBT1	36.4 $\pm$ 0.6	36.2 $\pm$ 0.5	0.00	36.1 $\pm$ 0.6	36.1 $\pm$ 0.6	0.47
DBT2	36.4 $\pm$ 0.6	36.2 $\pm$ 0.5	0.00	36.1 $\pm$ 0.6	36.1 $\pm$ 0.6	0.45
DBT3	36.4 $\pm$ 0.6	36.2 $\pm$ 0.5	0.00	36.1 $\pm$ 0.6	36.1 $\pm$ 0.6	0.58

Data were mean $\pm$ SD.

RF exposure and post-sham exposure temperatures were $0.4 \pm 0.3^{\circ}$ C during CBT, $0.3 \pm 0.3^{\circ}$ C during SBT and $0.2 \pm 0.3^{\circ}$ C, $0.2 \pm 0.3^{\circ}$ C and $0.2 \pm 0.3^{\circ}$ C during the three analysis periods of DBT ($P < 0.01$) (Fig. 2 left lower panel, Table 2).

No carry-over effects were observed either during the 900 MHz study or during the 1800 MHz study. A period effect was observed during the 900 MHz study as well as during the 1800 MHz study. Temperature of the ear canal increased at the beginning of the study independent of the intervention (RF exposure or sham exposure) (Figs 3 and 4, Tables 3 and 4). When the study started with sham exposure, the mean temperature increased throughout the sham exposure period by 1.0° C in the study with 900 MHz and by 1.2° C in the study with 1800 MHz. When the study started with RF exposure, the ear canal temperature on the exposed side increased throughout the exposure period by 1.5° C during the study with 900 MHz and by 1.3° C during the study with 1800 MHz.

When the studies (900 and 1800 MHz) started with sham exposure, the mean temperature values were significantly higher in every study performed when subjects were under RF exposure compared with sham exposure. Moreover during the postexposure period temperatures were significantly higher compared with post-sham exposure period ($P < 0.01$) (Fig. 3 left panels, Tables 3 and 4). In the 900 MHz study, the differences between both interventions range from $0.7 \pm 0.5^{\circ}$ C to $1.2 \pm 0.5^{\circ}$ C during the intervention period and from $0.4 \pm 0.2^{\circ}$ C to $0.6 \pm 0.3^{\circ}$ C during postintervention period ($P < 0.01$) (Fig. 3 left upper panel, Table 3). In the 1800 MHz study, the differences range from $0.6 \pm 0.4^{\circ}$ C to $1.3 \pm 0.7^{\circ}$ C during intervention period and from $0.3 \pm 0.3^{\circ}$ C to $0.5 \pm 0.4^{\circ}$ C during postintervention period ($P < 0.01$) (Fig. 3 left lower panel, Table 4).

When the studies were started with RF exposure, mean temperature values of sham exposure were significantly higher than RF exposure during CBT (for 900 and 1800 MHz, $P < 0.01$) and during SBT (for 1800 MHz, $P < 0.01$) (Fig. 3 right panels). Mean temperature values were significantly higher during RF exposure compared with sham exposure during the VM. The differences ranged from $0.4 \pm 0.5^{\circ}$ C to $0.5 \pm 0.5^{\circ}$ C for the 900 MHz study and from $0.3 \pm 0.2^{\circ}$ C to $0.4 \pm 0.3^{\circ}$ C for the 1800 MHz study ($P < 0.01$) (Fig. 3 right panels, Tables 3 and 4). In the 900 MHz study, temperature was higher also during the CBT of the postexposure period compared with the post-sham exposure ($0.4 \pm 0.6^{\circ}$ C, $P < 0.05$) (Fig. 3 right upper panel, Table 3). In the 1800 MHz study, temperature was higher during the whole postexposure period compared with the post-sham exposure period. The differences ranged from $0.2 \pm 0.2^{\circ}$ C to $0.3 \pm 0.3^{\circ}$ C ($P < 0.01$) (Fig. 3 right lower panel, Table 4).

Non-exposed ear

There were no statistically significant differences in the ear canal temperature between the sham exposure and 1800 MHz RF exposure (Fig. 2 right lower panel, Table 2). During the 900 MHz study the temperature at the end of VM was $0.1 \pm 0.2^{\circ}$ C higher during exposure compared with sham exposure ($P < 0.05$) (Fig. 2 right upper panel, Table 1). There were no statistically significant differences between postexposure and post-sham exposure periods neither during 900 MHz nor during 1800 MHz studies (Fig. 2 right panels, Tables 1 and 2). No carry-over effects were observed either during 900 MHz study or during 1800 MHz study.

A period effect was present in both studies (900 and 1800 MHz). Temperatures increased at the beginning of the study independent of the intervention (RF exposure or sham

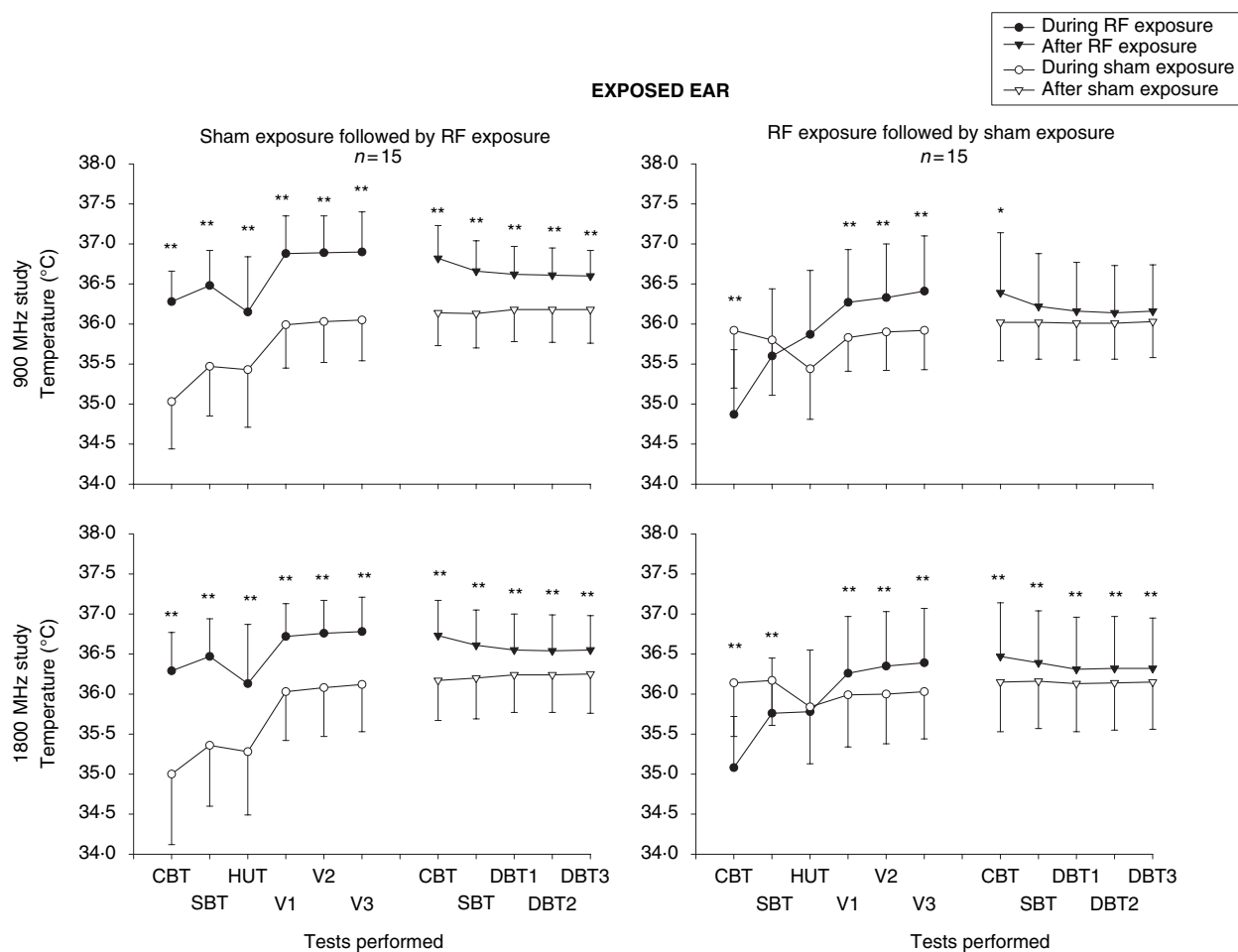


Figure 3 Average ear canal temperature values in the RF-exposed ear from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3). * $P < 0.05$; ** $P \leq 0.01$.

exposure). When the study started with sham exposure, temperatures increased by 0.7°C throughout the sham exposure period while during RF exposure period temperature did not increase. Mean temperature values were significantly higher during the CBT and during the SBT (for 900 and 1800 MHz, $P < 0.01$) and also during the HUT (for 900 MHz, $P < 0.01$) when subjects were under RF exposure compared with sham exposure (Fig. 4, left panels).

When the study started with RF exposure, temperatures increased by 0.8°C for 900 MHz and 0.7°C for 1800 MHz throughout the RF exposure period while during sham exposure period temperatures did not increase (Fig. 4 right panels). Mean temperature values were significantly higher during CBT and SBT (for 900 and 1800 MHz, $P < 0.01$) and also HUT (for 1800 MHz, $P < 0.01$), when subjects were under sham exposure compared with RF exposure (Fig. 4 right panels).

Temperature responses were not affected by age or gender either during sham exposure or during 900 or 1800 MHz exposures.

Subjective symptoms

There were no differences in the subjective symptoms reported by the study subjects between RF exposure and sham exposure regarding local heat. With 900 MHz exposure, four subjects experienced local heat (three in the RF-exposed ear and one in the non-exposed ear). Accordingly, for 1800 MHz exposure three subjects (two in the exposed ear and one in the non-exposed ear) experienced local heat. During the sham exposures, one subject referred to local heat during the 900 MHz study sham exposure and two during the 1800 MHz study sham exposure.

Discussion

The results of this randomized placebo-controlled crossover trial indicates that temperature in the canal of the RF-exposed ear (side of the head where the active phone was held), measured by NTC thermistors, increases during a 35-min RF exposure of

Table 3 Mean $\pm$ SD ear canal temperature values in the RF-exposed ear in the 900 MHz study according to order of intervention. Temperatures were calculated from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3).

Tests performed	Temperature Sham exposure followed by RF exposure			Temperature RF exposure followed by sham exposure		
	During RF exposure ($^{\circ}\text{C}$)	During sham exposure ($^{\circ}\text{C}$)	<i>P</i>	During RF exposure ($^{\circ}\text{C}$)	During sham exposure ($^{\circ}\text{C}$)	<i>p</i>
CBT	36.3 $\pm$ 0.4	35.0 $\pm$ 0.6	0.00	34.9 $\pm$ 0.8	35.9 $\pm$ 0.7	0.00
SBT	36.5 $\pm$ 0.4	35.5 $\pm$ 0.6	0.00	35.6 $\pm$ 0.8	35.8 $\pm$ 0.7	0.34
HUT	36.1 $\pm$ 0.7	35.4 $\pm$ 0.7	0.00	35.9 $\pm$ 0.8	35.4 $\pm$ 0.6	0.12
V1	36.9 $\pm$ 0.5	35.9 $\pm$ 0.5	0.00	36.3 $\pm$ 0.7	35.8 $\pm$ 0.4	0.00
V2	36.9 $\pm$ 0.5	36.0 $\pm$ 0.5	0.00	36.3 $\pm$ 0.7	35.9 $\pm$ 0.5	0.01
V3	36.9 $\pm$ 0.5	36.0 $\pm$ 0.5	0.00	36.4 $\pm$ 0.7	35.9 $\pm$ 0.5	0.01
	After RF exposure ($^{\circ}\text{C}$)	After sham exposure ($^{\circ}\text{C}$)		After RF exposure ($^{\circ}\text{C}$)	After sham exposure ($^{\circ}\text{C}$)	
CBT	36.8 $\pm$ 0.4	36.1 $\pm$ 0.4	0.00	36.4 $\pm$ 0.7	36.0 $\pm$ 0.5	0.04
SBT	36.7 $\pm$ 0.4	36.1 $\pm$ 0.4	0.00	36.2 $\pm$ 0.7	36.0 $\pm$ 0.5	0.19
DBT1	36.6 $\pm$ 0.3	36.2 $\pm$ 0.4	0.00	36.2 $\pm$ 0.6	36.0 $\pm$ 0.5	0.30
DBT2	36.6 $\pm$ 0.3	36.2 $\pm$ 0.4	0.00	36.1 $\pm$ 0.6	36.0 $\pm$ 0.4	0.31
DBT3	36.6 $\pm$ 0.3	36.2 $\pm$ 0.4	0.00	36.2 $\pm$ 0.6	36.0 $\pm$ 0.4	0.32

Data were mean $\pm$ SD.

Table 4 Mean $\pm$ SD ear canal temperature values in the RF-exposed ear in the 1800 MHz study according to order of intervention. Temperatures were calculated from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3).

Tests performed	Temperature Sham exposure followed by RF exposure			Temperature RF exposure followed by sham exposure		
	During RF exposure ($^{\circ}\text{C}$)	During sham exposure ($^{\circ}\text{C}$)	<i>P</i>	During RF exposure ($^{\circ}\text{C}$)	During sham exposure ($^{\circ}\text{C}$)	<i>p</i>
CBT	36.3 $\pm$ 0.5	34.9 $\pm$ 0.9	0.00	35.1 $\pm$ 0.6	36.1 $\pm$ 0.7	0.00
SBT	36.5 $\pm$ 0.5	35.4 $\pm$ 0.8	0.00	35.8 $\pm$ 0.7	36.2 $\pm$ 0.6	0.01
HUT	36.1 $\pm$ 0.7	35.3 $\pm$ 0.8	0.00	35.8 $\pm$ 0.8	35.8 $\pm$ 0.7	0.59
V1	36.7 $\pm$ 0.4	36.0 $\pm$ 0.6	0.00	36.3 $\pm$ 0.7	36.0 $\pm$ 0.7	0.00
V2	36.8 $\pm$ 0.4	36.1 $\pm$ 0.6	0.00	36.4 $\pm$ 0.7	36.0 $\pm$ 0.6	0.00
V3	36.8 $\pm$ 0.4	36.1 $\pm$ 0.6	0.00	36.4 $\pm$ 0.7	36.0 $\pm$ 0.6	0.00
	After RF exposure ($^{\circ}\text{C}$)	After sham exposure ($^{\circ}\text{C}$)		After RF exposure ($^{\circ}\text{C}$)	After sham exposure ($^{\circ}\text{C}$)	
CBT	36.7 $\pm$ 0.4	36.2 $\pm$ 0.5	0.00	36.5 $\pm$ 0.7	36.2 $\pm$ 0.6	0.00
SBT	36.6 $\pm$ 0.4	36.2 $\pm$ 0.5	0.00	36.4 $\pm$ 0.7	36.2 $\pm$ 0.6	0.00
DBT1	36.6 $\pm$ 0.5	36.2 $\pm$ 0.5	0.00	36.3 $\pm$ 0.7	36.1 $\pm$ 0.6	0.01
DBT2	36.5 $\pm$ 0.5	36.2 $\pm$ 0.5	0.01	36.3 $\pm$ 0.7	36.1 $\pm$ 0.6	0.01
DBT3	36.6 $\pm$ 0.4	36.3 $\pm$ 0.5	0.01	36.3 $\pm$ 0.6	36.2 $\pm$ 0.6	0.01

Data were mean $\pm$ SD.

900 and 1800 MHz, the operational frequencies used in the European cellular phones. Although temperatures were significantly higher also during the postexposure period, the maximal increases were found during the last three tests performed during the RF exposure period, reaching the maximum increases of 1.2 $^{\circ}\text{C}$ (for the 900 MHz) and 1.3 $^{\circ}\text{C}$ (for the 1800 MHz). There were no increases in the ear canal temperature on the side of the head where the inactive phone was held, suggesting that the increases in the temperature exist only in the local area where the RF exposure was given. In the present

study, the temperature started to decrease immediately after the RF exposure was switched OFF.

Temperature recordings in the present study were started almost immediately after the NTC thermistors were inserted through ear plugs into the ear canals. Independent that the air volume in the ear canal between tympanus and the ear plug is small, the time to obtain thermal equilibrium explains partly the increase of temperature during the first minutes of recording. This is due to normal thermal stabilization in the ear canal between the core temperature and the environment. The

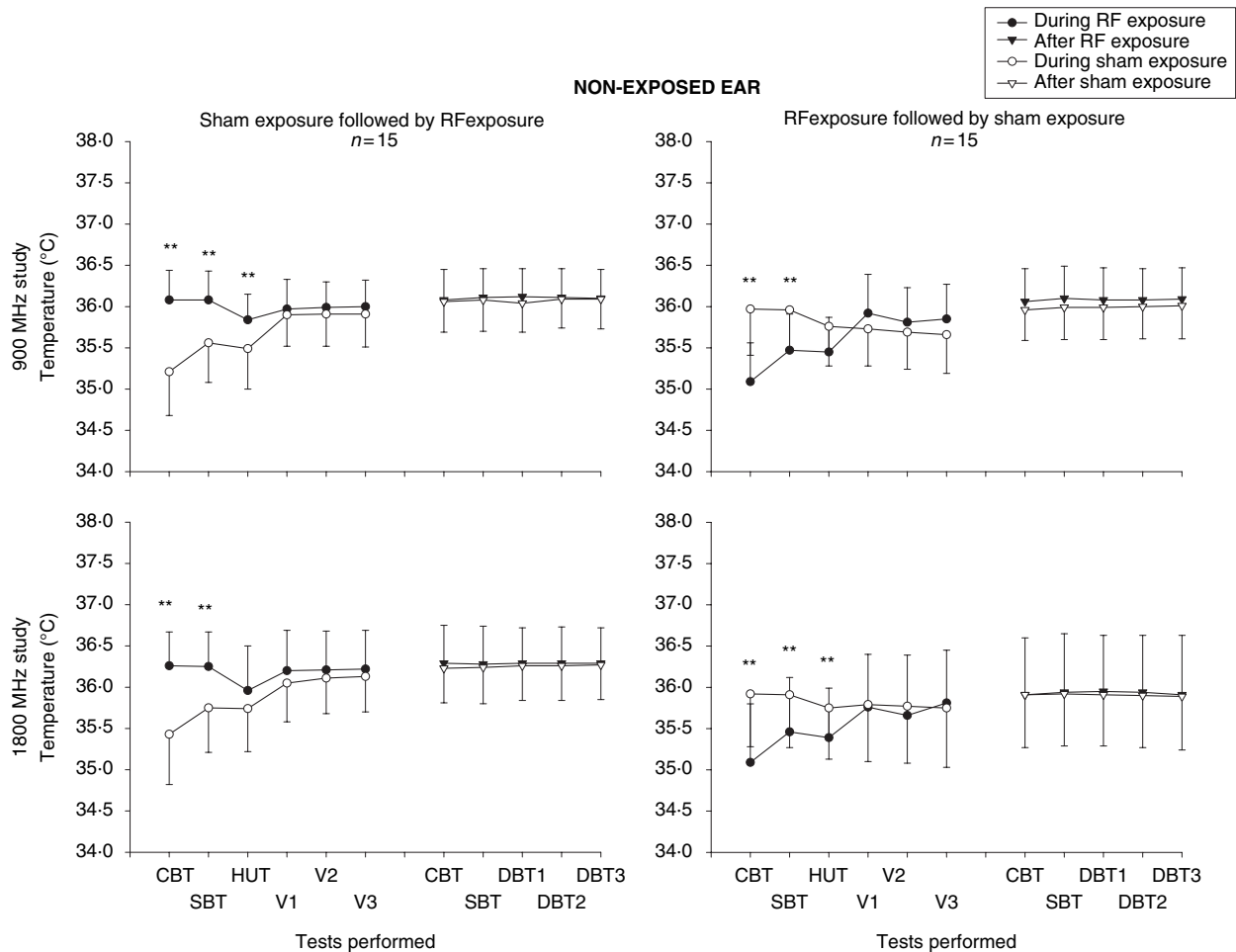


Figure 4 Average ear canal temperature values in the non-exposed ear from a 5-min period during the controlled breathing test (CBT), during the spontaneous breathing test (SBT) and during head-up tilting (HUT), from a 1-min period before the first Valsalva manoeuvre (V1), between the two Valsalva manoeuvres (V2) and after the second Valsalva manoeuvre (V3) and from a 1-min period before, during and after the deep breathing test (DBT1, DBT2 and DBT3). ** $P \leq 0.01$.

statistically significant period effect found in the present study, is a reflection of the time needed for the temperature inside the ear canal to get stable. In any case the increases were statistically higher in the RF-exposed ear when the subjects were under 900 and 1800 MHz RF exposure.

The increases in temperature (1.2 – 1.3°C) found in the present study are between the data obtained by Van Leeuwen *et al.* with an increase of 0.1°C in the brain temperature of a head phantom model, and the data obtained by Paredi *et al.* with an increase of 2.3°C in the occipital area skin temperature of human subjects. In the study by Straume *et al.* (2005) increases of 3.1 – 5.0°C in temperature were observed in the cheek and ear area, when measurements were made with infrared camera techniques.

In our study the temperature rise lasted approximately 19 min to stabilize, which is in agreement with the data found by Van Leeuwen *et al.* The maximal increase of temperature found by Paredi *et al.* was observed after 6 min of RF exposure. A marked difference between the study protocols in our study and in the study by Paredi *et al.* is the physiological challenge of

HUT in our study. Tilt table was moved to 70 degrees head-up position at 13–14 min from the start of 35-min RF or sham exposure. The blood volume decrease in the upper part of the body in head-up position obviously is reflected as a small decrease in temperature.

This study provides evidence that cellular phone use with maximal allowed antenna power results in statistically significant temperature elevation in the ear canal, when temperature is measured by NTC thermistors. It is most obviously a result of the conducted/reflected heat between the skin and the battery of the cellular phone and independent of RF fields emitted from the mobile phone.

As mentioned in the methods, 900-MHz RF exposure produced a small interference in the NTC thermistor signal (0.2°C elevation at maximum) in the temperature signal at the moment of switching the phone ON because the very thin thermistor wires are non-shielded and therefore sensitive to direct coupling to RF field. A 900-MHz mobile phone transmits very short-duration bursts with a frequency of 217 Hz. As the

sampling rate of temperature signals was 200 Hz, the mobile phone generated amplitude modulation was under-sampled resulting spectral folding and the generation of the 17 Hz signal. Thermistor amplifiers included heavy low-pass filtering for RF interferences but such a very low modulation frequency could not be eliminated totally, especially because the amplitude modulation was so strong. This results to the need of temperature correction, which was made carefully to the temperature observations during 900 MHz exposure. It is important to be aware of this kind of possible interference of the RF fields with the instruments used to measure the biosignals that may lead to biased conclusions without careful evaluation.

The decrease in both ear canals' temperature, both on RF-exposed and non-exposed side, during HUT is a reflection of blood volume changes in the head during orthostatic stress. The findings encourage the possibility to use ear canal temperature measurement as a signal that could provide noninvasive data about blood volume changes in the central nervous system.

With the increased public concern regarding RF field exposures and potential adverse human health effects, different authorities and scientific communities have been developing safety guidelines (International Commission on Non-Ionizing Radiation Protection (ICNIRP), 1998; Minister of Public Works and Governmental Services, Canada, 1999). The temperature changes in the tissues are the basis for SAR safety level determination. The major difficulties on the safety level determination are the acquisition of temperature signal and the impossibility of obtaining SAR measurements *in vivo*.

Knowing that the major concern of deleterious health effect of RF are the possible alterations in the central nervous system function, changes in human brain temperature during mobile phone use are of vital importance. As *in vivo* brain temperature measurement at the moment is not available, the ear canal is the closest, easiest and reachable way to get approximate picture of core temperature changes during cellular phone use. In general, measurements of temperature inside a human body are one of the most difficult tasks in terms of accuracy. The increase in ear canal temperature, which our study demonstrates, is mostly due to warming of phone RF output amplifier at maximum antenna powers, heat conduction from the phone, and the subsequent warming of cheek and ear tissues. Direct RF heating by the mobile phones has been modelled and estimated (Bernardi et al., 2001; Gandhi et al., 2001) to cause only a negligible temperature rise (a few tenths of degree Celsius) in the head area. Finally, we note that the dual-band cellular phone model (Nokia 6150) used in this study was introduced to markets in 1998, and may not be representative of current designs or battery technologies.

More clinical studies on this topic are warranted with alternative temperature measurement technologies and newest mobile phone and battery technologies since the articles published this far have provided some controversial results in the thermal effects during cellular phone use.

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